HYGIENE

VOLUME II

OCCUPATIONAL MEDICINE

INDUSTRIAL HYGIENE
OCCUPATIONAL DISEASES

EDITED BY

Prof. Dr. Dimitar Tsvetkov, DSci

2019

I dedicate this book to the memory of my father -
Prof. Dr. Lyubomir T. Tsvetkov (1899-1984),
my mother - Dr. Tsvetana Hr.Tsvetkova (1905-1985) and my family -
Kristina (1939-2009) , Mira and Dorothea

With gratitude and thanks to
Prof. Dr. Andreeva-Galanina Evgeniy Cesarevna (1888–1976),
Russia, Prof. Dr. Vladimir Boyadzhiev (1923–2011), long-time
Head of the Department of Hygiene and Occupational Diseases

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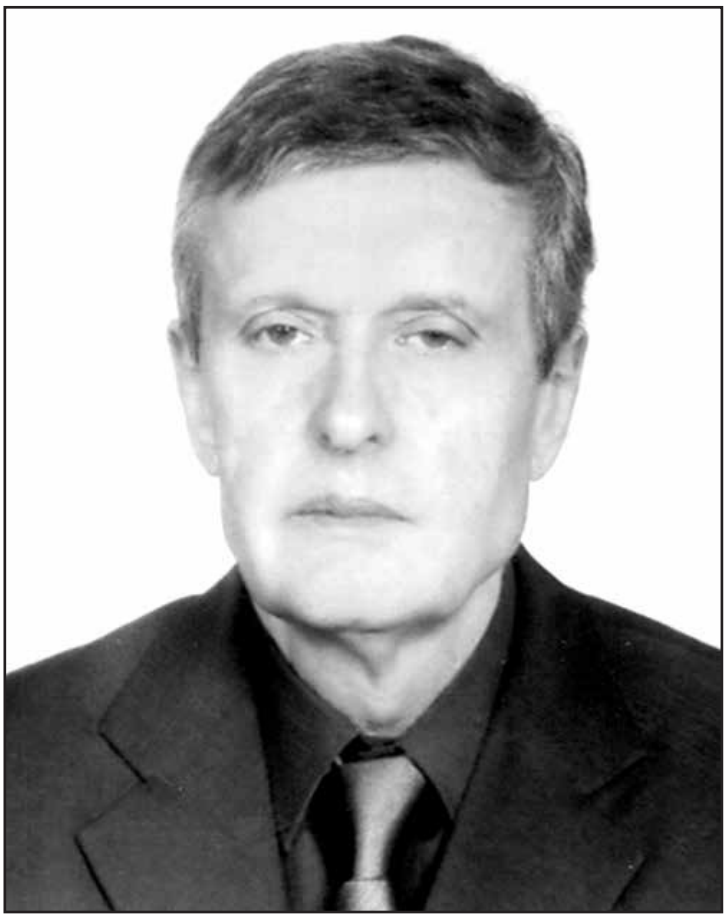


PROF. DR. DIMITAR TSVETKOV, MD
EDITOR

Prof. Dr. Dimitar Tsvetkov, MD graduated in medicine in 1961 from 1990 to 1999 was head of the Department of hygiene, ecology and professional MF diseases Sofia. MD happened in 1972 and Doctor of Medical Sciences in 1991. He was a deputy. dean and vice. Rector of the Medical University Sofia member of the Academic Board and a member of the senior educational council of Education, Director National Center of Hygiene.

His research interests are in the field of occupational medicine, in particular Physical work environment factors. Developing methodology for research risk role of physical stressors on cardiovascular diseases and dyslipidemia. There are numerous studies on working conditions and health of workers in mining, ferrous and nonferrous metallurgy, production of building materials, etc.; in the field of communal hygiene, nutrition, occupational toxicology, hygiene of children and adolescents, ergonomics.

Prof. Tsvetkov is a member of the leadership of the Scientific Society of hygiene Bulgaria, Bulgarian National Academy of Medicine, a member of New York Academy of Sciences, Polish Society of Hygiene, the leadership of Atlanto Euro Mediterranean Academy of Medical Sciences and Bulgarian Lipid league. Editor and member of the editorial boards of numerous scientific journals at home and abroad.

Author of over 190 scientific publications. He has participated in writing and edition of 5 textbooks, 3 manuals on hygiene and occupational diseases, one monograph.



Prof. Evgeniya G. Dincheva, DSci, in 1961, graduated in Medicine at the Medical Institute in Sofia. Since 1969 she is a research fellow, since 1982 - Assoc. Prof. Since 1985 she has been a Prof. in the Department of Physiology at the NCHMEN. She has been PhD since 1969 and since 1984 has been a Doctor of Medical Sciences. From 1989 to 1992 she was the director of the NCMEN. Member of BNAM, IBRO, Scientific Society of Hygiene. He is a specialist in the field of occupational medicine and more closely in the field of physiology and psychophysiology of labour. More than 150 scientific papers, papers on occupational medicine and monographs in physiology and psychophysiology of labour have been published.



Ass. Prof. Bistra M. Tsenova, PhD, graduated from the Humboldt University in Berlin in 1976. She defended her doctorate in psychology at St. Petersburg State University in 1982. She received a degree in Medical Psychology and Pedagogical Rehabilitation. She has specialized psychosocial factors and stress resistance, psychotoxicology, modern epidemiology and health promotion (Germany, Russia, Finland). For more than 20 years she has worked as a research fellow at the National Center for Hygiene, Medical Ecology and Nutrition. It deals with the psychological aspects of the analysis and evaluation of work activities (content and conditions) and their consequences for health, personal development and organizations. She has participated in international projects - research on the motivation for achievement and neurobehavioural effects of lead in children (WHO). There are over 120 publications and reports to international and national scientific forums in the fields of work psychology, stress, psychotoxicology, individual differences, and more.



Assoc. Prof. Zachary K. Ivanov, PhD, graduated in medicine in MU - Sofia and then a doctorate in NIHPZ in 1967 PhD from 1968. Worked as a Assistant and Ass. Prof. in Scientific Institute of labour and Institute of economy and organization of engineering (where is chief of the "Ergonomics and physiology of labour") until 1990. Since 1990 Ass. Prof. in the department" Hygiene, ecology and Prof. Diseases" of MF - Sofia. There are over 135 scientific publications and reports in the field of ergonomics, hygiene and safety. He has authored over 15 scientific and popular brochures, 2 educational film on ergonomics and hygiene, co-author in the manual of Hygiene and Professional Diseases.



Chief Assist. Dr. Ftyana Y. Kalburova graduated from higher education in 1971 at the Higher Institute of Mathematics - Plovdiv. Since 1974 she has been an assistant in the Department of Hygiene at the Higher Medical Institute - Sofia. In 1978-1985, she was a research associate at the Joint Aeromedical Institute at the Military Medical Academy. Since 1985 she has been a general assistant in the Department of Hygiene at the Sofia Medical University. She has a specialty in occupational hygiene - 1982 and occupational diseases - 1993. Scientific interests are in the field of physical factors of the working environment - noise, vibrations, electromagnetic fields, microclimate. She has over 70 scientific works, rationalizations, and participations in manuals and monographs on Hygiene and Occupational Medicine.

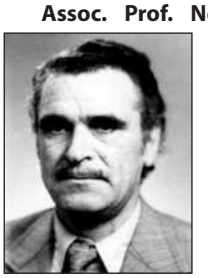


Prof. Dr. Bozhidar Stefanov DSci. graduated in medicine at the Medical University Donetsk - Ukraine in 1970. Two years later he completed dissertation - PhD. He has worked in HEI Varna and at the Medical University of Varna as an assistant. Since 1977 he is Associate Professor and Head of Department "Hygiene, Medical Ecology and Occupational diseases" at the Medical University - Pleven. Since 1984, Doctor of Medical Sciences and Professor. During the period 1980-1987, and the 1989-1991, he was vice Rector of the Medical University - Pleven. Since 2001 he is Chairman of the General meeting of the Medical University - Pleven. There are specialties of general hygiene occupational, hygiene and communal hygiene. Since 1988 a member of the Global Environmental Epidemiology Network (GEENET). He published over 200 scientific papers in the field of hygiene, ergonomics, medical ecology and medical informatics, 10 brochures and monographs, including. "Glossary of Hygiene", "Radio waves and magnetic fields (hygienic aspects)."

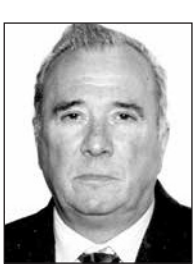


Prof. Dr. Zlatka Stoyneva, PhD, graduated in 1979 at the Medical Academy, Sofia. From 1980-1983 she is a regular postgraduate at the Clinic of Occupational Diseases - MA, Sofia. Since 1983 she has been a PhD III-I degree assistant, chief assistant at the Clinic of Occupational Diseases. He has been Head of the Clinical Neurovegetology Laboratory since 1996. Associate Professor - 2005. Specialist in Neurology and Occupational Diseases. She has been a PhD since 1983. Professor - 2016. She has specialized in Russia, Sweden, Japan. Her scientific interests are in the field of professional pathology and neurology, working on vibrational disease, occupational pathology of overintension and microtraumatism of the nervous and musculoskeletal system, diagnostics of autonomic neurovascular, thermoregulatory, sudomotor, microcirculative and trophic disorders in acrial syndromes and diseases. Its main scientific contributions are in the field of neurovegetology and microcirculation. She is the author of more than 100 scientific publications, monographs, textbooks and guides on occupational diseases and neurology.

Assoc. Prof. Nestor Mitrev, PhD,



graduated from the Higher Institute of Medicine - Plovdiv in 1961. Since 1969, he has been an assistant professor, since 1984 an associate professor at the Department of Hygiene at the Higher Institute of Medicine - Plovdiv. Since 1988 he has been head of the same department. PhD (1979). Co-author of many practical manuals and hygiene textbooks for medical and dental practitioners, author of three monographs, two inventions and one rationalization. His scientific interests are in the field of occupational medicine - mainly the impact of noise and vibration.



Prof. Dr. Dimitar G. Dimitrov, DSci, graduated in medicine in 1961 at the Higher Medical Institute - Sofia. Aviation doctor from 1962 to 1965. Since 1966 he has been a research associate at the Joint Aeromedical Research Institute - Sofia. It passes through all the scientific degrees (PhD and DSci) and titles. There are over 330 scientific publications, 5 monographs, a significant part of which are printed and popularized in other countries. His main

scientific interests are in the field of physiology of dynamic flight factors, brain circulation in extreme situations, the role of human factor in the origin of aviation catastrophes and accidents.



Chief Assist. Dr. Adriana Iv. Agovska, PhD graduated from higher medical education in 1989 at the Medical Faculty in Sofia. From 1989 to 1991 he was a doctor at HEI - Vratsa. Since 1991, she has been an assistant at the Department of Hygiene, Ecology and Occupational Diseases at the Sofia Medical University. Her scientific interests are in the field of psychophysiology and school medicine - nutrition of children and socially significant diseases, problems of training of chronically ill students, assessment of working capacity and stress in different age and professional groups, etc. Since 2000 she has been a PhD. More than 50 scientific and research papers have been published in Bulgaria and abroad.



Chief Assist. Dr. Sabina S. Sabeva, PhD, graduated from the Medical Academy in Sofia in 1982. She started her professional career as a pediatrician in Sliven. From 1989 to 2002 she is an assistant - chief and senior at the Department of Hygiene, Ecology and Prof. Diseases at the Medical Faculty - Sofia. He has been a PhD since 1999. She holds a Master's degree in Public Health from Geneva University in 2002. She has over 40 scientific publications and reports in Bulgarian and foreign scientific journals and congresses.



Ass. Prof. Dencho Tsonevski, DSci. He graduated in medicine in 1961 at the Medical University - Plovdiv. PhD happened in 1974. From 1979 he was Ass. professor and Head of the Department of Hygiene and Occupational Diseases at the Medical University - Varna (branch - Dobrich). From 1985 to 1990 he was Director of the "Prevention and State Sanitary Control" at the Ministry of Health, and from 1990 he is Associate Professor at the Department of Hygiene, Ecology and Prof. Diseases - Sofia. Doctor of medical sciences - 1996. In 1997 he was elected an extraordinary professor at the University of Ruse. His research is related to the combined effects of pesticides and the toxicology of heavy metals and nitrites. He authored over 170 scientific publications in the fields of hygiene, ecology and toxicology. Co-wrote 10 monographs and textbooks on hygiene and occupational diseases.



Prof. Dr. Margarita Koleva, DSci., graduated in medicine in 1970 at the Higher Medical Institute - Kharkiv. Since 1976 is assistant in the Department of Hygiene - Sofia. Since 1990 - Associate Professor, 2006 - Professor PhD from 1976; DSci - 2004 Member of Medichem (Occupational and Environmental Health in the Chemical Industry), EAA (European Anthropological Association), EASO (European Association for the Study of Obesity), BASORD (Bulgarian Association for the Study of Obesity and Related Diseases), member of the leadership of the Bulgarian lipid league, Bulgarian Society for Hygiene, Vice Ch. editor of the magazine. "Hygiene and Public Health".

Her scientific works are mainly in field of toxicology, occupational medicine and professional diseases, the assessment of working conditions and health risk, anthropological characteristic and obesity in workers, toxicological assessment of heavy metals, pharmaceuticals, pesticides and organic solvents.

She has authored more than 130 scientific publications in Bulgaria and abroad, co-author of monographs, textbooks and manuals.



Ass. Prof. Anna K. Mihailova, PhD, graduated from the Higher Medical Institute, Sofia. Since 1975 he has been a research associate at the National Center of Hygiene, Department of Production Toxicology, PhD since 1975, Associate Professor - 1993. She is a member of NS in Hygiene the Union of Scientists, MEDICHEM, the Society of Pharmacology and Toxicology. Works in the field of: experimental toxicology: study of the toxic effects of chemicals, pesticides, biopreparations and drugs on critical organs and systems, on different routes of administration and modes (monotonous and intermittent), isolated or in combination, toxicokinetics, action and cumulation and adaptation processes; risk assessment for workers in the impact of work environment factors, screening to detect early abnormalities in the worker's liver status. Her scientific output covers more than 140 publications, monographs, scientific conferences, and symposia.



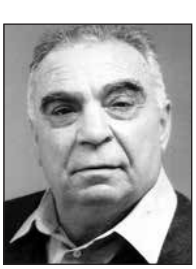
Prof. Emil Vodenicharov, PhD, graduated in medicine at the Institute of Medicine - Sofia in 1980. He is a graduate of the Department of Hygiene, Ecology and Occupational Diseases, Medical University, Sofia since 1981, since 1984 is an assistant since 1989 - Chief Assistant in the Department, since 2004 - Associate Professor, and since 2008 - Head of the Department. Professor -2013.

Since 1984, he has been PhD. In 1996-1997 he specialized in France. There are two recognized rations in the field of experimental medicine.

His scientific interests and publications are in various areas of hygiene, but mostly in occupational medicine, experimental medicine, and the impact of the dust factor in the work environment. More than 100 scientific papers and contributions to monographs and textbooks have been published and reported.



Ass. Prof. Maya Lyapin, PhD graduated in medicine at the Higher Medical Institute - Sofia in 1989. Since 1996 she has been an assistant at the Department of Hygiene, Ecology and Occupational Diseases, Medical University, Sofia. In 2015 PhD. In 2017 Ass. Prof. She has specialized in Sweden - Caroline Institute and Belgium. Her scientific interests and publications are in the study of the effects of physical and chemical factors on the working environment on neutrophil function and activation, immunology and immunotoxicology. She has specializations in occupational medicine and clinical immunology.



Prof. Rachamin Shekerdjiyski, DSci, graduated from the Pharmaceutical Faculty at the Medical Institute Sofia in 1956. Since 1973 he has been an Assistant at the Department of Technology of Pharmaceuticals at the Faculty of Phar-

macy. PhD - 1972. DSci - 1990. Habilitated in 1980. Professor - 1992. Head of Department of Industrial Pharmacy - 1992 - 2002. Dean of Pharmaceutical FacultyAuthor of over 150 scientific publications, 2 textbooks and 2 monographs in the field of formulation and testing of pharmaceutical forms. Member of the European Association of Faculties of Pharmacy.



Ivan N. Denev graduated from the Faculty of Pharmacy of the Medical University - Sofia in 2003. He participated in national and international student scientific conferences. Since 2004, he has been doing research work in a Bulgarian company (Natstim) for the production of antioxidant and immunostimulating food supplements.



Assoc. Prof. Tsanka Voditscheva, DSci, graduated in medicine in 1963 at the Kharkov Medical Institute - Ukraine. Since 1973 she has been a senior and chief assistant in the Department of Hygiene - Sofia. Since 1988 she has been a senior research associate. She was a PhD in 1970 and a Doctor of Medical Sciences in 1997. Ass. Prof. in 1992. Her scientific work is in the field of community hygiene and in particular on the problems of hygiene normalization, atherogenic and co-toxic effect of chemical components of drinking water; environmental pollution with heavy metals and research on the transport status of the population in industrial areas; hygienic assessment and normalization of microclimate in hospital rooms and living quarters; epidemiological studies and development of a concept of the importance of chemical composition of drinking water for the spread of cardiovascular diseases in Bulgaria. There are also studies in the field of occupational hygiene, child health and prevemtive nutrition. She is the author of over 200 scientific publications, co-author of monograph, three textbooks and 6 hygiene tutorials.



Assoc. Prof. Antoaneta MANOLOVA, PhD, graduated in medicine at the Higher Medical Institute in Sofia in 1977. In 1978-82 she was a regular post-graduate student in Moscow, where she defended a PhD thesis in 1982. Since 1982 she has been a research associate in the child health direction at NCHMEN. Ass. Prof. at 2008. Specializes in the issues of maternal and child health care in France and healthcare marketing in Switzerland. Its research activity is related to the assessment of the health, environmental and social risks in children and adolescents and the development of strategies and programs for risk management and prevention. She is involved in developing legislation on child health issues, vocational training and adolescent orientation in various areas of work.



Prof. Mira Tzvetkova-Arsova, DSci, graduated from Sofia University "St. Kliment Ohridski "specialty" Special Pedagogy "in 1989. Since 1990 she has been a regular post-graduate student at the Sofia University and since 1991 an assistant of pedagogy for the visually impaired at the Department of Special Pedagogy of the Faculty of Primary and Pre-school Pedagogy of Sofia University. Chief assistant since 1997, associate professor since 2006 PhD since 1998, DSci - 2014. Professor -2016. She has specialized in Boston College and the Perkins Blind School in Boston, USA, and at institutes for visually disabled with additional disabilities in Finland. She is the author of more than 120 publications in Bulgarian and foreign specialized publications and six independent textbooks on orientation and mobility for the blind and on the education of visually impaired with multiple disabilities and a monograph. She has participated in a number of international projects with partners from USA, Germany, Great Britain and others.



Eng. Kalcho Nikolov graduated in 1963 at MEI Sofia. From 1967 to 1972 he worked as an expert at the Research Institute of Hygiene at the Ministry of Health Sofia. Since 1978 he has been head of the Department of Sanitary and Technical Expertise at NCHMEN - Sofia. In 1984 he acquired a specialty "Sanitary Engineer". Specialized in the World Health Organization in France, Belgium, Italy in 1971-73. He passed an Environmental Assessment Course organized by the US Environmental Protection Agency in 1993. His department carried out at national level expert activity in the preliminary sanitary control of the hygienic requirements and rules in the project documentation. Eng. Nikolov participated in the preparation of environmental and health assessments in the development of hygiene standards. He is a representative of the Ministry of Health in expert councils of ministries and departments, participates in the training of NCHMEN in the training and postgraduate qualification of medical and technical specialists from the preliminary sanitary control.



Assoc. Prof. Dr. Nedyalka Koleva Stamova, PhD, graduated from the Medical Institute in Sofia in 1963. PhD since 1969. In 1986 she became an Associate Professor at the Department of Hygiene - MU - Sofia. The main and scientific interests are in the field of occupational medicine. She is the author of more than 100 scientific papers, co-author and editor of three textbooks, four guides, one monograph.



Chief Assist. Nina Gincheva, PhD, graduated from the Higher Institute of Medicine - Sofia in 1966. In 1967-1968, Head of Department of Labour Hygiene at HEI - Sofia, branch of Blagoevgrad. Since 1968, she has been assigned to a regular postgraduate at Laboratory of Industrial Toxicology in the Department of labour Hygiene at the Institute of Hygiene of Labour and Prof. Diseases at the Academy of Medical science - USSR. PhD - 1972 In the same year she was appointed as a research fellow at the National Center of Hygiene, Section "Production Toxicology". Since 1978 he has been Ch. assistant at the Department of Hygiene at the Higher Medical Institute - Sofia. She has a specialty in occupational medicine. Its main scientific interests are in the field of occupational medicine - production and experimental toxicology and labour hygiene in industries of vari-

ous branches. Her occupational hygiene and epidemiological studies are mainly in the chemical and petrochemical industries - organic synthesis, polymers, synthetic fibers, etc. There are over 90 publications and reports in Bulgaria and abroad. Co-authored in 2 manuals and 2 hygiene textbooks.



Dr. Ivan N. Paunov, PhD, graduated in medicine in 1976 at the Medical Institute in Sofia. From 1979 to 1994 he was a research associate at the Transport Medical Institute - Sofia. Doctor of Medicine since 1990. From 1994 to 1998 he was Director of Transport HEI. Occupational Hygiene Specialty since 1980. There are about 80 scientific publications in Bulgaria and abroad in the field of transport hygiene, impact of noise, vibrations and infrasound on transport drivers.



Ass. Prof. Lilanda T. Elenkova, PhD, graduated from the Faculty of Law at the Sofia University in 1977. She defends her PhD in 1986. Ass. Prof. in 1991. She specialized in economic management in 1992. Chief Legal Advisor of the University hospital "Ivan Rilski ". She has participated in the development of laws and regulations in the field of health and social security.



Prof. Nevena Tsacheva - Hristova, PhD, graduate in medicine at the Higher Medical Institute - Sofia in 1976. Since 1978 she has been an assistant in Medical Institute - Pleven, Department of Social Medicine. Since 1983 she has been a research fellow at the Institute of Hygiene and Occupational Diseases. PhD since 1987, Ass. Professor since 1991. Professor in 2008 at the Faculty of Public Health at the Medical University - Sofia. Since 1994 he has been Head of the department "Organization of the Scientific, Educational and Methodological Activities" of the NCHMEN and since 2000 has been the Head of the National Register of Professional Morbidity.



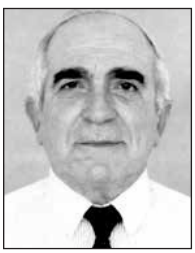
Ass. Prof. Veselin Bliznakov, PhD, graduated in medicine at the Higher Medical Institute - Sofia in 1969. Since 1975 he working at the National Center for Radiobiology and Radiation Protection, Head of the Department of Medical Radiological Protection and Epidemiology at the NCRRP. He has specialized in Russia, Austria, France and the Czech Republic. PhD since 1981, Associate Professor since 1986. He has over 100 scientific publications in the field of radiation medicine and protection, nuclear energy, uranium mining, radiation carcinogenesis and epidemiology, health monitoring, etc. Chairman of the Bulgarian Nuclear Society and the Bulgarian Scientific Society for Radiobiology and Radiation Protection. He is a member of the Managing Board of the European Nuclear Society and the Management Board of the Bulgarian Atomic Forum.



Chief Assist. Latinka G. Nakova graduated from Medical University in Sofia in 1972. Since 1975 she has been an assistant at the Clinic of Occupational Diseases at the Department of Hygiene and Occupational Diseases at MA Sofia. There are two clinical specialties - neurology and occupational diseases. Her scientific interests are focused on the problem of neuro-vegetative reactivity and specialized sensitivity in electromagnetic influences of the working environment. In this field and other problems of professional neurology she has over 50 publications in Bulgaria and abroad. She has specialized in France - Paris.



Ass. Prof. Veneta Kostova, PhD, graduated from Medical University in 1971. Since 1974 she has been a regular post-graduate at the Clinic of Occupational Diseases - Sofia. In 1980 he defended his doctorate on "Some Changes in the Hypothalamus-Pituitary-Adrenal Cortex System in Patients with Vibrational Disease". Since 1981 she is a research associate, since 1998 - Ass. Prof. and Head of the Department of General Neurology and Occupational Disabilities of the Nervous and Musculoskeletal System at the CPD - Sofia. Her scientific interests are in the field of occupational nerve diseases and vibrational disease. She is the author of more than 100 scientific publications and scientific reports. She participated in the writing of 3 textbooks and guides on occupational diseases and 1 independent monograph.



Ass. Prof. Dr. Anton St. Savov, PhD, graduated in medicine in 1968 at the Higher Medical Institute - Varna. Since 1976 he has been assistant in the Clinic of Occupational Diseases at the Center of Hygiene - Department of Occupational ORL Diseases and Otoneurology. PhD since 1979. Since 1992 he has been Head of the Clinic of Occupational ORL Diseases and Otoneurology at the University Hospital "St. Ivan Rilski "- Sofia, and since 2000 he has been head of the Center for Occupational Diseases at the Medical Faculty - Sofia. His scientific work is mainly in the field of clinical, diagnostic, expertise, prophylaxis and rehabilitation of occupational and para-professional diseases of the upper respiratory tract and ears. He has written more than 120 scientific publications, participations in monographs, textbooks, and guides in the field of professional pathology.



Prof. Elisaveta Petrova, DSci, graduated from the Medical University in Sofia in 1973. Since 1980 she has been senior assistant assistant and Ass. Professor at the Clinic of Pulmonary Occupational Diseases at the National Center of Hygiene and occupational diseases. Head of the Clinic of Occupational Diseases at the University Hospital "St. Ivan Rilski" at 1997. She has written over 130 scientific publications in Bulgaria and abroad, 3 independent monographs and over 70 papers at scientific congresses, conferences and symposia. She is a specialist in "pneumology and phthisiatry" and "occupational diseases". PhD in medicine since 1988, DSci - 2005, professor - since 2007. She has taken part in qualification courses in Poland, USA and Bulgaria. Member is the Scientific Society for Lung Diseases and the Hygiene Society.



Prof. Dr. Todor Burilov, DSci. He graduated in medicine at the Higher Medical Institute - Sofia, in 1951. From 1965 to 1989 he worked as a research associate - professor in the Clinic of Occupational Diseases at the Hygiene Center. PhD (1961) and Doctor of Medical Sciences (1983). Works in the field of occupational pulmonary diseases, silicosis, silicotuberculosis, asbestosis, exogenous allergic alveolitis and granulomatosis. Specialized in Germany and Poland. Author and co-author of six monographs in the field of pulmonary pathology. There are over 200 scientific publications in Bulgaria and abroad. Foreign member of the German Society of Ecology. Awarded by the French Medical Academy.



Chief Assist. Dr. Krasimira Dinkova, PhD, graduate in medicine at the Higher Medical Institute - Sofia in 1974. Since 1977 she has been a post-graduate of occupational diseases at CPD in NCHMEN. Since 1980 she has been an assistant in clinic of occupational lung diseases. PhD since 1987. Since 1996 he has been head of a intensive care department in COPD at "St. Ivan Rilski" hospital. She has more than 60 publications in Bulgaria and international congresses, mainly related to occupational diseases of the respiratory system. Co-author of textbooks, guides and monographs on hygiene and occupational diseases.



Ass. Prof. Dr. Lilyana Dimitrova, MD, graduate from the Medical Institute in Varna in 1974. She was a 1974 trainee-assistant of occupational diseases and a regular assistant at the Clinic of Occupational Diseases MU - Varna since 1977. PhD since 1984. Since 1988 she has been Head of the Clinic of Occupational Diseases at Medical University - Varna. Her main research work is on the problems of heavy metal intoxication and occupational pulmonary and allergic diseases.



Chief Assist. Dr. Daniela G. Medzhieeva, PhD graduated in medicine at the Medical Faculty in Sofia in 1986. Since 1988, she has worked as a research associate at the Department with Clinic of Occupational Diseases at the Scientific Center of Hygiene in the Clinic of Occupational ORL Diseases. PhD -2002. She is a member of the European and Bulgarian Society of Otorhinolaryngology and the Headache Society. There are publications and reports in Bulgarian and foreign magazines and congresses in the field of professional ORL and otoneurological diseases.



Prof. Dr. Venceslav Hr. Bosnev, DSci He graduated from Medical University in 1952. Since 1960 he has been working in the field of neurology - neurovegetology, neural therapy, diseases of kinematic hand-arm chain. He created a Laboratory of Clinical Neurovegetology at the First Neurological Clinic at the Alexandrovska Hospital and later at the Neurology Clinic at the University Hospital "St. Ivan Rilski ". He was the head of the Clinic of Occupational Diseases at the Medical Institute in Sofia and a national consultant on occupational diseases. He is the author of 240 scientific papers, including 4 monographs and 20 collective monographs. President of the Atlantic-Euro-Mediterranean Academy of Medical Sciences, member of American, European, Bulgarian scientific societies and editorial colleges. Specialist in neurology, occupational diseases, neurovegetology.



Chief Assist. Dr. Gergina Oncheva, PhD, graduated in medicine in 1982 at the Medical Faculty - Sofia. She has been working as a specialist researcher at the Electrophysiology Laboratory at the Institute of Brain Research at the Bulgarian Academy of Sciences and since 1988 has been a post-graduate student at the Neurological Clinic of the Department for Occupational Diseases in Sofia. PhD and Chief Assistant at the Neurological Clinic since 1991. She is the author of more than 40 scientific papers and a co-author in the monograph on occupational diseases.



Prof. Ivan Dikov, DSci, graduated from the Higher Medical Institute - Sofia in 1956. Since 1968 he has been working in the department of Inf. diseases, epidemiology and parasitology as an assistant, associate professor and professor. Doctor of medical sciences since 1985. Specialized in Lyon and Paris. Honorary Assistant of the Faculty of Medicine in Lyon. Many years lecturer, and researcher in infectious diseases. There are over 180 scientific publications, 10 monographs and three textbooks.



Assoc. Prof. Dr. Violina Boeva-Bangyozova, PhD, graduated from Higher Medical Institute "I. P. Pavlov "- Plovdiv in 1965. Since 1972 she has been a research associate in medical parasitology at NCIPD. From 1991 to 2003 she was head of the Parasitology Department at the Department of Infectious Diseases, Epidemiology, Parasitology and Tropical Medicine at the Sofia Medical Faculty. PhD since 1979, senior research associate since 1988. She has more than 130 scientific publications, 4 monographs and 6 textbooks in the field of clinical parasitology. She has specialized in Moscow, Athens, Hanoi.



Ass. Prof. Dr. Maria Demirova, PhD, graduated from Medical University in 1967. Since 1968 she has been working as a research associate at the Clinic of Occupational Diseases at the National Institute of Hygiene and Professional Diseases in Sofia. PhD since 1977, Ass. Prof. in occupational diseases since 1985. Since 1990 he has been the head of a clinic for occupational pulmonary diseases, since 1996 - of the Center for Occupational Diseases at the Medical Faculty - Sofia. Republican Consultant at the MH, for prof. pathology in 1995.



Prof. Dr. Vanko Nichev, DSci. graduated from medicine at the Higher Medical Institute - Sofia in 1953. Since 1959 he has been an assistant at the Institute of Pulmonary Diseases - Sofia. PhD of 1972, Doctor of Medical Sciences-1987. Associate Professor-1975, Professor-1988. Since 1984, he has been Head of the Clinic of Pneumophthisiatry and, Director of the Institute of Pulmonary Diseases - Sofia, since 1994 is the Director of the Pneumophthisiatric Hospital - Sofia. National consultant of the Ministry of Health in pneumology and phthisiatry. Author and co-author of over 200 scientific publications, monographs, textbooks and guides in the field of pulmonary pathology.



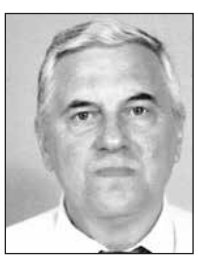
Dr. Daniela Nikolova-Baltadzhieva, PhD, graduated in Medicine at the Medical Academy in Sofia in 1982. From 1986-1991 she was a researcher at the Laboratory of Allergies at the NCIPD Medical Academy. PhD since 1997. Since 1999 he has been working at the Central Laboratory of Clinical Immunology at the University Hospital "Alexandrovska". Specialized Immunology and Allergology in Belgium, USA and Portugal.



Chief Assist. Dr. Yuri Simeonov graduated in medicine at the Medical Faculty in Sofia in 1983. Since 1986 he has been an assistant, and since 1996 has been a Chief assistant at the Department of Propedeutics of Internal Diseases at the Medical University - Sofia. He has clinical specialties in internal and lung diseases. His scientific interests and publications are in the field of diagnostics and therapy of bronchial asthma and atypical pneumonia. Member of the Scientific Society of Pneumology and Phthysiatry.



Prof. Dr. Todor Meshkov, DSci. Born in 1935, he graduated in Sofia in 1960. Since 1963, after a competition, he has been a research fellow at the National Center of Hematology and Transfusiology (III, II and I degree). In 1980 he was habilitated as Sen. res II deg., and in 1988 as Sen. res. I degree. He was elected Professor of Hematology in 1991. Director of the National Center of Hematology and Transfusion (1989-1997). He defended Ph.D. in 1976 and Doctor of Medical Sciences in 1988. He is the author of the Atlas of Hematology, 5 monographs and over 100 scientific papers on haematological cytology, bone marrow regeneration, preleukemic conditions (myelodysplasia), problems with leukemias and lymphomas, membrane changes in drug resistance. He has specialized in Germany, Poland, Russia and the United States. Member of Bulgarian and foreign scientific societies.



Prof. Dr. Dimitar Todorov, DSci He graduated from Higher Medical Education in Sofia in 1962. Since 1972 he has been working at the National Oncology Center as founder and head of Lab. for oncopharmacology. PhD since 1970, Doctor of Medical Sciences - 1986, Professor - 1987. Specialized in England (London, Solsburne) and Germany (Heidelberg). He was Deputy Director and director of the NOC. His baseline studies - over 200 scientific publications, are in the field of pharmacology and toxicology of antitumor agents, liposomal carriers, molecular mechanisms of action, combined drug therapy (including molecular biotherapy), pharmacological approaches to overcoming multiple drug resistance (MDR) of tumors. He was chairman of the Bulgarian Oncology Society, member of the American and European Cancer Association, editor-in-chief of Bulgarian Oncology Magazines.



Ass. Prof. Dr. Ekaterina Stoinova, PhD, graduated in medicine in 1969 at the Higher Medical Institute - Sofia. Since 1980 she has worked as assistant - ch. assistant at the Clinic of Occupational Diseases at Medical University - Sofia. PhD - 1984, Ass. Professor and Head of Internal Medicine Clinic at the Transport Medical Institute since 1992. Scientific interests and publications - over 100 are in the field of internal diseases, professional pathology and transport medicine.



Prof. Mois Nissim Melamed, DSci. He graduated from Medical University - Sofia in 1954. In 1961 he became an internal at the ORL clinic of Medical University - Sofia. Since 1968 he has been a research associate at the Institute of Oncology. Selected for Assoc. Prof. and Professor of Otorhinolaryngology and Oncology. Doctor of Medicine and Doctor of Medical Sciences. He has more than 160 publications in Bulgaria and abroad. He participated in 8 monographs. Member of the Scientific Society of Oncology and Otorhinolaryngology.



Dr. Hristo Damyanov, PhD, graduated in 1974 at the Higher Medical Institute - Sofia. Since 1980 he has been working in a urological ward at the Military Hospital - Sliven. From 1984-1993 he leads a urological department in the Military Academy - Sofia. Since 1994, he has been chairing a urological department at the National Cancer Center. PhD - 1998 Member of the National Society of Urology - Bulgaria, World and European Urology Associations, Balkan Cancer Association. Scientific studies in the field of urooncology - kidney and bladder tumors, prostate cancer, immunotherapy, endourology.



Prof. Dr. Petar Mihaylov, PhD, graduated from the Medical Faculty of the Sofia University in 1945. Scientific associate at the Institute of Dermatology and Venereology, 1951; PhD - 1960; Associate Professor - 1963; Senior Research Associate - 1973; Professor - 1975 Specialized in Hungary, Russia, Germany, Switzerland. In 1975-1985 he was Head of the Department of Skin and Venereal Diseases and Director of the Scientific Institute of Dermatology and Venereology at the Medical Academy - Sofia. Prof. Mihailov has been a

long-standing national consultant in dermatology and venereology. His scientific interests are in the field of allergy and professional dermatoses, medical cosmetics, climatic treatment, sexually transmitted diseases. He has published more than 350 scientific papers - messages, monographs, textbooks and guides. He is the author of the first monograph in Bulgaria "Professional Skin Diseases". President of the Bulgarian Dermatological Society, honorary member of the societies of France, Austria, Germany, Poland, Israel and others.



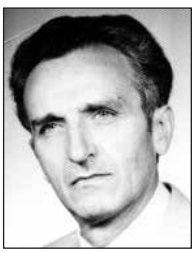
Chief Assist. Dr. Dilyana Nyagolova - Smilova graduated from Medical University in Sofia. For more than 30 years she has worked in the Department of Dermatology and Venereology as an assistant, senior assistant, chief assistant. Author of more than 50 scientific articles, guides and textbooks in the field of dermatology and venereology. There are studies mainly in the field of sexually transmitted diseases - their clinical course, diagnosis, treatment and prophylaxis, social significance.



Chief Assist. Dr. Reni Toncheva graduated from the Medical University - Sofia in 1982. Since 1988, she has been an assistant, senior assistant and chief assistant at the Clinic of Occupational Diseases at the Department of Hygiene and Occupational Diseases. She has participated in over 250 scientific publications and reports in Bulgaria and abroad in the field of occupational diseases of the eye. She has specialized in Belgium, Hungary, Italy. He is a member of the Societies of Eye and of Ergoophthalmology, Society of the International Eye research Association.



Prof. Dr. Zlatka Panova, DSci. She graduated from the Higher Medical Institute in Sofia in 1955. Since 1970 she has been working as a research associate at the Clinic of Occupational Diseases at the Medical Academy in Sofia. Ass. Professor in 1983, Professor - 1993. PhD - 1986 and doctor of medical sciences - 1992. Her research activities cover over 100 publications (scientific works, monographs, textbooks and manuals), in the field of professional obstetric-gynecological pathology mainly on the damaging effect of chemical and physical factors on genitals and female reproduction.



Dr. Ivan Grozdev, PhD, graduated from the Faculty of Dentistry in Sofia in 1951. He works for 25 years as Head of Professional Dentistry at the Clinic of Occupational Diseases of the Medical Academy in Sofia. PhD - 1971. He has over 50 scientific publications and reports of international and national congresses in the field of occupational dental diseases.



Dr. Kornelia Kotseva, PhD, graduated from the Higher Medical Institute in 1977 - Sofia. She works as a Chief assistant in the clinic of occupational diseases of the National Hygiene Center - Sofia. PhD since 1984, since 2003 is a senior researcher in the Department of Cardiovascular Medicine of the National Heart and Pulmonary Institute, London. She has developed over 70 scientific papers, co-authored in 7 monographs on risk factors, epidemiology, professional dependens, primary and secondary prophylaxis, clinical trials of new drugs for cardiovascular diseases. Member of national and international scientific societies of cardiology and occupational medicine.



Assoc. Prof. Vera Petkova, PhD, graduated from the Kharkiv Medical Institute - Ukraine in 1970. Since 1971 she has been working as a research associate in the Clinical Department of Occupational Diseases of the National Hygiene Center. In 1982, she defended in St. Petersburg dissertation on the combined effects of pesticides and vibrations in mechanics. In 1991 she was habilitated in the field of chronic professional intoxications. Since then he has been leading the Clinic for Chronic Occupational Intoxications at the Center for Occupational Diseases of Medical University - Sofia. She has over 120 scientific publications in our and foreign journals, participations in collective monographs, textbooks of hygiene and occupational diseases, participation in international scientific projects. She has specialized in Russia, Ukraine, Poland, USA. She was a WHO expert on professional pathology.



Dorothea Tsvetkova graduated in Biology - Gene and Cell Engineering at Sofia University "St. Kliment Ohridski "in 2000. Since 2001 he has been working in the Department of Structural Biology at Memorial Sloam Kettering Cancer Center, New York. There have been developments and publications in the art of elucidating the structure of receptor proteins involved in tumor angiogenesis and testing of active macromolecules to inhibit tumor growth.



Ass. Prof. Ivanka Kirova, PhD, graduated from Higher Medical Institute-Sofia, in 1961. Since 1991 she is Ass. Professor at the Department of Physiotherapy and Rehabilitation at Medical University - Sofia. Specialized Physical Medicine in Poland. Author of more than 100 scientific papers, 10 monographs and textbooks. He works in the field of treatment and prophylaxis with natural physical factors, reflexology (especially acupressure), physical therapy of occupational and internal diseases.



Ass. Prof. Margarita Neykova, PhD, graduated in 1973 from the Faculty of Kinesitherapy at the NSA and started work at the Department of Physiotherapy and Rehabilitation of MA - Sofia. PhD - 1982, Ass. Professor since 1987. He has over 100 scientific studies in kinesitherapy, co-author of monographs and textbooks. She is author of 1 invention and 7 rationalizations for new diagnostic and therapeutic methods and apparatuses in medicine.



Prof. Tanya Kuneva, PhD, graduated in medicine at the Higher Medical Institute - Varna in 1976. In 1978 she joined the Clinic of Occupational Diseases as a post-graduate student. He has been a PhD since 1981 and since then has worked as an assistant, Senior and Chief Assistant at the Clinic of Chronic Occupational Intoxications at CPD. In 1986 she was elected associate professor. Since 2000 he has been head of the Center for Occupational Diseases at the Medical Faculty - Sofia. At 2016 - Professor. She has specializations in internal diseases, occupational diseases and clinical toxicology. Her scientific work is in the field of chronic occupational poisoning. She is the author of more than 100 scientific publications, has participated in monographs, textbooks and guides.



Prof. Dr. Valentina Zlateva, DSci, graduated in Medicine at the Higher Medical Institute in Sofia in 1963. In 1970-1973 she was assistant at the Ophtalmologic Clinic at the Higher Medical Institute in Varna. Since 1974 Head of Eye department of CPD at MA-Sofia. PhD - 1977, Doctor of Medical Sciences - 1991 Associate Professor - 1983, Professor of Ophthalmology - 1998 Specialized in France, Germany, Ukraine, USA, Japan. She has developed over 160 scientific publications, monographs, textbooks and manuals on occupational ophthalmology. Member of national and international ophthalmic scientific societies.



Chief Assist. Dr. Shiboy Kuzmov, PhD, graduated in medicine in 1988 in Sofia. From 1988 to 1990 he worked in distribution in Pernik as a workshop therapist. From 1990 to 1991, he was assistant at the Institute of Pulmonary Diseases at MA - Sofia. From 1991 to 1994 he was a post-graduate student for occupational diseases to the National Institute for Hygiene and Professional Diseases, and later also to the hospital "St. Yves. Rilski "- Sofia. PhD in 1995. Since 1996 he has been assistant III-I degree, Head of FID office. He has specializations in internal and occupational diseases. Author and co-author of scientific publications and communications, 1 guide for medical students and 2 textbooks.



Prof. Dr. Yordanka Hadjieva, DSci, graduated in medicine in 1966 at the Higher Medical Institute - Sofia. Since 1976 she has been a postgraduate at the Clinic of Occupational Diseases. Ass. Professor - 1984, Doctor of Medical Sciences since 1991, Professor of Occupational Diseases since 1996. Head of Department of Occupational Diseases Clinics at the Department of Hygiene and Occupational Diseases from 1990 to 1994. She has developed 250 scientific papers in the field of clinical and occupational diseases diagnostics (early laboratory diagnostics of chemical damages - self and combined action), physical damage, treatment of occupational intoxications, work capacity and morbidity of occupational diseases. Co-author of 10 monographs and textbooks in the field of occupational diseases. National Consultant on Occupational Diseases in 1999 - 2001 Member of Bulgarian and Foreign Scientific Societies of Occupational Diseases, Hygiene and Clinical Laboratory.



Prof. Konstanta Timcheva, DSci, graduated medicine in Medical University-Sofia in 1975. Since 1982 she has been working in a chemotherapy department at the National Cancer Center. PhD - 1982, Associate Professor of Oncology - 1997, Head of chemotherapy clinic at NOC - 2000, professor - since 2010. There are over 100 scientific publications in the field of chemotherapy at oncological diseases. She is a member of the European Oncology Research Association. European Society of Chemotherapy, New York Academy of Sciences.



Assoc. Prof. Dr. Mariana Stoynovska, PhD, graduated in medicine in 1975 at the Higher Medical Institute - Sofia. From 1979 to 1983 she was a post-graduate student at the Department of Occupational Diseases of the LSGMI - St. Petersburg. She defends a dissertation on the role of immunological processes in the clinic of noise-vibration pathology. Since 1984 Ass. Professor in the Clinic of Occupational Diseases at NCHMEN. Head of Department of Hygiene, Med. Ecology and Prof. Diseases in Medical University - Pleven at 2008. She has specialized in Portugal and the United States. Major scientific interests in the field of occupational medicine and occupational allergy diseases, where there are over 80 publications.



Ass. Prof. Sonia Pavlova, PhD, graduated in biology, in 1968 at the Faculty of Biology at Sofia University. Since 1968 she has worked as a specialist in the field of biochemistry and asistent III-I degree. At 1999 as Ass. Prof. in the Center for Occupational Diseases at the Medical Faculty - Sofia. PhD since 1982 Author of more than 170 scientific publications and reports, including studies on toxicochemical and clinical-chemical changes occurring under the influence of various professional injuries. Co-author of monographs and guides on hygiene and occupational diseases.



Ass. Prof. Dr. Rouzhka Stefanova, PhD, graduated in medicine in 1963 at the Higher Medical Institute - Sofia. Since 1967 she has been a postgraduate at the Clinic of Occupational Diseases of the Institute of Hygiene and Preofessional Diseases - Sofia. PhD since 1972, since 1973, is assistant - chief assistant in the CPD. She has a specialization in biochemistry. Since 1991 she has been habilitated as Assoc. Prof. at CPD (Medical Diagnostic Laboratory). Her scientific work - over 120 articles and reports, is mainly for clinical and laboratory research in chronic intoxications with organic solvents, pesticides, heavy metals, ecological problems and others. She is a member of the International Association of Agricultural Medicine (AMIEF) and the Balkan Society of Clinical Laboratory.



Prof. Vladimir Radulov, DSci, graduated in 1969, Sofia University "St. Kliment Ohridski specialty" Special Pedagogy. In 1976, he defended doctoral dissertation and in the period 1977-1991 he worked as a research associate in pedagogy of the visually impaired at the NIE. In 1991 he was elected Associate Professor at the Department of Special Pedagogy at the Faculty of Primary and Pre-school Pedagogy of the Sofia University. In 1999 became Doctor of Science and from 2000 was elected Professor. He is the author of more than 120 publications in Bulgarian and foreign specialized publications on the problems of training and rehabilitation of the visually impaired. He is the President of the Bulgarian Association for the Education of Visually Impaired Children and a member of the European Regional Committee at the International Council for the Education of Visually Impaired Children. He has participated in numerous international conferences and in a number of international projects with partners from USA, Germany, Great Britain, Belgium and others.



Prof. Dr. Dobrin Charakchiev, DSci, graduated from the Higher Medical Institute in Sofia in 1956. Since 1960 he has been a research fellow at the Clinic of Occupational Diseases - Sofia. Since 1972 he has been Assistant Professor and since 1991 - Professor. Since 1968 he has been PhD, and since 1989 he has been a doctor of medical sciences. He has specializations in Occupational Medicine, Occupational Diseases, Internal Diseases and Biochemistry. He has specialized in occupational diseases in Russia and the Czech Republic. There are over 200 publications in Bulgaria and abroad, 6 monographs. His work covers all major sections of occupational diseases. His main scientific theoretical contributions are in the field of homeostasis (alkaline-acid regulation in the body and its clinical significance). He is a member of the European Society of Clinical Physiology, the European Society of Pulmonology and the International Aerosol Society.



Ass. Prof. Dr. Naiden I. Mirchev, PhD, graduated in medicine in 1959 in Med. University "Iv. P. Pavlov "- Plovdiv. Since 1970 he has been working in the Department of Clinical Toxicology at NIUMA "N. I. Pirogov ". In 1976 he defended a dissertation in the field of acute and occupational poisoning with benzodiazepine preparations. Since 1977 he has been the head of the Children's Section in the Clinical Toxicology Section. Ass. Prof. from 1988, from 1991 to 1999 - head of Clinical Toxicology Section. Chief expert of Clinical Toxicology at the Ministry of Health - 1997-99 There are over 100 scientific papers in the field of toxicology.



Ass. Prof. Dr. Stanka G. Andonova, PhD, graduated from the Higher Institute of Medicine - Plovdiv in 1963. She works in the district health service, then in the intensive sector of the First Municipal Hospital - Plovdiv. Since 1973 she has been an assistant at the Higher Medical Institute - Plovdiv. PhD in 1982. Habilitated as Ass. Professor and Head of Occupational Diseases Clinic, Plovdiv, since 1984. She has specialized in internal diseases, occupational diseases and clinical toxicology. She has over 100 scientific publications, participation in scientific forums and monographs on toxicology.



Chief Assist. Diana Apostolova, PhD, graduated from Higher Medical Education at the Medical Academy in Sofia in 1979. Since 1999 she has been working in the Clinic of Occupational Diseases at the Medical University in Sofia. He has specializations in occupational medicine and occupational diseases. PhD since 2000. Her research activities cover problems of the combined effect of the working environment factors (ionizing and non-ionizing factors) on the health of workers from the metallurgical, chemical and uranium mining industry.



Prof. Dr. Alexander Monov, DSci, graduated from the Medical Faculty in Sofia. He is the founder of the Bulgarian School of Clinical Toxicology, the Scientific Society and the National Toxicology Center (at the Pirogov Institute), whose head has been for 30 years. He has over 200 scientific publications and monographs in the field of emergency medicine, poisoning and allergies , ecological disabilities of man. Chairman and member of the management of scientific societies of medicine and clinical toxicology in Bulgaria and abroad, chief editor of scientific medical journals.



Prof. Dr. Iskra Altinkova, DSci, graduate in medicine at the Higher Medical Institute - Varna in 1974. Since 1974 she has been working as an assistant of immunology at the Department of Nephrology, NINUTH, Medical Academy - Sofia and since 1977 as a research associate of Clinical Immunology at the Department of Internal Medicine, NIID, MA - Sofia. In 1984, she defended PhD, and in 1998 DSci. Since 1994 she has been the Head of the Clinical Immunology Laboratory at the University Hospital "St. Ivan Rilski ". Since 1999 he has been a professor at the Department of Clinical Laboratory and Immunology, Medical University - Sofia. She has specialized in Great Britain, Austria, Russia, Germany, France and Belgium. She is a member of the European Association of Nephrology, Dialysis and Transplantation. Her scientific interests and publications are related to the development and implementation of new, modern diagnostic immunological methods in nephrology and gastroenterology practice and to the study of the pathogenetic mechanisms of chronic glomerulonephritis and inflammatory diseases of the kidneys. There are over 100 scientific publications in our and foreign magazines.



Prof. Dr. Zhelyazko Iv. Hristov, PhD. He graduated in medicine in 1976 at the Higher Medical Institute - Plovdiv. Since 1982-1996 he has been assistant

- Chief Assist. at the Higher Medical Institute - Plovdiv. PhD since 2005, Associate Professor - 2006, Dean of FPH at MU - Plovdiv - 2007. Professor - 2008. Since 1997 he has been Chairman of CIUB. There are over 100 scientific publications, the latest of which in the field of stress. Specializing in social and medical problems related to work in the USA, Argentina, France.



Ass. Prof. Dr. Andrey Milchev, PhD, graduated from the Medical University in Sofia in 1988. Since 1992 he has been a researcher at the Radiation Medicine Laboratory of the National Center for Radiobiology and Radiation Protection. He has specializations in internal diseases and radiobiology. He has specialized in Italy and the Czech Republic. His scientific publications are in the field of health monitoring and occupational disabilities of persons working in the environment of ionizing radiation. He is a member of the Scientific Society of Radiobiology and Radiation Protection.



Prof. Dr. Assen Petkov Petkov, DSci. He graduated in Medical University in Sofia in 1979. Since 1983 he has been in the Military Medical Academy and graduated from the academic positions as Assistant, Assistant, Chief Assistant, Ass. Professor (1998) and Professor (2010). PhD since 1988, Doctor of Medical Sciences since 2009. There are a number of publications on military medicine, the organization of military healthcare and health management in national and foreign scientific journals. He has specialized in Austria, USA, Germany and others. He participated in NATO, EU and NATO conferences and workshops in Bulgaria and abroad, related to the military provision of troops. Head of Department of Military Medicine since 1998. Deputy Chairman of the Bulgarian Scientific Society for Military Medicine. President of the Bulgarian National Board and Member of the Board of the Balkan Military Medical Committee since 2003, Member of the Association of American Military Doctors.



Dr. Andrey Rangelov Galev, PhD. graduated in Medicine at the Medical Academy in Sofia in 1986. Head of Medical Service at outfit 26620 - Sliven (1989-1990). From 1990 to 2004, he worked at the MMA Infectious Diseases Clinic, where he served as assistant, head of department, and clinic head. Since 2004 he has been Head of the Center for Military Epidemiology and Hygiene. In 2003 he defended dissertation. In 2004 he was elected expert of the Republic of Bulgaria to the UN on the issues of combating bio-terrorism. He has specialties in skin and venereal diseases, infectious diseases and tropical medicine. Dr. Galev is the author and co-author of over 250 publications in the field of dermatology, epidemiology, prevention, clinic and treatment of infectious and parasitic diseases of social and military significance. There are publications in the field of occupational medicine. In 2010 he established a Bulgarian Association for Preventive Medicine.



Ass. Prof. Ivan Popivanov, PhD. He graduated in 1995 at Medical University - Pleven. Since 2004 he has been working at the Military Medical Academy, as a consecutive assistant, senior assistant and chief assistant in the Department of Military Epidemiology and Hygiene at the Department of Military Medicine. He has a recognized specializations in "Epidemiology of Infectious Diseases" and "General Medicine". PhD in 2013, Ass. Prof. - 2015, is scientific interests are in the field of preventive military medicine, with over 70 publications in national and international specialized periodicals and participation in textbooks and manuals. He participates in NATO and MD working groups. Member of the BNS on Epidemiology of Infectious and Non-infectious Diseases.



Chief Assist. Dr. Vlayko Vodenicharov, PhD, graduated in 2014 in Medical University - Sofia. From 2015 he worked as assistant, senior and chief assistant in Department of Hygiene, med. ecology and nutrition of Medical University - Sofia. PhD - in 2018. He participate in scientific conferences of labour medicine in Fukuoka - 2014, and Groningen - 2015; of bioethics - Basel and Napoli - 2013. There are more of 30 scientific publications in bulgarian and foreign journals. Chief editor of scintific magazine "Praemedicus".

CONTENT

FOREWORD **27**
A. OCCUPATIONAL HYGIENE **29**
1. DEVELOPMENT AND PRESENT STATE OF THE ORGANIZATION, FORMS AND WORKING CONDITIONS
D. Tsvetkov **29**
1.1 Evolution of concepts for organization (structure of tasks) of labour **31**
1.1.1. Earlier systems of labour organisation 31
1.1.2. The movement for "humane treatment" 32
1.1.3. Restructuring tasks 33
1.1.4. The importance of the physiology of labour and ergonomics 33
1.1.5. Electronics and modern labour 34
1.2. Factors of working environment **36**
1.2.1. Work environment factors – industries and professions 36
1.2.2. Work environment - health status and performance 37
1.2.3. Hygiene standardization and health risk 38
1.3. Forecasts for the development of postindustrial society **41**
1.3.1. Industrial society 41
1.3.2. To postindustrial society. 42

2. PHYSIOLOGY OF LABOUR
E. Dincheva **43**
2.1. Physical work **43**
2.1.1. Types of physical work 43
2.1.2. Categorization of labour 44
2.1.3. Changes in organs and systems during physical work 45
2.2. Non-physical or mental work **48**
2.2.1. Classification of mainly intellectual work 48
2.1.2. Changes in organs and systems in mental work 49
2.3. Working capacity **50**
2.4. Fatigue **53**
2.4.1. Classification of fatigue 53
2.4.2. Overwork 54
2.4.3. Signs and symptoms of fatigue 54
2.4.4. Prevention of fatigue and overwork 55
2.5. Professional stress **56**
2.5.1. Work-related stressors 57
2.5.2. Symptoms of stress. Other stress-similar states. 58
2.5.3. Stress and illness 59
2.5.4. Prevention and stress management 60
2.6. Modern approaches and methods to assess the state of mental stress and intervention
Zhel. Hristov, B. Tsenova **62**

2.6.1. Modern psychological theories of occupational stress 62
2.6.2. Practice for research on workplace stress 63
2.6.3. Studies in Bulgaria 65
2.6.4. Strategies and policies to limit stress - modern practices 66
2.6.4.1. Monitoring of stress 66
2.6.4.2. Types and levels of intervention in companies and organizations 66
2.6.4.3. Fighting burnout syndrome 67
2.6.4.4. Prevention of mental violence (mobbing) 67
2.6.4.5. Proactive approach - an overall policy to limit and reduce stress 68

3. PSYCHOLOGY OF LABOUR
B. Tsenova....70
3.1. Stages of development by psychology of labour 70
3.2. Criteria for psychological assessment of work activities 71
3.3. Methods for psychological analysis of the content of the work and its impact on humans 73
3.4. Methodological approaches to psychological analysis of labour 74
3.5. Psychological states at work 75
3.6. Psychological study of occupations 79
3.7. Professional success and motivation to work 82
3.8. Psychosocial factors of labour and health....83

4. ERGONOMICS
Z. Ivanov....86
4.1. Workplace and anthropometric characteristics of man. 86
4.2. Workplace and working posture. 89
4.3. Working furniture and tools 94
4.4. Working movements 94
4.5. Means of presenting the information (MPI) and controls (MC) 95
4.6. Lighting and vision 97
4.6.1. Elements of physics 97
4.6.2. Physiology and functional possibilities of vision in labour....98
4.6.3. Optimization of visual activity at work. 102
4.6.4. Lighting in the workplace 104

5. BIOLOGICAL RHYTHM AND REGIMEN OF WORK AND REST
Z. Ivanov....108

6. PHYSICAL FACTORS....112
6.1. Thermal conditions of environment (microclimate)
Ft. Kalburova, D. Tsvetkov 108
6.1.1. Thermal factors.... 112
6.1.2. Thermal condition of the body 113
6.1.3. Influence of thermal conditions – pathological disorders and diseases. 117

6.1.4. Hygiene regulation of thermal conditions 117
6.1.5. Prevention of adverse effects of uncomfortable microclimate 118
6.2. Non-Ionizing radiation 121
6.2.1. Infrared radiation
F.Kalburova, D. Tsvetkov 122
6.2.1.1. Biological effects 123
6.2.1.2. Hygiene normalization 124
6.2.1.3. Prevention of adverse biological effects 124
6.2.2. Ultraviolet radiation
D. Tsvetkov 127
6.2.2.1. Sources to UV-radiation 127
6.2.2.2. Absorbtion and general biological action 128
6.2.2.3. Health effects (risk) under irradiation with UV-rays 129
6.2.2.4. Endangered professions 131
6.2.2.5. Hygiene standards 131
6.2.2.6. Prevention of adverse effects 132
6.2.3. Radio-frequency electromagnetic fields (radio waves)
B. Stefanov 134
6.2.3.1.General characteristics 134
6.2.3.2. Main sources of radio waves 134
6.2.3.3. Biological action. Clinical manifestations 135
6.2.3.4. Prevention of damages 136
6.2.4. Low-frequency electromagnetic radiation. Magnetic field
B.Stefanov 138
6.2.4.1. General characteristics 138
6.2.4.2. Main sources 138
6.2.4.3. Biological activity 138
6.2.5. Static electricity
B.Stefanov, D. Tsvetkov 141
6.2.5.1. General characteristics 141
6.2.5.2. Main sources 141
6.2.5.3. Biological action 141
6.2.5.4. Prevention 142
6.2.6. Laser
D. Tsvetkov, M. Lyapina 144
6.2.6.1. Use of lasers 144
6.2.6.2. Occupational risk factors 144
6.2.6.3. Classification of lasers in the degree of occupational risk. 145
6.2.6.4. Biological action 145
6.2.6.5. Prevention of laser impact 146
6.3. Air ionization
Ft. Kalburova 150
6.3.1. Main characteristics and sources 150
6.3.2. Ionization of the air environment of indoor and outdoor 150

6.3.3. Biological effects of air ionization 151
6.3.4. Prevention of adverse effects. 152
6.3.5. Artificial ionization of air. Ion generators. 153
6.4. Noise
N.Mitrev, D.Tsvetkov, Zl. Stoyneva 154
6.4.1. General condition, basic characteristics and sources 154
6.4.2. Specific - hearing, effects of noise 155
6.4.3. Nonspecific - extraaural, effects of noise 156
6.4.4. Prevention of adverse noise effects 156
6.5. Infrasound
N.Mitrev 161
6.5.1. Effects of infrasound on the body 161
6.5.2. Prevention of adverse effects 163
6.6. Ultrasound
N.Mitrev, D.Tsvetkov 165
6.6.1. Impact of ultrasound on the human body 166
6.6.2. Prevention of adverse effects 167
6.7. Vibration
N.Mitrev, D.Tsvetkov, Zl.Stoineva 169
6.7.1. General and basic features 169
6.7.2. Impact of local vibrations on the body 171
6.7.3. Effect of general vibration on the body 173
6.7.4. Prevention of adverse effects. 175
6.8. Reduced atmospheric pressure
D. Dimitrov, A. Agovska 179
6.8.1. Physiological effects of reduced atmospheric pressure 179
6.8.2. Pathological conditions and diseases 181
6.8.3. Prevention of altitudinal disabilities 184
6.9. Increased atmospheric pressure
D.Dimitrov, S.Sabeva 185
6.9.1. Toxic effects of increased atmospheric pressure 185
6.9.2. Other effects of the increased pressure 186
6.9.3. Decompression 187
6.9.4. Prevention of compression and decompression disabilities 189
6.10. Gravity, acceleration, and micro-gravity (weightlessness)
D.Dimitrov, S.Sabeva 191
6.10.1. Gravity and acceleration 191
6.10.2. Preventive measures 192
6.10.3. Microgravity (weightlessness) 193

7. CHEMICAL FACTORS 195
7.1. Industrial Toxicology - basic assumptions, parameters of toxicity.
Prevention of occupational poisoning
D. Tsonevsky 195

7.1.1. Factors determining the toxicity of substances 196
7.1.2. Toxicocinetics 198
7.1.3. Toxicometrics 200
7.1.4. Toxicodynamics (mechanisms of toxicity) 203
7.1.5. Prevention of occupational poisonings. General principle. 205
7.2. Metals and non-metals
M. Koleva 207
7.3. Alkalis and acids
M. Koleva 232
7.3.1. Alkalis 232
7.3.2. Acids 232
7.4. Alcohol
M. Koleva 234
7.5. Aromatic hydrocarbons
M. Koleva 236
7.6. Gases
A. Mihaylova 238
7.7. Ketones
M.Koleva 245
7.8. Nitro - and amino derivatives of benzene and toluene
M.Koleva 246
7.9. Polycyclic aromatic hydrocarbons (PAHC)
M.Koleva 248
7.10. Chlorinated hydrocarbons
M.Koleva 250
7.11. Containing Fluorine chlorine hydrocarbons
M.Koleva 252
7.12. Other organics
M.Koleva, A. Mihaylova 253
7.13. Pesticides
D.Tsonevsky, M. Lyapin 261

8. DUST AEROSOLS
E.Vodenicharov 274
8.1. Classification of the aerosols 274
8.2. Deposition of dust particles into the respiratory system 275
8.3. Chemical composition of the dust and fibrogenIC action 276
8.4. Exposure to dust and the risk of lung damage 277
8.5. Express experimental methods to determine the extent of fibrogenity and silicosis hazard of powders 277
8.6. Health risk from exposure to dust 278
8.7. Adverse health effects from exposure to dust 278
8.8. Prevention of adverse effects 279

9. CHEMICAL FACTORS WITH CARCINOGENIC EFFECT
D. Tsonevski....281
9.1. Methods for evaluation and classification 281
9.2. Mechanisms of carcinogenesis 282
9.3. Dose and health risk 282
9.4. Modifying factors 283
9.5. Carcinogens in production 284

10. FACTORS ACTING ON THE IMMUNE SYSTEM
I. Altankova, M. Lyapina....287
10.1. Immune system 287
10.2. Basic mechanisms of immune damage 288
10.3. Health effects of immunotoxicity effects 288
10.4. Allergens causing professionally-dependent sensitization 289
10.5. Immuno-dependent occupational diseases 290
10.6. Diagnosis of immune impairment 291
10.7. Impact of physical factors on the immune system
M. Lyapina, E. Vodenicharov 294
10.8. Possibilities for treatment of adverse factors by antioxidant supplements
R. Shekerdjiiski, I. Denev....298

11. HYGIENE REGULATION OF ENVIRONMENTAL FACTORS. ASSESSMENT AND MANAGEMENT OF OCCUPATIONAL RISK - APPROACHES AND MODELS
Ts. Vodichenska, D. Tsvetkov 303
11.1. Environmental factors 304
11.2. Work environment factors 305
11.3. Assessment and management of occupational risk - approaches and models. 307

12. PRODUCTION TRAUMATISM
Z. Ivanov 313

13. WORK - AGE AND SEX
E. Dincheva....316
13.1. Changes in physical performance with age 316
13.2. Changes in neuro-psychic performance with age 317
13.3. Adaptation of women to work 319
13.4. Osteoporosis
A. Manolova 322

14. PROFESSIONAL TRAINING AND WORK OF PERSONS WITH DISABILITIES
M.Tsvetkova-Arsova, Vl. Radulov....325
14.1. Vocational rehabilitation of persons with disabilities 326
14.2. Labour for people with disorders 326

15. MANUFACTURING ENTERPRISES - ARCHITECTURAL BUILDING CONCEPTS AND REQUIREMENTS. VENTILATION
K.Nikolov. 329
15.1. Architectural and planning. Solutions and requirements 329
15.2. Ventilation 332

16. PERSONAL PROTECTIVE EQUIPMENT
Z.Ivanov 334

17. MANUFACTURING AND PROFESSIONS - BASIC TECHNOLOGIES, HEALTH RISK, PREVENTION 338
17.1. Agriculture
D.Tsonevsky 338
17.1.1. Labour hygiene in plant-growing 338
17.1.2. Labour hygiene in livestock breeding 340
17.2. Mining
N. Stamova 342
17.2.1. Mining 342
17.2.2. Ore processing and dressing 350
17.3. Ferous and non-ferous metallurgy
Z.Ivanov, M.Lyapin, Em. Vodenicharov 350
17.3.1. Ferous metallurgy 350
17.3.2. Non-ferrous metallurgy 352
17.4. MECHANICAL ENGINEERING
N. Gincheva 358
17.4.1. Foundry production 358
17.4.2. Forge-press production 362
17.4.3. Mechanical machining of metals 363
17.4.4. Assembly production 365
17.5. Chemical industry
N. Gincheva 371
17.6. Textile industry. Manufacture of cotton fabrics
N. Stamova 375
17.7. Construction and production of building materials
N. Gincheva, M. Lyapina 380
17.7.1. Construction 380
17.7.2. Production of building materials 385
17.8. Labour hygiene when working with computer equipment
B. Stefanov 391
17.9. Electricity production
E. Dincheva 396
17.9.1. Thermal power plant 396
17.9.2. Hydroelectric power plants 397
17.9.3. Nuclear Power Plants 397

17.10. Transport
Iv. Paunov 400
17.10.1. Water transport 400
17.10.2. Rail transport 402
17.10.3. Metro 404
17.10.4. Road transport 405
17.10.5. Air transport 406
17.11. Chemical-pharmaceutical production. Pharmacies
M. Koleva 410
17.11.1. Production buildings 411
17.11.2. Working environment and production process 412
17.11.3. Fermentation (production of antibiotics) 416
17.11.4. Synthesis of Medicines 417
17.11.5. Biological (natural) extraction 418
17.11.6. Production of ready-to-use pharmaceutical forms 419
17.11.7. Pharmacies 422
17.12. Armed Forces
As. Petkov, An. Galev, Iv. Popivanov 426

B. PROFESSIONAL DISEASES 431

1. OCCUPATIONAL DISEASES. INTRODUCTION
J. Hadjieva, L. Elenkova 431
1.1. Occupational diseases – basic characteristics 431
1.2. Occupational disease and occupational accidents. Social Risks Provided. 437

2. EPIDEMIOLOGY OF OCCUPATIONAL DISEASES
N. Tsacheva 441

3. OCCUPATIONAL POISONING 446
3.1. Metals 446
3.1.1. Antimony - J. Hadjieva 446
3.1.2. ArsenIc - V. Petkova, S. Pavlova 448
3.1.3. Beryllium – T. Kuneva 451
3.1.4. Kadmium - T. Kuneva 453
3.1.5. Chromium - J. Hadjieva 454
3.1.6. Cobalt - J. Hadjieva 456
3.1.7. Lead - D. Charakchiev, St. Andonova 457
3.1.8. Manganese - V. Petkova 459
3.1.9. Mercury - St. Andonova 460
3.1.10. Nickel - J. Hadjieva 461
3.1.11. Phosphorus - T. Kuneva 461
3.1.12 Selenium - S. Pavlova 463
3.1.13. Tellurium - S. Pavlova 464

3.1.14. Thallium - V.Petkova 465
3.1.15. Vanadium - T. Kuneva 465
3.1.16. Zink - J. Hadjieva 466
3.1.17. TIN - J. Hadjieva 467
3.1.18. Copper - T. Kuneva 468
3.1.19. Aluminum - S. Pavlova 470
3.2. Organic solvents 473
3.2.1. Benzol - J. Hadjieva 475
3.2.2. Alcohols - R. Stefanova 476
3.2.3. Ethylene Glycol - D. Apostolova 477
3.2.4. Phenol - J. Hadjieva 478
3.2.5. Ketones - D. CHarykchiev, St. Andonova 479
3.2.6. Chlorinated hydrocarbons - D. Apostolova, J. Hadjieva 480
3.2.7. Amino- and nitro derivatives of benzene. Trinitrotoluene - J. Hadjieva 482
3.2.8. Gasoline - R. Stefanova 483
3.2.9. Carbondisulfide - St. Andonova, S. Pavlova 485
3.3. Polymers 488
3.3.1. Acrylamide - V. Petkova 489
3.3.2. Acrylonitrile - T. Kuneva 490
3.3.3. Styrene - T. Kuneva 491
3.3.4. Vinylchloride - V. Petkova 492
3.3.5. Ethylene - T. Kuneva 492
3.3.6. Methyl methacrylate - T. Kuneva 493
3.4. Pesticides 495
3.4.1. Cholinesterase-inhibiting pesticides - St. Andonova, R. Stefanova 495
3.4.2. Chlororganic pesticides - V. Petkova, St. Andonova 497
3.4.3. Mercury pesticides - V. Petkova 498
3.4.4. Dipyridyls - St. Andonova 499
3.4.5. Nitrophenols (dinitro compounds) - V. Petkova 499
3.4.6. Pyrethroids - St. Andonova 500
3.5. Toxic gases 502
3.5.1. Carbon monoxide - St. Andonova, D. Charakchiev 503
3.5.2. Hydrogen cyanide and cyanides - V. Petkova 504
3.5.3. Hydrogen sulphide - St. Andonova 505
3.5.4. Arsine (hydrogen arsenide) - T. Kuneva 505
3.5.5. Chlorine - St. Andonova 506
3.5.6. Fluorine, hydrogen fluoride - St. Andonova 507
3.5.7. Ammonia - St. Andonova 508
3.5.8. Sulfur gases - St. Andonova 508
3.5.9. Nitrogen Oxides - T. Kuneva 509
3.6. Other chemical compounds 511
3.6.1. Acids - S. Pavlova 511
3.6.2. Bases - T. Kuneva 512
3.6.3. Chloromethyl ethers - R. Stefanova 513

3.6.4. Formaldehyde - R. Stefanova 514
3.6.5. Surfactants. - R. Stefanova 514
3.6.6. Ethylene oxide - J. Hadjieva 516
3.6.7. Tetraethyl-lead - J. Hadjieva 516
3.6.8. Polycyclic aromatic hydrocarbons - D. Apostolova 517
3.7. Mass acute poisoning - toxochemical traumatism. Mass poisoning from industrial poisons
Al. Monov 520

4. OCCUPATIONAL DISEASES BY PHYSICAL FACTORS 524
4.1. Professional cryopathology
Zl. Stoineva 524
4.2. Professional thermopathology
Zl. Stoineva 528
4.3. Radio-wave disabilities
L. Nakova 531
4.4. Radiation illness
Ves. Bliznakov, A. Milchev 533
4.4.1. Acute radiation illness 533
4.4.2. Chronic Radiation Disease 537
4.5. Vibrational disease
V. Kostova 540
4.5.1. Vibratory disease from local vibrations 541
4.5.2. Vibrational disease of general vibrations 543
4.6. Professional deafness
A. Savov 547
4.7. Occupational disability of the vestibular analyzer
A. Savov 553

5. OCCUPATIONAL DUST DISEASES OF THE RESPIRATORY SYSTEM 556
5.1. Pneumoconiosis. Silicosis
E. Petrova 556
5.1.1. Pneumoconiosis. General part. 556
5.1.2. Silicosis 557
5.2. Silicotuberculosis
T. Burilkov, Kr. Dinkova 568
5.3. Asbestosis
T. Burilkov, Kr. Dinkova 569
5.4. Silicatoses
L. Dimitrova 571
5.5. Anthracosis
E. Petrova 575
5.6. Metalconiosis
Kr. Dinkova 577
5.7. Mixed pneumoconiosis
Kr. Dinkova 579

5.8. Organic powder
D. Charakchiev 580
5.9. Professional chronic bronchitis
Sh. Kuzmov, M. Demirova, Kr. Dinkova 583
5.10. Occupational diseases of URT
D. Medjidieva 587

6. OCCUPATIONAL OVERTENSION DISEASES
V. Bosnev, G. Oncheva, Zl. Stoyneva 590
6.1. Diseases of peripheral nervous system overtension 590
6.2. Vegetative polyneuropathy from overintension or microtraumatism (ICD-X-90.0) 595
6.3. Diseases of overtension of the musculoskeletal system 596
6.4. Coordination dyskinesias 599

7. PROFESSIONAL INFECTIOUS AND PARASITIC DISEASES
Iv. Dikov, V. Boeva 601
7.1. Bacterial and viral infectious diseases
Iv. Dikov 601
7.2. Professional parasitic diseases
V. Boeva 609

8. OCCUPATIONAL ALLERGIC DISEASES 614
8.1. Introduction. Classification
M. Demirova 614
8.2. Allergic diseases of the respiratory system 616
8.2.1. Allergic diseases of the upper respiratory tract
A. Savov, M. Stoynovska 616
8.2.2. Professional bronchial asthma
M. Demirova 618
8.2.3. Hypersensitive pneumonitis
M. Demirova, Y. Simeonov, M. Stoynovska, D. Baltadjieva 621
8.3. Professional allergic conjunctivitis
V. Zlateva, R. Toncheva, M. Stoynovska 624

9. PROFESSIONAL MALIGNANT NEOPLASMS 626
9.1. Oncological diseases - basic situations
D. Todorov 626
9.2. Malignant neoplasms of the respiratory system 633
9.2.1. Pulmonary cancer
M. Demirova, Y. Simeonov, E. Stoynova 633
9.2.2. Larynx, nasal and laryngeal cavities cancer
M. Melamed 637
9.3. Bladder cancer
Hr. Damyanov 641

9.4. Myeloproliferative diseases
T. Meshkov **644**
9.5. Modern approaches to chemotherapy of oncology
D. Todorov, K. Timcheva, D. Tsvetkova **649**
10. PROFESSIONAL SKIN DISEASES
P. Mihaylov, D. Nyagolova **656**
10.1. Irritant contact dermatitis **656**
10.2. Allergic contact dermatitis **657**
10.3. Phototoxic contact dermatitis **660**
10.4. Allergic contact photodermatitis **661**
10.5. Skin pigmentation **661**
10.6. Chloracne **662**
10.7. Professional dermatoses from plants (Phytodermatitis) **662**
10.8. Occupational skin infections **662**
10.9. Professionally determined skin tumors **662**

11. PROFESSIONAL DISEASES OF THE VISUAL ANALYZER **665**
11.1. Physical and chemical agents and load
V. Zlateva **665**
11.2. Acute occular traumas
V. Zlateva, R. Toncheva **672**

12. OCCUPATIONAL DISABILITIES OF FEMALE REPRODUCTION
Zl. Panova **675**

13. OCCUPATIONAL IMPAIRMENT OF THE ORAL CAVITY AND TEETH
Iv. Grozdev **679**

14. OCCUPATIONAL DISABILITIES OF THE CARDIOVASCULAR SYSTEM
K. Kotseva **682**

15. PHYSIATRICS AND REHABILITATION OF PREFESSIONAL DISEASES AND INTOXICATIONS
Iv. Kirova, M. Neykova **687**
15.1. Occupational intoxications **687**
15.1.1. Rehabilitation of professional metal poisonings 687
15.1.2. Physiotherapy and kinesitherapy in organic solvents intoxication 688
15.1.3. Physiotherapy and kinesitherapy in polymers intoxication 688
15.1.4. Physiotherapy and kinesitherapy in pesticides intoxication 689
15.1.5. Physiotherapy and kinesitherapy in irritant gases intoxication 690
15.2. Occupational diseases by physical factors **690**
15.2.1. Physiotherapy and kinesitherapy in cold angioneuropathy 690
15.2.2. Physiotherapy and kinesitherapy in thermal shock, thermal cramp, thermal exhaustion 691
15.2.3. Physiotherapy and kinesitherapy in radiowave damages 691
15.2.4. Physiotherapy and kinesitherapy in radiation disease 691

15.2.5 Physiotherapy and kinesitherapy in caisson disease 692
15.2.6. Physiotherapy and kinesitherapy in mountain sickness 692
15.2.7. Physiotherapy and kinesitherapy in vibration disease 692
15.2.8. Physiotherapy in professional deafness 693
5.3. Professional dust respiratory diseases 693
15.4. Occupational over-tension 694
15.4.1. Physiotherapy and kinesitherapy in musculoskeletal diseases 694
15.4.2. Physiotherapy and kinesitherapy in peripheral neurvous system diseases 695
15.4.3. Physiotherapy and kinesitherapy in vegetative polineuropathy at the upper limbs 696
15.4.4. Physiotherapy and kinesitherapy in occupational dyskinesias 697
15.5 Occupational allergic diseases - bronchial asthma 697
15.6. Professional malignant neoplasms 698
15.7. Occupational disorders of the visual analyzer 698
15.8 Occupational cardiovascular damage 699
15.9. Occupational disabilities of the vestibular analyzer 699

FOREWORD

Medicine includes three key areas-prevention, treatment and rehabilitation. This division is provisionally, since they are interconnected, and the main purpose of medicine is health (not disease), which determines its preventive orientation. Among numerous of medical disciplines-fundamental, clinical, rehabilitation, hygiene is the primary preventive science. It is a new science in the modern sense and at the same time one of the oldest units of human knowledge and practice related to medicine.

Like any modern science hygiene constantly enrich their knowledge and dynamic besides interacting with medical and other sciences.

The relationship of hygienic science with other preventive medical sciences (social medicine, epidemiology, microbiology and parasitology) show that unity of integration and differentiation. From one historical aspect of science, now they have their specificity in mutual relations and penetration into each other in some areas. Hygiene is directed to the health of the individual, but above all to the health of the larger population groups. Achievement of its objective-studying, preserving, strengthening and improving the health and human performance, is done by optimizing the interaction of to "man-environment", to determine the most favorable parameters of the environment and increased adaptation capabilities of the organism.

The proposed volume of Hygiene guide includes two volumes: Hygiene and ecology Volume I; Hygiene and occupational diseases (Occupational Health) Volume II. In writing they involved a large number of Bulgarian authors mainly from higher medical institutes and also authors from abroad. In his writing we have tried not only to present hygiene in its modern light, as a wide and comprehensive preventive science, but to direct its contents to more training and knowledge needs students of medical specialties and doctors.

Compared with previous textbooks of hygiene, content now is not only voluminous, but many altered. Processed entirely all sections, it is mainly emphasized changes in health (biological action, diseases, changes in the adaptation) under the action of environmental factors.

In focus-for doctors and students, and voluminous, this guide in Hygiene is the modern successor of issued more than 40 years two volumes Textbook hygiene first and second parts, ed. Prof. Dr. L. Tsvetkov (1958) and Occupational medicine ed. Prof.. Dr. M. Lukanov (1984). Now in the second volume is on and discipline professional diseases.

We are grateful for the quality requirements of higher education concerning environronment, and support the project JEP 2154-91/94 program "Tempus" with the Head Professor Andre Pavia, University of Montpellier II, France.

The guide can be used by other nonmedical professionals. Gratefully we will accept all criticism and views on quality.

Prof. Dr. D. Tsvetkov, DSci

PREFACE

La médecine comprend trois directions fondamentales-prophylaxie, traitement médical et réhabilitation. C 'est une division relative puisque ce sont des partie mutuellement liées, et le but principal de la médecine est la santé (non pas la maladie), qui détermine son orientation prophylactique. Parmi les nombreuses disciplines de médecine: fondamentales, cliniques et de réhabilitation, l'hygiène est une des moderne, mais en même temps une des branches les plus anciennes de la connaissance et de la pratique humaines, liées a la médecine.

Comme chaque science contemporaine, l'hygiène enrichit constamment ses connaissances et sa coopération avec les autre sciences de médecine ainsi que avec des sciences dans les domaines homologues.

Les relation réciproques entre l'hygiène et les autres sciences medico-prophylactiques-mèdecine sociale, èpidèmiologie, microbiologie et parasitologie, progressent surtout durant les dernières décades. Elles témoignent d'une unité d'intégration et de différenciation. Etant parties, historiquement d'une seule science, actuellement elles présent une specifite sur le fond des liens réciproques et d'une interpénétration. L'hygiène est orientée vers le santé de l'individu, mais avant tout vers celle de large groupes de la population. L'hygiène envisage la réalisation de son but – l'étude, la protection, la promotion, l'amélioration de la santé et de la capacité de travail par l'optimisation de l'interaction entre l'homme et l'environnement, c'est a dire, par l'identification les plus favourables paramètres de l'environnement et la promotion de la capacités d'adaptation de l'organisme.

Le présent manuel d'Hygiène est constitue de deux volumes: „Hygiène et écologie" – v.l et„Hygiène du travail et maladies professionnelles» (Médecine de travaille)-v.11 a ètè rédigé par des nombreux auteurs bulares – surtout des Instituts supérieurs de médecine, et d'étranger. Comparant ce cours avec les manuels précédents d'Hygiène on constate un changement ainsi bien au point de vue volume et composition que d'amélioration d'approche et de quantité. Toutes les parties sont remaniées et revissées. L'accent est mis principalement sur les changements de l'ètat de santé (influence biologique, maladies, modification d'adaptation) sous l'influence des facteurs de l'environnement.

Dans son esprit, contenu de base, but et destination le cours est un continuateur contemporain de Manuel en deux volumes du prof. L. Tzvetkov (1958-1958) et de Medicine de Travail, sous la dir. de prof. M. Loukanov (1984). Le nouveau v. Il comprend aussi la discipline maladies professionnelles.

Nous sommes reconnaissants pour les exigences a l'égard de la qualité de l'éducation supérieurs dans le domaine de l'environnement et l'assistance du Projet JEP 2154 – 91/94 du programme TEMPUS sous la direction du prof. André Pavia, l'Université de Montpellier II (France).

L'ouvrage peut être utile a nombre de spécialistes qui travaillent dans des secteurs qui ne sont pas en liens étroits avec la médecine. Les auteurs seraient contents de recevoir des critiques et de commentaires concernant les qualités de cette publications.

Prof. D. Tzvetkov, D.Sc

D. Tsvetkov

1 DEVELOPMENT AND PRESENT STATE OF THE ORGANIZATION, FORMS AND WORKING CONDITIONS

Hygiene is part of hygiene science, studying labour activity and working environment, with a view to their impact on the employees - their health condition and performance. Hygiene develops hygienic norms, rules and various preventive measures to create favorable for health and performance work conditions.

Occupational medicine is wider medical disciplines related hygiene of labour with occupational diseases and more broadly with other diseases - paraprofesionals and everyone else involved in the so-called general morbidity, i.e. occupational medicine connects preventive and curative aspects of medicine in the area of humans labour activity.

Early and late history. Hygiene of labour and occupational medicine have their beginning from the time of Hippocrates and before him. Individual ideas of occupational diseases and the role of the favorable conditions of work in the miner, metal production and the need for correct labour regimen for the preservation of health, has in ancient Egyptian and Chinese script, in the works of scientists and writers of ancient Greece and the Roman Empire in ancient Jewish collection of Judaism "Talmud". Inhabited in the Balkan Peninsula thracians (later the proto-bulgarians) have left also written and other archaeological data about high at this time health culture.

Hippocrates (Hippokrates, 460-377 to S.J.) Is described in his treatises damaging effects of dust in mining, toxicity of lead ("lead" colic). Pliny the Elder (Gaius Plinius Secundus, about 23-79 to J.C.) and Galen (Claudius Galenus, 129-201 to J.C.) are indicated harmful effects of dust, in mining of sulfur and cinnobar (mercury paint). Ovidius (Publius Ovidius Naso '43 to - and about 18 after J.C.) and Plutarch (Plutarchos about 46-127 years) have described the hazards at tanners and metallurgists, and diseases in miner described by Carr Lucretius (Lucretius Carus Titus, about 99-55 to J.C.) and Juvenal (Decimus Junius Juvenalis, about 60-127 y.).

Early Middle Ages not bring new knowledge in the field of occupational medicine. Only in 1473 Ulrich-Elenbok describes poisoning by fumes of mercury, lead and coal smoke. In the sixteenth century. **Paracelsus** (Paracelsus-Philipus Theophrastus Bombast, 1493-1541) In the three-volume work (1530) describes in detail the working conditions and occupational diseases in miners in Tyrol (acute and chronic mercurialism) and



Paracelsus (Paracelsus-Filippos Teofrastus Bombast, 1493-1541 years)

in 1556. Agricola (G. Agricola, 1494-1555 years) in his work "De re metalica", describe in detail silicosis and other dust disabilities in the e silver and gold mines Yoahimstal in the Harz mountains.

Ramazini (Bernardino Ramazini, 1633-1714 years), called the father of occupational medicine, a doctor and professor of medicine in Modena, published in 1700 his treatise "De morbis artificum diatriba" ("For diseases to craftsmen"), in which systematize all knowledge to that time in the field of occupational medicine based on about



Ramazini (Bernardino Ramazini, 1633-1714 years)

70 different professions ("diseases of pharmacists," "diseases of writers and scientists," etc.). He points out that "when the doctor visited the sick worker ... .. to Hippocrates recommended questions at examination should add another - what is your profession?". In 1742 the founder of Russian science and culture M. V. Lomonosov (1711-1765) has exhibited in detail the problems associated with hygiene and safety at work in miners - mine ventilation, organization of work and rest, work wear, extraction of mine water, air pollution, etc.

In the mid-nineteenth century and early twentieth century development of industrial production on the one hand, and scientific knowledge in the field of exact sciences (physics, chemistry, mathematics), biology and medicine on the other, gives a new development of research and practical activities in the field of hygiene of labour and occupational pathology.

In Russia in 1847 A. N. Nikitin published the first book on labour hygiene and occupational diseases ("Diseases of workers with instructions on preventive measures") and in 1877 F. F. Erisman published the first original guidance "Professional hygiene or hygiene of the mental and physical labour." Appear many new books and specialized journals on various issues of occupational medicine. Famous scientists in this field are then Lehmann (K. Lehmann) and Teleki (L. Teleky) in Germany; Simon (J. Simon), Hill (L. Hill), Breadford, Ledge (T. M. Legge - the first inspector of labour hygiene, 1898) in England; Bruardel (P. Brouardel) in France. In 1910 in Milan Devoto (L. Devoto) establish, existing and now (named on him), the first specialized clinic for occupational diseases. Institutes of occupational medicine were opening in Japan, Finland, Spain, USA. The founder of occupational medicine in the US is **Alice Hamilton** (1869-1970 years) published in 1925 the book "Occupational poisons in the US," professor of medicine at Harvard from 1919.



Alice Hamilton (1869-1970)

In Bulgaria in the early and mid-twentieth c. in the field of occupational medicine worked G. Zlatarski, L. Vankov, Hr. Piyanechki who carried out research on silicosis in miners. T. Petrov issued the first guidance on "Occupational Hygiene" (1927). He was the first professor of hygiene in Bulgaria and head of the first Department of Hygiene (1919) In 1944 **Hr. Hadzhiolov** established the first clinic for occupational diseases. In the area of hygiene of labour and occupational pathology work L. Tsvetkov (lead poisoning), B. Slavkov (mining), V. Kuzmin (microclimate), Kr. Kirjakov (transport medicine), M. Spasovski (occupational toxicology) D. Dimchev (vibration, noise) Vl. Boyadzhiev (branch hygiene, heavy metals), N. Tsaneva (physiology of labour), M. Dobreva and T. Burilkov (dust, silicosis) At. Kehaiov (hearing and vestibular analyzer), G. Bobev (sanitary chemistry) and others. In 1948 is created Institute of hygiene of labour with founder Iv. M. Ivanov (occupational hygiene), and in the 50s of XX c. create Scientific research institute of occupational safety and occupational diseases with long time director **M. Lukanov** (physiology of labour) and head of the clinic for occupational diseases Ts. Aleksieva (poisoning metals, organization of profpatological activity).



Prof. Dr. Hr. Hadzhiolov (1905-1983)



Prof. Dr Mircho Lukanov (1918-1994)

Later (early 80s) Institute developed as Research institute of hygiene and occupational diseases with longtime director F. Kaloyanova (occupational toxicology, pesticides). Create and Transport medical institute, Aviation medicine institute, Institute for radiobiology and radiation hygiene and Laboratories of physiology of labour and ergonomics to branch institutes.

1.1 EVOLUTION OF CONCEPTS FOR ORGANIZATION (STRUCTURE OF TASKS) OF LABOR[1]

Over the last two centuries, the process of labour has undergone major changes in the principles and practice of labour. A little by little craftsmanship gives way to mass production and ever more sophisticated machines replace workers, leading to a division of tasks and specialization. This evolution in the content of labour reaches a **"rationalization"** of the worker's activities in the production process.

Labour is the subject of numerous studies, that led to the **"scientific organization"** of labour, aimed at applying scientific methods to control human labour in disregard of psychological approach and social consequences. In response, appeared new forms of work organization, developed by researchers in psychology and sociology of labour and specialists in ergonomics.

1.1.1. EARLIER SYSTEMS OF LABOUR ORGANISATION

Taylor (F.W. Taylor - 1856-1915). Examine is the implementation of labour operations of various workers and is determined after analysis and synthesis best method that removes unnecessary or too lengthy movements and replacing them with more-efficient (spary) in terms of effort and time required by workers for their implementation.

This type of study has been widely known as timing and consists of dividing the work of elementary motions and measuring the duration of each of them. Disposed with the time required for elementary movements, can determine precisely the task that must be completed for a fixed time.

Knowing the time allows for the introduction of salary depending on performance. This, however, is differentiated wage, as a worker is paid per piece: the more you make, the more you receive, but with a minimum number of manufactured items (deliberately difficult attainable for working day).

The physiological risks of implementing the system Taylor is connected to:

• "Requiring, said Taylor, must always be well above the productivity of the average worker."

• The system of remuneration no account the "cost" of workers. Workers from densely populated areas, should make much more effort for the same wage. "If businesses are in a country with a large workforce like Philadelphia, wrote Taylor, the rhythm of work should be higher."

• Working movements and the smallest gestures are defined with precision by engineers. The worker has no right to change them, i.e. it turned into a machine.

• Workers who can not follow the required rhythm, removed.

This system contains no real study of fatigue. Despite careful selection workers exhibit obvious physical fatigue and after several years of intensive work removed from the workshops. The system Taylor survived only because the wear of labour in America was offset by immigration.

Moreover, in the United States workers actively protested. US Congress appointed a committee of three members, which makes careful study, resulting, in the following active protest of workers, the parliament of the United States decided - the credits voted in 1913 for government contracts, can not be used in enterprises where used the system of Taylor.

Ford (H. Ford - 1863-1947). – His name is associated primarily with the concepts of work conveyor and the theory of high wages. His contributions to the organization shall consist of:

- Mass (or large series) production;
- Interchangeability of parts and appliances;
- Replacement of transport by hand of the conveyor;
- Balancing the work places of the conveyor (uniform loading);
- The creation of tools and machines with high performance;
- Higher wages and replacing the work at piece with the imposed rhythm.

Bedeaux. – Before the last war the influence of Bedeaux lead to a revision of the methods for studying the labour market.

Bedeaux offers measurement unit *Bedeaux (B)*, which represents the amount of work, that is normally done by a worker for 1 min, taking into account the proportion of time spent on rest.

The maximum pace of the average worker is equal to 80 B, and normal 60 B. Such a pace reached the average worker without much effort and it corresponds to the wage specified in the collective agreement. In pace 80 B premium is 33% of the basic wage. An extremely handy and energetic worker can no problems to reach 100 B.

The positive contribution of Bedeaux system, that is integrated in the French methods of work organization, is the assessment of the pace of work and rest, as factors offsetting fatigue.

Gilbreth - 1868-1924. - His influence is felt slower in industrial environments (system implemented

[1] In **Leplat J., H. Monod** - Evolution de conception d, organisation du travail. Precis de medicine du travail, sous la dir. De H. Desoille, J. Sherrer, R. Truhaut, 6 ed., Masson, Paris, 1992.

first at masons), but nowadays it prevails due to the new direction, which gives research of professionals of psychotehnology and physiology. All organizers now know that reducing the execution time of a task is a function primarily of the used method, not by the speed of movement. Efforts in the organization should be directed towards a better adaptation of machines and tools to people and creating a work process that matches their needs (posture, environment, movement, rhythm ...)

A.G. Stahanov. - Miner, a specialist in the extraction of ore with a hammer for breaking into the Soviet Union, which, after analyzing his work, separates the two operations - supported by a beam and dislodge and achieved record results in digging coal. The method created in 1935 reveals the importance of the contribution of the worker in case of favorable organizational conditions, ie contractors can actively make good proposals and give new ideas after critically analyze technology and working methods.

Total consequences of these systems for "scientific" organization of labour are:

• In the field of payment - The number of workers without qualification, without meet the needs of skilled workers. Increases range of no labour categories related to planning, testing, supervision and recruitment.

• In the field of premiums - They are very important to compensate for the fixed basic salary, that corresponds to the minimum rate, and are typical for simple work, that is not of interest to the worker.

• With regard to the division of tasks and content of work. - Tasks more and more repeat, that leads to monotony imposed rhythm and significant mental stress. Physical load is associated with maintaining a specific posture, while high speed and accuracy of gestures when working with objects in motion.

• This Labour Organisation has social consequences. Unemployment among young, who refuse functions unattractive for received vocational training and an influx of immigrants.

• Social effects, which are individual (more absences from work in enterprises with monotonous work, irresponsibility, indifference to the quality of the product) or collective (conflicts between management and workers).

1.1.2. THE MOVEMENT FOR "HUMANE TREATMENT"

It originated in the US in the thirties of last century **Mayo (Elt. Mayo)** and occurs as a reaction against the excesses of the "scientific" organization of labour. It pleads for return of intolerable working conditions created by taylorism, with good physical and psychological environment in the enterprise. The purpose of human respect is better integration of the worker in the enterprise.

The heads of enterprises rely on the appeal of human respect, to change the behaviour of workers, without aiming to change their own behaviour, or the organization of work and even do nothing to

Tabl. 1. New forms of work organization

	Principle	Benefits	Difficulties and limitations
Rotation of tasks	Dislocation of operators holders of different jobs within a workgroup or conveyor for assembly or manufacture.	Reduced monotony of labor	Alternating plain and unmotivated tasks can not create real interest in labor 0+0+0=0 (Herzberg)
Extending the tasks	Extending the cycle time without changing the nature of the work.	- Reducing the number of jobs - Decrease mental fatigue of operators	Adding ordinary and unmotivated tasks can not create real interest in labor
Enrichment of tasks	Inclusion in the tasks to perform tasks associated with the preparation (regulation, supply, etc.) and control. Accompanied in many cases with the organization of space and jobs or improve the safety and comfort of workers.	- Increased responsibility, the ability to identify with the end product, more respect - In many cases: qualification and payment	- The need for additional training - Too often increase funds for salaries in the enterprise
Semi-autonomous teams	Groups of workers who take collective responsibility for a production with more or less rights for assessment the objectives.	- A good solution for some basic needs of workers, as an initiative, responsibility, relationships. - Frequently increase of qualification and remuneration	- A system limited to the manufacturing process and allowing the formation of small teams - Is needed too long preparation - Multiplying the risk of tensions and conflicts: interpersonal or between teams (since the tension increases with the increase of contacts) - Need additional training - Often, excessive increase in funds for salaries in the enterprise

change the unfavorable material conditions of labour.

As workers, they adopt generally manifestations of human respect and the fact, that work in a factory, of which benefit, but realize that more and more are involved in a model developed without their participation, in an organization that used them as a necessary object.

1.1.3. RESTRUCTURING TASKS

Following the problems of previous forms of work organization, began to talk about restructuring tasks to revive interest in the work.

Restructuring tasks stems from two trends that deal with the theory of human needs at work. According to **Maslow (A. Maslow)**, operating there is a danger man be deprived not only of social needs, but also entire hierarchy of needs - physiological, which are the basis of the rankings, to the need for implementation (ie the ability to exercise their own creativity at work), which is located on top of the "pyramid of Maslow." Requests workers covering all steps of the pyramid.

Herzberg used the conclusions of Maslow and creates a new theory of human needs at work. He distinguished:

1. needs, which if not implemented lead to dissatisfaction, but if you realize, do not provide the necessary satisfaction - hunger, safety ...

2. needs, the realization of which gives some satisfaction and arising from the specific nature of human nature . Workers express them through their demands for the content of labour, responsibilities, promotions and more.

As a result, they proposed and experimented new forms of work organization **(Table. 1)**.

After various forms of restructuring tasks are enriching tasks and the formation of semi-autonomous groups that are positively and give the work a more human nature - the employee can influence his work. Some workers, however, accustomed to repetitive work operations is hostile like restructuring, yielding new tensions and increases the effort.

The most often cited example of semi-autonomous groups of Volvo plant in Kalmar, Sweden. The car conveyor is removed and is replaced with small groups, each of which assembles a separate car.

In France, Renault management has applied this experience and is followed by other companies (more than 300 in total), mainly from the production of electric appliances and machinery.

The results for employees are:

- need of professional training and improvement
- abolishing the incentive remuneration (neoteylorizam)
- greater responsibility and autonomy of the performers
- reducing staff absenteeism and instability
- improving quality and productivity.

This requires, however, large investments that again raise questions about the role of leadership and the principle of hierarchy of authority. On the other hand, trade unions fear of overexploitation for, psychological manipulation of workers.

1.1.4. THE IMPORTANCE OF THE PHYSIOLOGY OF LABOUR AND ERGONOMICS

One important perspectives for the development of general physiology is the study of people at work. Possible aspects here:

1. Physical (muscle) activity of the worker.

2. Psychosensory, mental activity

3. The impact of the work environment (mainly physical factors - microclimate, lighting, sound, in the presence of undergoing work).

Whether it is for primarily psycho-sensory or muscle activity, or to study the working environment, physiology of labour deals mainly with elementary or integrated functional responses of individuals in certain working conditions. Ergonomics can be seen as primarily practical development of physiology and psychology of labour. It is usually defined as the science of adaptation of man to work.

Changes in the economy in the twentieth century are significant. Of mostly craft a system where generations used the same methods and tools, which produce the same products, proceed to this stage of industrialization, namely to produce significant quantities of products through integrated production structures. At present, it is important in any preliminary study of a production to take into account the following two issues:

- Whether the product is adapted to the needs of the individual, who should use it?
- Whether the jobs are adapted to the needs of workers?

For example, it makes no sense to produce cars that can not be used conveniently and securely from people or to build a plant in which in some places can work only under exceptional stress. These problems must be solved at the stage of design.

Social evolution greatly reduced and continues to reduce the level of allowable stress. In the XIX century working conditions are often unfavorable and enforcement, and working time – longer. Adverse factors progressively reduced, but the requirements

of social progress include continuous reduction of their level.

So in the middle of the twentieth century. Working conditions are sufficiently studied as effects on workers to build ergonomics as an independent discipline. The need for it is felt both in society and in the economic sphere. Along with the physiology and psychophysiology of work, dealing with human functioning at work, discipline was formed (beginning as so-called "corrective ergonomics"), which aims to modify the tools or the task of labour for to adapt to the individual. Thus reducing the load with which has to cope the body and increases productivity, quality and reliability of work.

1.1.5. ELECTRONICS AND MODERN LABOUR

Development of modern electronics is many applications. These include computers and machines for automatic processing of information, operated by programs when performing arithmetic logic operations, enabling not only to produce multiple reports, but also for decision-making. Two large technical advantages are miniaturization that allows to record a large number of data (created term memory) and speed of operation (eg. In program chess game, the computer can calculate three million data and more for 3 minutes).

Electronic devices can replace humans in many areas: statistical reporting, monitoring and control of machines, allowing more high level of automatic operation.

Contribution of electronics allows the man to replace the machine, suggesting earlier availability of information, hence the terms **automation** and **informatization**, or **robotics**.

The robot is a machine composed of two parts: a device performing operations and software that manages the sequence.

Guittet defines it as "an automatic machine that can be adapted to a complex environment and to replace or extend one or more functions of a person being able to influence the environment." Theoretically there are three generations of robots with different levels of complexity (from monovalency to complex robotic systems).

Stages of informatization. A review of the history of technics over the last 50 years makes it possible to distinguish three main periods of informatization. By the early seventies are known two main types of automatic machines, computers of large enterprises and automatic controls of fixed production processes (chemical, petrochemical, etc.). Then appear processing machines with numerical control (PMNC). Later seen extremely rapid multiplication of types of automatic machines, as occurred a year dozens of new types.

They can be distinguished two phases of informatization with very different technical and social characteristics.

Phase of centralization is characterized on the one hand centralized computing, which is owned by large organizations, and the other a substitution. The last is to replace some devices (such as processing equipment) of mechanical components with electronic. Originally used for scientific calculations (especially in warfare), informatics progressively begin to apply in the management of enterprises. Informatization of management focuses primarily large enterprises and less in administration.

Gradually this "elitist information" gives way to mass informatization - ie. "**Informatization boom**." The second phase is characterized by a multiplicity of applications and users of automatic machines and foremost with their integration into "automated systems".

Today distinguish at least 7 types of machines:

- PMNC.

- Automatic machines for continuous production of large series. Besides robots in this category includes manipulation devices, integrated systems of robots and programming machines.

- Machinery for process automation. Refineries or water power plants are an example of the use of machines for the automation of continuous processes.
- Design with computer. Currently used mainly in aerospace, automotive, shipbuilding, this method allows to automatically do all the calculations required for the design of a product.
- Automatic devices for optical reading: these are automated distribution of letters or systems for processing checks in banks.
- Machine tools for word processing (typewriters with memory). They are connected to a computer, which allows for automatic paging and corrections.
- Fax - system that enables you to transmit photocopies of distances on the phone lines.
- Database - computerised libraries that can be used with a computer (terminal).

Can indicate other applications that are less likely to be classified. These are **mini terminals** and **microcomputers**, which are found almost everywhere, and differ only in program management. Microcomputers are used in workshops and offices for accounting and inventory management. Another form of informatization, which has developed very fast, **electronic payment**, which is used in various fields such as selling tickets, selling merchandise and ATMs.

Qualitatively new phase of mass information is the creation and development of the **global informatization system** (Internet). Form a futuristic concept of teleworking by telecommunications, which will put the issue of working from home.

New working conditions. Informatization often presented as the safest way to release the man from some difficult production tasks associated most often with removal of intense physical effort in a hostile environment, performed by robots or operations that are characterized by no uniformly and repeatability.

In informatization however, new problems appeared associated with psychosensory tension. Studies have shown that the operation of the terminal, there are lots of symptoms associated with the visual fatigue, such as headache (80% of cases), blinding (70%), burning of the eyes (70%) and reduction of visual acuity (50%).

On the other hand psychological factors also change. Before the master commanded the team. Placed in front of the controls, however, it no longer does. Moreover, this transformation may at first lead mainly in older workers to emotional instability and psychosomatic disorders due to fear of dismissal in reducing staff, into another category with a change of perspective for development and the possibility of moving to another post not complying with the skills and professional experience.

Studies in these workers indicate tendency to a high incidence of psychiatric symptoms: anxiety (40%), insomnia (20%), depression (25%).

Informatization not lead, however, to a uniform deterioration of working conditions. Informatization is primarily a heterogeneous from mental perspective phenomenon. For carrying out an order is appropriate and inappropriate automation systems and devices. In informatization can and should be selected, ie to apply the requirements of ergonomics.

Consequences of the organization of work (structure of tasks). The consequences of informatization in the field of labour organization vary depending on the phase of informatization.

A) A centralized information - Each automatic machine consists of two separate elements: the device and software. In this phase of informatization most significant changes undergoes work with the machine, and preparation (development program) continues to be organized in a fairly traditional way.

Working with the machine: the big novelty at this level is the emergence of an entirely new function, control and surveillance. By automatic devices worker involved in the implementation of several operations that were previously separated. This is a reversal of the tendency to split the tasks. The share of workers it however less because it is "unable" to perform alone the various operations carried out by the machinery, but only controls the execution. So the worker has no direct contact with the product of their labour.

Preparation: Regardless of the type of automatic device preparation is associated with program development. This includes three categories of staff:

- Analysts who determine the logic of Information systems;
- Programmers who transcribed this logic, the language of machines;
- Operators, who make this transcription readable by computer.

The very existence of information services leads to so-called deskilling/excessive qualification by M. Freyssenet, namely that technical progress is linked to the reduction of qualification for the majority (operators) and qualification for the elite, the one that programe.

Centralization of information has resulted in large enterprises to increasingly teylorism in the process of preparation of labour with machines. At this stage there was no real break from previous forms of work organization.

B) **"Boom of informatization."** Three trends characterize in the beginning of the 80s the arrival of a new "era", that of " informatic without informatics", namely:

- The creation of a "generator of programs" and the multiplication of "library for programs", leading to a substantial reduction the role of programmers.
- The increasing simplification of languages (creation of APL - conversational language and specialized languages), facilitating the use of IT by non-specialists.
- The integration of the program into programming package.

The development of integration is closely linked to the progress of the operation of the terminal. The specifics of the operation of the terminal is in the dialogue between man and machine. Therefore among operators, supervisors, and those dealing with the introduction of data, need to add a new category - operators working with interactive keyboard.

The progress in the integration of automated systems increases the need for teamwork. The team of workers in this case is composed of three sub-teams - control; surveillance; guidance, maintenance and transportation. First team of which depends mostly on the proper functioning of the enterprise is characterized by very high stability in the workplace, while the other two are "peripheral" and have tem-

porary status - provisionally performed functions and subcontractors.

Based on all the consequences to man of information it is necessary to conduct adequate **social protection** and **maintain good health psychosomatic condition**. Indicate four priority areas:

• **information:** full and early information is the only way not to multiply the phenomenon of rejection by continuous influx of new information technologies in the enterprise

• **prevention:** 1) thanks to the development of ergonomics and the intervention of specialists in occupational medicine (SOM) may to adapt the machine to the man and so to improve working conditions; 2) psychological factors change in the period of transition and SOM must scrutinize the cases of transfer to a new job.

• **training:** informatization create jobs, but also led to the closure of others. Vocational training allows for adaptation to new knowledge and a guarantee against mass unemployment.

• **negotiations:** They allow for controlled opposing interests and through them over time, thanks to mass informatization to create social democracy, i.e. benefit of all.

1.2. FACTORS OF WORKING ENVIRONMENT

Working conditions, affecting the health and performance can be divided into two groups.

Work environment factors, which include: the microclimate factors (temperature, humidity, speed of air movement and infrared radiation), production radiation (ionizing, non-ionizing, laser), industrial lighting (i.e. visible light), mechanical oscillations (noise, vibration, infra- ultrasound), atmospheric pressure; chemical factors (gases, vapors, liquids, aerosols with toxic action); dust (physico-chemical) factors, biological factors.[1]

Factors of labour process related to heaviness and tension of labour; regime of work and rest; working posture; workplace and working movements; psychosocial factors.

Some authors indicate indirectly acting on the health status and factors related to **labour environment**: planning, design and technical solution in production premises; location and connections of technological machinery and equipment, manufacturing furniture and furnishings; sanitary equipments (industrial heating, ventilation).

1.2.1. WORK ENVIRONMENT FACTORS – INDUSTRIES AND PROFESSIONS

In the modern labour massively entering high-performance machines and equipment, processes, instrumentation, using and which are the sources of **physical factors (hazards)** of the working environment. Most diverse industries, and especially textile production (spinning and weaving mills), metallurgy and machine-building, logging and wood processing, ore mining and others characterized by intense noise; intense local vibrations are typical for logging; "general" vibration - for transport; adverse microclimate (excessive heat, cooling) - when working out (agriculture, construction, metallurgy, shipping). For contemporary work are characteristic radio frequency electromagnetic fields (induction furnaces in metallurgy, radar, telecommunications); laser radiation (holography, radio electronics, automotive); ultrasound in the air (in high speed machines) and ultrasound in liquids (cleaning of metal parts); infrasound (large machinery and equipment in metallurgy, transport). The atmospheric pressure (high, low and rapidly changing) is a "classic" and present harm at work under water and under water pools or at high altitude. Typical of contemporary work is, and so-called **"chemization of labour."** Y.V. Sanotskiy (1987) states that the rapid development of chemistry and the growing needs, currently is characterized by existing over 5 million chemical compounds, weekly synthesis in the world of 5-6 thousand new compounds and now use over 60 thousand chemicals and more - an annual introducing in production of over 1000 substances. "Chemical industries" can be called the production of plastics, fertilizers and pesticides, paints and varnishes, chemical-pharmaceutical industries, petrochemistry, production of acids, paints, detergents and others. Chemical factors related to technological processes as hazards in the working environment can be released in a variety of industries - metallurgy, machine building, textile industry, transport, construction, mining, etc. Characteristically, however for modern technology and procedures is the mass automation of technological processes, replacement of highly toxic chemicals with less toxic or non-toxic (eg. replace organic solvents with water) and ultimately higher degree of protection of labour and the environment by chemical hazards. A major problem with them still carrying out repair works on the equipments and especially the cases of accidents - eg. accident at the chemical plant in Bhopal - India in 1984, where divisions gas (methyl isocyanate) affected over 20 thousand people and caused two thousand deaths.

[2] I here may also be included traumatic factors (mechanical, chemical, electrical, etc.).

Dust such as harmfulness of the working environment changes its meaning in modern working conditions. The high degree of dust with roughly dispersed and with high crystalline SiO_2 (fibrosogenic) dust is rare meet now state (often in the past in underground mining - i.e. "dry" drilling in the manufacture of building materials). Currently actual effect is of finely divided dust (mining, construction, agriculture), dust with allergenic effect (agriculture, construction materials - cement, pharmaceuticals), with carcinogenic effects (asbestos, aerosols containing nickel, chromium, etc.).

Forty years of the twentieth century. It is characterized by rapid development of manufactures for so-called biologically active substances - antibiotics, amino acids, proteins, enzymes etc., based on microbiological synthesis. Working conditions in modern enterprises of the chemical-pharmaceutical industry (antibiotics); microbiological industry (production of a food protein, microbial fertilizer, insecticides, etc.); textiles; agriculture (livestock, poultry farming, husbandry) pointed to the forefront and so-called **"adverse biological factor"**. Biological factors are expressed now hazards and for working with liquid and solid waste (cleaning of settlements), in the leather industry, working in places with vectors (carriers - eg. Insects) of infectious diseases - geologists, logging, working with biological materials in human and veterinary medicine. Now and in the near future biological factor is a major adverse for health and caused high mortality factor in countries of warm climate in their main industries of agriculture (farming, husbandry), mining and others. Biological factors - micro- and macro organisms, viruses and products of their metabolism (biologically active substances) are the causes of infectious and parasitic diseases; dysbacteriosis; mycosis (eg. candidiasis - Cand. albicans) and others.; intoxications (eg. mycotoxicosis when working in silos, in wood processing); allergic diseases (eg. hypersensitivity pneumonitis or so called "farmer's lung", first described by Ramazini in 1713); malignancies (eg. angiosarcoma of the liver in aflatoxicosises - toxins of the fungus Aspergillus flavus). Modern ventilation systems (air conditioning systems) with recirculation of air in rooms (offices) with closed windows or no windows workshops are important for the growth of mold into the air.

1.2.2. WORK ENVIRONMENT - HEALTH STATUS AND PERFORMANCE

Enacted in 1949 by the WHO definition of **health** as "a state of physical, mental and social well-being" implies:

- not only the absence of somatic and psychiatric disorders and diseases of the body, but also high resistance to various diseases and high performance;

- opportunities to restore and enhance the cultural and intellectual level;

- state of health, as a constant process of complete adaptation of the organism to changing conditions of environmental and working environment (labour).

Factors of working environment and labour process, as adverse health factors, influencing performance (reducing it, and quickly led to fatigue), cause the development of professional and paraprofessional diseases, affect the general morbidity (increased frequency, heaviness, altered structure).

Now, a person is subjected to adverse effects of many factors in production and the environment. **Typical for them now is that:**

- Usually are with less intensity;
- Act combined, sequential, continuous or intermittent with constant or variable intensity;
- They are of a different characteristics (nature) with similar or different effects on the body;
- The combined presence of chemical factors leads to the physico-chemical reactions and the formation of new substances, and conditions, also acting on the body;
- Acting chronic, and combined with a healthy or not lifestyle - proper daily regimen, nutrition, physical activity, alcohol use and smoking, drug addiction.

When work environment factors are of **less intensity, but act chronic** and combined with other adverse factors and lifestyle (healthy or not), they do not cause development of the occupational diseases, but reduce efficiency and immunological reactivity.

Adverse effects herein are characterized by the so-called small and "hidden" symptomatology (occurring after provocation), affects the whole organism, the reactions are mostly phases[2]. In this case the work environment factors appear (with varying degrees of probability) as one of the risk factors for formation the general morbidity.

For example, epidemiological studies of **diseases of the cardiovascular system and conditions of dyslipidemias** in workers in contact with carbon disulfide or affect of noise levels below those causing specific disorders (decrease in auditory sensitivity) showed that as at relatively low concentrations (below and about MAC) of carbon disulphide and the noise factor is the risk factors for development (high-

[2] Place in three phases: Ist - functional or weak structural changes (i.e. a biochemical "adaptation"); IInd - a period of "hidden" decompensation (apparent adaptation); III th - deterioration, with lasting changes and clinical signs of damage

er incidence) of cardiovascular pathology (together with other risk factors for it, with production and non-production character). Such studies, combined with experimental studies have shown through use of more sophisticated methods of statistical analysis (factorial, discriminant, logistic regression) the importance of vibration factor in the higher incidence of dyslipidemia, coronary heart disease and hypertension in workers with and without specific clinical manifestations of vibration damage.

Also is known the cumulative effect of factors of working environment with less intensity, with such of the labour process, resulting in **reduced performance** - eg. noise factor of intensities much lower than 80-85 dB increase significantly neurosensorial tension in operators.

Occurrence or not of these "non-specific" effects of work environment factors is closely related to the functionality of the body's systems of workers to maintain homeostasis (opportunities conditioned as genetic and as of lifestyle - healthy or not).

Factors of working environment with less intensity and chronic effect, on impact on small or large populations (groups of workers in industries) often cause the development of allergic reactions and diseases and are factors for carcinogenesis.

Currently around 10% of general morbidity are various **allergic disorders** (US indicate for the general population about 50% slight and 10% with more serious allergic reactions). A typical and severe damage to the respiratory system - asthma is often a professional etiology (5-15% of cases). Contact dermatitis is about 90% of all cases of occupational dermatoses (in 80% affect the main body part of work - hands). Typical occupational allergic diseases are also farmer's lung; bagasosis; byssinosis; allergic rhinitis (pollen fever) and conjunctivitis. Global trend is a steady increase in the incidence of allergic disabilities.

Chemical factors in the work environment are often allergens - phenol and its derivatives, benzene, arsenic, mercury, zinc and manganese compounds, chromium, cobalt, aromatic- nitro and amine compounds, isocyanates, detergents, organic solvents. Also such factors are dust factors - cement, flour, vegetable; biological factors - antibiotics, food yeast, microbial insecticides and fertilizers; physical factors - cooling microclimate, intense insolation, UV-radiation.

Modern production of chemical-pharmaceutical industry, plastics, building materials, paints and varnishes, petrochemistry, agriculture, textile industry have a higher incidence of allergic disabilities.

It is noted that 30-40% of the population of developed industrial countries develop **cancer,** as one person in five is dying of cancer. The importance of professional factors for developing cancer is described for the first time by **Percival Pott** in 1775 in London (cancer of the scrotum in chimney sweeps - soot) later, at the end of the nineteenth century, high incidence of lung cancer in miners of mines for uranium and nonferrous metals in Czechoslovakia (radon combined with dust containing quartz, nickel, haematite).

In the second half of the twentieth century various authors already indicate that 4 to 40 percent of cancer cases are caused by occupational hazards. Many modern productions at risk of developing cancer by work environment factors - chemical and chemical-pharmaceutical, petrochemical, work with ionizing radiation, metallurgy and machine building, agriculture, mining, production of plastics, rubber industry.

International Agency for Research on Cancer (IARC) divides agents (factors), depending on the presence or absence of the carcinogenic effect of the 5 groups, the first 3 groups (1, 2^a and 2^b) have such effects.

The first group, known to be carcinogenic in humans are 22 chemicals, such as arsenic, asbestos, benzene, beryllium, cadmium, chromium and their compounds, mineral oils, nickel compounds, vinyl chloride, wood dust and others. It also includes radon, solar radiation, some biological factors (hepatitis B and C virus, Helicobacter pylori, Schistosoma hematobium). All of them, without biological factors present real production risk. **In the second group** (probable carcinogenic risk) are 20 chemical factors - benzidine, formaldehyde, polychlorinated biphennyls crystalline SiO_2, tri- and tetrachlorethylene, etc., and UV-radiation. **In the third group** (2^b) - a possible carcinogenic risk, included 62 chemical substances and their compounds - diesel fuel, hydrazine, lead, methylmercury, nickel, some dyes (o-toluidine, ponceau), styrene, vinyl acetate, foundry smoke, etc. Separately, in the above groups are also included 22 pesticides (eg. DDT, heptachlor) and 31 drugs (phenacetin, cyclosporine, estrogens, oral contraceptives, chloramphenicol, etc.). This variety of chronic relevant factors with proven or possible carcinogenic effect in humans, involving many industries and large groups of workers, shows great medical and social importance of preventions in the occupational cancer.

1.2.3. HYGIENE STANDARDIZATION AND HEALTH RISK

Modern definition of hygienic standard of **chemical agents** in the air of the working environment was "such a limit concentration (MAC), which in

everyday work (8h) during the working week and throughout the all service can not cause deviations in health (established by the most modern methods and knowledge), over the full lifetime of the worker and his offspring. " This definition is basically the same for other factors of working environment.

Generally hygienic standardization, as mandatory and recommended separate regulations and standards starting in the second half of the nineteenth century and in early twentieth century - perhaps first for the qualities of drinking water - i.e. "Limit values" - the Brussels Commission in 1853, and the first standard for water - USA, 1914 The development of hygienic norms and standards for work environment factors starts during the first half of the twentieth century. It is known that the process of hygienic normalization is continuous, despite the vast accumulated material, due to the continuous development of human knowledge, both in the field of production and technology, and the impact of factors on the body.

Basic concepts (criteria) for hygienic normalization of working environment factors are:

- "Medical" - avoiding damage to health;
- "Publicly acceptable risk" - comparing with the situation in a "successful" (compared to others) production in relation to health, or to the so-called "natural background";
- "Economic criteria" - funds for the improvement of working conditions and the economic losses from diseases, retirement and reassignment;
- "Zero levels" - non-factor and reduce it to that in "natural" conditions of the environment.

Since in the base of hygienic normalization is the concept of so-called "threshold" (quantify) of the adverse action - to remind the definition of Paracelsus **"Everything is poison, only the dose makes the poison invisible"** health criteria are the main. Certainly in specific cases, linked to the other conditions of work - eg. time of exposure, combined with other hazards, path ways of entry, time for practical implementation, etc., into account are also the other concepts (criteria). This applies also and in a stochastic factors (no-threshold action) - eg. ionizing radiation with regard to gene mutations in somatic and sex cells.

The theory and practices of hygiene standards is based on two sets of criteria:

- Evaluation of biological action - indicators of health status, performance, psychological state;
- Assessment of factors - the degree of expression (intensity, concentration), exposure (shifts, total length of service).

These criteria reflect the relationship man-environment. In observing hygienic standards (concentrations, levels) are not followed:

- Acute or chronic occupational diseases and poisoning;
- Does not increase the susceptibility of the organism to the development of other diseases - cardiovascular, cancer, allergic, etc .;
- Not lowering reproduction capabilities and worsen the health status of the offspring;
- No accelerates aging and shortens life expectancy.

Dose's approach is fundamentally correct, but there are features associated with the pathway of chemical substances in the body, different exposure time (shift, day, week, year, total length of service), area of exposure and others. Eg. in chemical factors determine the maximum-single time and mid-shift allowable concentrations, similar is a normalization of non-ionizing electromagnetic fields (allowable energy for work shift and allowable maximum intensity).

It is especially difficult in normalization to: low doses, different path ways of entrance, entrance and by environmental (not only by labour environment), combined and intermittent action. More exactly and easy is normalization using experimental models of animals and humans (but the latter is very limited), but it is difficult interpretation in the obligatory epidemiological study of people in real working conditions and taking into account the influence of factors of lifestyle and nutrition.

It was pointed out that in the field of hygienic standardization has gained vast material. Although it is an ongoing process, established productions now it will be done only in the emergence of new factors, qualitatively distinct from previously known - eg. now for evaluating the toxic effects of constantly emerging chemicals and compounds, used quick "account" methods followed by monitoring the health status of people working with them. **Now normalization will target in two main areas:**

• the impact of factors with less intensity on efficiency in modern forms of work and on comfort - eg. and now standards for production microclimate are admissible and optimal (providing comfort and high performance), with Fanger's indicator (ISO-7730) is defined thermal comfort/discomfort; the standard of ISO-2631-1 for general vibration defines three levels of permissible impact, two of which ensure "no influence of performance" and "no disturb comfort"; permissible sound levels in mainly mental work are much lower (50-65 dB/A) than for enterprises - 85 dB/A (with a view harmless auditory senses).

• connecting hygiene standards with determination (evaluation) of the health risk. Until now, existing data accept the position, that the joint action of factors prevails so-called total action. It reflected in a widely known formula to chemical factors $C_1/TLV_1 + S_2/TLV_2 + ... Cn/TLVn \leq 1$, but it turned out that it is only applicable to xenobiotics with a similar action (eg. substances with narcotic effect). The enormous variety of factors in the working environment and conditions of the employment process with less intensity (about hygiene standards), combined with the conditions of lifestyle and nutrition, does not require continual introduction of new and new standards for the individual factors but a **new approach** to the assessment of their adverse impacts. These impacts are associated primarily with a reduction in immunological reactivity of the organism and hence with increased incidence of general morbidity - cardiovascular, cancer, allergic, respiratory and others. Also with reduced capacity and with the states of discomfort. In the 80^s of the twentieth century studies by NIOSH (USA) and development of the expert group of the WHO introduced the term "building associated ilness", which occurs when working in modern buildings (offices, administrative premises, control services, etc.), subjected to the action of many factors with a small intensity. Described are real and potential adverse conditions with short and long latency period and is not always associated with certain exactly agent - i.e. "syndrome of confined office""mass psychogenic illness," "hypersensitivity pneumonitis" and others. All this necessitates for the health risk assessment of action to already normalized factors in the work environment.

Health risk assessments is relatively new, current, and prospective area of hygiene science and occupational medicine in particular. The risk assessment includes an evaluation of the ability of the factor (s) to participate for the occurrence of adverse health conditions. The risk assessment includes the so-called four main paradigms - identification of risk; determining the dose/response; exposure assessment; risk characterization. They are interrelated and include both experiments on laboratory animals and humans (when possible) and epidemiological studies for working in industries with health risk factors (including that/those of whose effect will be demonstrated). Here should be forecast **some specifics**:

• Not always found exact dose-response relationship. In operation at low intensities (doses) or factors with expected fewer degree to contribute to the occurrence of a condition (eg. a toxic factor and hypertension) is sought, most often in the experiment the presence of the effect (biological response) relative to this factor, or by epidemiological studies an increased incidence (i.e. a dose-effect) of cases with adverse health condition (disease or other).

• Certain factors have no threshold effect - eg. ionizing radiation and carcinogenic effects. With mathematical models here are account of the risk of increased incidence of fatal malignancy in large populations irradiated with an appropriate dose, compared with natural (background) rate of the general population. In these so-called stochastic health effects can not determine at what dose will arise a disease (and with what degree of damage) in the individual (with probability $p \leq 0.05$), but how many cases will increase the frequency of the disease, according to the number of impacting people.

When factors of low intensity or in those who are unable to establish a dose-response relationship **(shown dose-effect)** are used multi-factor complex methods for statistical analysis - factorial, discriminant, logistic regression and the like. Create models that seeks significance more than 3/4 (75%) - ie, that a combination of adverse health factors (including studied by us) in over 75% of people (employees) will develop this condition (disease). Basic statistical method here is the definition of so-called. odds ratio (OR) and relative risk (RR) based on method x^2 for non-parametric statistical analysis - i.e. assess the reliability of differences in incidence (diseased/non-diseased) when operating in industries with research risk factors and those with absence of this risk factor. Use also other methods of statistical analysis - variance, correlation, regression, and make comparisons of data obtained through them. **An interesting feature (not a contradiction!) here is justified combination of proven probability $p \leq 0.05$, with the importance of forecasting (participation in model) phenomenon (illness or other) at 75% or more of the people.**

The so-called "philosophy of health risk assessments" based primarily on the so-called principle ALARA (As Low As Reasonably Achievable). "Philosophy" is based on the following situations:

- "Zero" risk does not exist - each activity is associated with some degree of risk;

- Safety (full) no - relative safety is related to acceptance of some degree (level) of risk;

- The level of risk is manageable with appropriate personal and collective preventive measures;

- Health risk varies between two conditional limits - from 0 to 1 or $1 \geq R \leq 0$, where R = 1 when the risk is unacceptably high, and when R = 0 - very low;

- In all cases (values) of R, take appropriate preventive action based on the principle ALARA;

- The safety level of the system (B) - B = 1 - R.

Applying ALARA principle shown in **Fig. 1**, where the cost to compensate the effects of risk "Y" are costs to compensate victims (temporary disability, occupational diseases, prolonged work, work at high risk, etc.), property damage, unrealized production, worsened quality and rejects etc. These costs are a linear function of the magnitude of the risk. Cost (X) to enhance safety (B) are exponential curve. Curve "S", expressing the sum of the cost "X" and "Y" has its optimum, which can be associated with the application of the principle ALARA. The application, however, is closely related to the setting of **minimum requirements for safety**, ie hygienic norms and standards, which should not be exceeded. Moreover, there intervene a number of health, ethical and humane considerations in the valuation of "reasonable eligible costs", especially when considering prevention and health promotion.

In the diversity of the relevant factors, health risk assessments, as current prospective area of occupational medicine has its very great practical value for the introduction of mass control for health workers.

Solving the problems caused by the influence of work environment factors on health in contemporary work focuses on:

- **technical facilities and technology** - massive automation (computerization) of production lines (processes) and use of non-waste technology with low emergency risk;
- **training of workers** - higher professional qualifications; training for maximum safe operation of facilities repair and emergency situations; creating sustainable habits for a healthy lifestyle;
- **health risk assessment** in the specific working conditions in modern productions. Establishment of the organization and participation of the occupational doctor into it permanent comprehensive monitoring of the health status of workers;
- **health policy** - to favorably resolve problems related to workers' health.

1.3. FORECASTS FOR THE DEVELOPMENT OF POSTINDUSTRIAL SOCIETY[3]

1.3.1. INDUSTRIAL SOCIETY

This society is often defined as industrial for countries that have reached a high degree of economic development. This name covers other activities, including unless industry itself also raw material

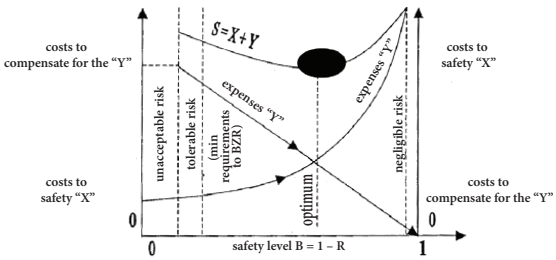


Fig. 1. Scheme for implementation of the "ALARA" (by D. Dimitrov, 2002)

extraction, agriculture and services. Economy and active population of each country are distributed in different sectors.

In industrialized countries, such as France and Germany, for example, employed in industry are about 40-50% of the active population. But in each of these countries has a modern agricultural sector, which is industrializing and services sector (respectively around 6-9% and 45-50% for France and Germany in the 80[s]).

The part of industry in relation of employment of the active population and the economy is a key and explains the use of the term industrial society although the balance between the different spheres of employment and activity is not final and changes. Beyond the differences between countries due to differences in their resources at the beginning and their successful use, in developed countries is a tendency for informatization of the active population and economy, and with a significant reduction in the agricultural sector at the expense of industry. The latter initially developed rapidly after it reaches a level and no progressed (or progressing too weak and tends to regress), while the services sector has steadily increased. As a result, the term industrial society rather reflected a balance of activities in the economy, built on high technology and informatization (electronization).

Evolution of industrial society. The transition from craft to industry in the early nineteenth century led to profound changes in working conditions and put its mark on modern society.

The main characteristics of the artisanal work, conceived and carried out by the same person, using the technology and knowledge that are the result of a long process, and motivation, whose ultimate goal is creativity, largely removed from industrial labour.

The division of tasks, hierarchy functions, collective framework of the production units, typical of industrial labour leads to dehumanization of work.

Technological progress deepens the discomfort of the transition from craft to industry in the growing replacement of manual labour with mechanization. Fragmentation and complexity of manufacturing operations makes human subject, crafted a

[1] In **Leplat J., H. Monod** - Evolution de conception d, organisation du travail. Precis de medicine du travail, sous la dir. De H. Desoille, J. Sherrer, R. Truhaut, 6 ed., Masson, Paris, 1992.

article with tools or machines, in person – object, connected or even subordinated to the machine. So artisan who became a worker, loses his creative freedom and functional autonomy, despite the fact that social life offers more knowledge and opportunities. These are all conditions for "industrial disadaptation." Its manifestations in the form of labour conflicts, absenteeism and increasing disrespect for industrial labour. Connected with this, and characteristic of industrial society is still relatively high frequency of accidents (whose importance and seriousness were denied), but also disease (professional or not), wear the organism and premature aging.

1.3.2. TO POSTINDUSTRIAL SOCIETY.

Most modern enterprises and government explore means of eliminating more obvious disadvantages of working conditions in industrialized countries.

In technical terms the changes that lie ahead should lead to the development of automation in industrial production (to free the worker from mechanical tension) as well as research in areas where automation is not yet implemented.

To relieve labour and increase its attractiveness to introduce systems of work, aimed alternation task by staff rotation to break the monotony or reduce physical effort; be grouped again fragmented small operations to obtain a work task with more importance for the worker; to re-evaluate their professional activity, as it integrate organizational and managerial functions.

In this connection, the more likely it is the smaller production units with greater autonomy to progressively displace the larger.

With regard to professional qualifications, this evolution will lead to a significant reduction in the proportion of unskilled workers and back increase the technical staff, engineers, researchers. There is for example the emergence of a new category of workers working with industrial equipment or operators to surveillance. Their work is primarily intellectual, not excluding some manual operations.

These changes will lead to a **change in hierarchy and social relations in the enterprise, account** that the operators (or at least most of them) will have a full and broad education and new responsibilities related to the need for automation and organizational and managerial functions. The transition from mechanization to automation, from manual labour to more intellectual tasks, that require control and regulation of processes, allows for a return to autonomy and initiative specific to artisanal labour. Interpersonal and intergroup relations is also determined by the greater autonomy and responsibility of people, teams and manufacturing units. So strictly hierarchical relationship lost its advantage, in order to reach what is referred to as "industrial democracy."

Primary **evolution towards postindustrial society is marked by the importance given to the human factor.** His place will grow in proportion to the complexity of production processes, whose capacity (quantity and quality) will depend again by the operator, as before by the machine or working movements. The importance attached to human factors will allow to balance professional life with social (reduction of working hours, work at home, etc.). However, before the period of transition to a postindustrial society, which will be prolonged, it should be expected inevitable tension. It will be mainly due to internal reorganisations and conversions of enterprises; uncertainty in the productive sectors; the discrepancy between the process of adaptation to the labour and relevant training tools and processing, etc. Because of this transition to a new form of society will include largely scientific predictions, based not only on existing methods and disciplines in the field of exact sciences, but also the disciplines of human science, such as psychology, physiology and sociology.

As far as the interdependence between labour and the environment will increase, they will converge ergonomics as a scientific approach to labour and ecology as a scientific approach to the environment.

This multidisciplinary perspective of the postindustrial society, participation of sciences who study human will be mainly, and occupational medicine will takes its place.

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2 PHYSIOLOGY OF LABOUR

Human in its existence is in constant interaction with the environment. The whole existence of the organism is a continuous chain of stimuli and responses to these stimuli. The framework in which these changes in the body in response to external conditions do not lead to pathological changes are physiological limits or conditions of existence. In his work the person meets specific changes in environmental conditions and with different types and weight loads. For this reason, in general physiology as a science, departed branch or department studying processes in the body in the course of balancing his work environment and requirements of the workload. On the other hand knowledge of the many factors of working environment and labour process is important in assessing the response of the body. Therefore, currently, the physiology of labour is accepted as part of hygiene/or medicine of labour.

In the last 2 decades of technological progress have even more complex relationship between body, working environment and social environment. In industry the tools undergo not only enormously development and of appendage of the human hand became a leading unit, but the development of new technologies led to the "extension" of the human brain or enhance its "capacities". The highest forms of work organization associated in a functional complex large groups of people and machines, to build complex systems and networks pokrivashi major regions and parts of the Earth. Employment in these systems and networks is subject to the same requirements, sometimes a rhythm, responsibility, etc. All this puts the man in front of new requirements in the process of balancing his employment. These changes in the nature of work, place issues of psychology and psychophysiology of labour as a part in the general labour - hygienic problems.

Physiology of labour used physiological, biochemical, psychological and occupational hygienic methods. Subject to the study of the physiology of labour are: problems that occur on the organism in different types of workloads and performance issues; the problem of fatigue as an important defense mechanism, whose physiological function is to prevent the occurrence of pathological changes; problems of intellectual labour and occupational stress; individual abilities and personal characteristics; problems of shift work and night work and others.

Physiology of labour is the object of a number of science-practical tasks: the standardization and categorization of severity of labour; developing science-based regiments of work and rest; development of ergonomic requirements for work equipment, furniture; rationalization of working movements, etc.

2.1. PHYSICAL WORK

Depending on mostly what body systems are involved in the implementation of the work the work is divided into physical and non-physical or mental in the most general sense. In any kind of physical work involved and nervous system and in any mental work - and sceletal-muscular system. Therefore it is more correct to speak of mainly physical and mostly mental work.

2.1.1. TYPES OF PHYSICAL WORK

According to physiological characteristics of muscle contraction work can be dynamic or static. As a physiological phenomenon, dynamic work is a isotonic muscle contraction, that involve various units of kinematic chain, i.e. moving relatively to each other, to carry and mechanical work. It is accepted that dynamic work is divided into two types: "positive" and "negative." Usually the first is associated with concentric contraction of the muscles and the second with extension of the muscles actively resisting to the stretching force by developing tension. For example, when climbing stairs extensors of feet made positive work, and when he came down they stretch and made a negative work. Energy consumption in the negative job is much smaller.

Static work we have in isometric muscle contraction in which the muscle does not change its length and growing its internal tensions. In static tense muscles blood irrigation is insufficient due to constriction of blood vessels, creating conditions for the accumulation of acid products, delays in the release of metabolic products and fatigue occurs rapidly. Static work occurs with less energy expense, but immediately after stopping its oxygen consumption and the amount of evolved carbon dioxide increased and the recovery process due to oxygen accumulated debt is slower.

Static working there is when the muscle is unable to overcome the load. In practice these are all working operations in which there are elements of maintaining weights, tools or uncomfortable posture. The static operation leads not only to the development of fatigue, but has pathogenetic factor for diseases of the skeletal muscular system - myositis, tendinitis, tendovaginitis and the like. For example, in tiling of teared fibers hands of spinners are extremely unfavorable posture - raised up and this static tension, repeated a hundred times in the course of the working day is pathogenetic factor for the development of professional damage to the shoulder joint.

Different types of physical work require different muscle strength and hence the inclusion of different strength and amount of muscle (muscle mass). Physical work is defined as common as 2/3 of muscles are engaged to perform it. Typically, such work requires great energy expense and often refers to the category of heavy physical labour. In local muscle work involved mainly the muscles of the hands. This kind of work is widespread - meeting at different manipulative activity, at work on conveyor, assembly work and many others. Very often, the effort required to perform labour operations is not consistent with the smaller muscle groups of hands, working often with great pace and rhythm imposed. Occupational injuries sceletal-muscular system in this type of work are common. In the expert opinion of work, to take professional nature of the disease, using the following classification of working movements:

1st class - are involved muscles of the fingers and wrist;

2nd class - are involved muscles of the fingers, wrist and forearm;

3rd class - are involved muscles of the fingers, wrist, forearm and elbow;

4th class - includes muscles and arms;

5th class - in movement is included and shoulder.

For prevention it is important to know that with the increase of the class of movement their rate must be reduced. It is considered that for small classes of movement optimal time for performing the movements should be 0.3-0.4 s, which corresponds to a rate of about 2.4 movements s., and for large classes - 0.7-0.9 s. Another classification proposes to distinguish three degrees of rhythm of work:

Moderate rate - up to 20 movements of the arms or legs and the body 10 for 1 min.

Average rate - from 21 to 40 movements of the arms or legs and 11-20 body movements for 1 min.

High rate - more than 40 movements of the arms or legs and over more than 20 of the body for 1 min.

2.1.2. CATEGORIZATION OF LABOUR

Dosing workload is an important issue through which reach major labour hygienic purposes - maintaining performance at an optimal level long, distances to the development of fatigue, avoid the development of overstain and that related illnesses. Dosing of the workload is important, especially for the so called vulnerable groups - persons after have ill in the recovery period, women, new or young workers, subjected to reassignment. The categorization of physical labour is legally governed and greatly assist in solving these issues. Classification of work is important for the standardization and remuneration of labour.

The criteria for the degree of physical labour is mainly based on indicators of energy consumption, pulse rate and indicators of external breathing.

Energy consumption is very accurate quantitative indicator to determine the heaviness of labour for severe and moderate loads and less accurate at light loads and in static operation. Energy consumption can be measured in kcal/kJ / for 24h, for 8h, for 1h or for 1 min. Daily balance of energy consumption is the sum of power consumption during sleep (average for 8 h about 560-600 kcal - 2300-2500 kJ, i.e. approximately basal exchange), energy consumption in their free time (1100-1200 kcal - 4,600-5,000 kJ, or twice the basal exchange for 8h) and energy consumption for 8 h work (in heavy and very hard work can reach 2500-3000 kcal - 10500-12500 kJ). This is an estimate of energy consumption in uniform for the whole day activity.

In practice, the most common classification of work is based on a reading of energy consumption for 1 min. This involves measuring the energy consumption at the main operating procedures and calculation of averages based on timing research. Thus in addition to the average energy expense for a given profession we can have and details of peak moments and worst working operations in a profession. On **Table. 1** is a classification of work adopted in our country.

Tabl.1.

	Category	Man kcal/min	kJ/min	Wemen kcal/min	kJ/min
1	Light work	up to 3	up to 12.57	up to 2.4	10.04
2	Moderate work	3-5	12.57-20.95	2.4-4	10.04-16.72
3	Hard work	over 5	over 20.9	over 4	over 16.72
4	Very hard work	over 6.5	over 27.3	it is not allowed	it is not allowed

Note: *Power consumption is presented in gross values.*

The data presented in Table 1 are average for both sexes. When we need to assess the possibilities of a person to perform a certain kind of work best to use categorization consistent with individual possibilities. This approach is particularly important in reassigned workers. The assessment is made on the basis of maximum aerobic capacity (or maximum working capacity - MWC). It is believed that person for a long time may perform work that is 30% of its MWC. For example, if the person at its maximum load (to failure) can reach energy expense 12 kcal/min (50 kJ/min), then work with energy expense 3.6 kcal/min (15 kJ/min) is practically no fatigue him. Based on the MWC's work is classified as follows: 1. Light work - load is 10-20% of the MWC; 2. Moderate work - load is 20-30% of the MWC; 3. Hard work - load 30-40% of MWC. 4. Very heavy - load over 50% of MWC.

In our country has adopted the following categorization of labour according to pulse frequency:

- Light work - up to 90 beats/ min.
- Moderate work - from 90 to 100 beats/min.
- Hard work - from 100 to 110 beats/min.
- Very hard work - over 110 beats/min.

Pulse rate can reach up to 200 beats/min. at sporting events in well-trained athletes. It is believed that above these values begin to suffer supply the heart muscle with blood. In workload heart rate should not exceed 130-135 beats/min. On **Table. 2** shows the valaes of the pulse rate in one of the most hard operations in same professions.

But by working pulse rate, the heaviness of labour can be characterized by the dynamics of the heart rate during recovery. It is estimated that in the first minute of recovery pulse rate should not exceed 110 beats/min and the third can be below 90 beats/min.

Tabl.2.

№	Profession	Business operations	Pulse in beats/min
1.	Miner (underground)	Perforate	125-135
		Caving	132-138
		Fastening	130-135
2.	Miner (open pit)	Drilling	114-120
		Bomber	125-130
3.	Woodcutters	Felling trunk (manual chainsaw)	130-135
		Limbing	120-130
		Measuring and sorting	120-125
		Landslide logs (with animal traction)	128-135

In assessing indirectly for energy consumption can be judged by oxygen consumption, but also by lung ventilation. It is believed that oxygen consumption to 0.5 l/min. meets the light work, from 0.5 to 1.0 l/min - moderate work and oxygen consumption over 1.0 l/min corresponds to the very hard work. The determination of energy consumption on lung ventilation can be done only in those activities in which are included large muscle groups.

The ability pulse to be accepted as an indicator of the heaviness of labour stem from the fact that oxygen consumption and heart rate showed a high correlation coefficient up to the critical values of oxygen consumption around 3.0 l/min and heart rate around 200 beats/min., then the increase of the pulse is greater.

In assessing the physical load by pulse rate, should be borne in mind and the influence of factors such as age, sex, fatigue, emotional state, diurnal rhythm and more.

2.1.3. CHANGES IN ORGANS AND SYSTEMS DURING PHYSICAL WORK

1). Changes in the cardiovascular system

Changes in the cardiovascular system during physical load is important for the adaptation of the organism to labour. The state of the cardiovascular system is largely the factor that limits the efficiency. External and tissue respiration have a significantly larger reserves and oxygen deficiency is determined by the ability of the cardiovascular system to pro-

vide necessary for a physical effort circulation. In the resting pulse rate at 70 beats/min and stroke volume 80 ml, minute volume is 5.6 l/min, systolic index (the ratio of stroke volume on 1 kg of body weight) is 1-1.5 and cardiac index (minut volume on 1 m^2 of body surface) is about 2.8. When loads stroke volume gradually increases, but upon reaching the oxygen consumption up to 2 l/min remains constant - about 150-170 ml. These values show that the maximum amount of blood that can pass through the circulatory system is approximately 30 l/min. This allows maximum transport of oxygen 3.5 l. Meanwhile, the lungs can pass about 100 l air to deliver 5-5.5 l oxygen.

Functional setting of the cardiovascular system is aimed at creating adequate circulatory balance to ensure the necessary substantial humoral exchange of tissues, according to the increased requirements in connection with the work performed. In hemodynamically terms this means to deliver relevant inflow of arterial blood and out flow in the vein of the working body and flow of venous and arterial blood to the heart. The setting of the circulatory system becomes by neuro-reflex tract, allowing greater speed of changes. The implementation of neuro-reflex regulation includes a series of mechanisms in which the main factor is the heart, but along with it however no small role playing and peripheral circulatory system, the movement of muscles and respiratory system.

First with the start of a movement (or before him - conditional reflex reaction) becomes redistribution of blood volume. It is pushed from blood depot in the abdomen to working muscles. Abundant blood supply to the muscle allows for their contraction to push a significantly greater quantity of blood to the veins. Thus muscles act as an additional pump that assists the heart in particular with respect to venous flow. Respiratory movements also assist venous flow through sequential decrease and increase intraabdominal pressure. These mechanisms provide quick and plentiful supply of blood to the heart.

Filling of the heart depends on the duration of diastole. In fast heart rate reduces its duration, so harmonious consistent output of venous flow and duration of diastole is essential for efficient and economical cardiac output. This is achieved by a complex neuro-reflex regulation featuring baroreceptors of v.cava and proprioreceptors to working muscles, and also with the participation of humoral regulation of simpatico-adrenal system.

When exercise the maximum blood pressure is generally increased in healthy people, with this increase is the greater, as the intensity is greater. Minimum blood pressure rises slightly, remains at the same level or may drop slightly. In this regard, the pulse pressure increases and serves as the sign of the stroke volume of the heart. Of this normotonic kind of reaction differ hypertonic type, asthenic and dystonic type of reaction, that sign of untraining of the cardiovascular system or pathological changes in heart muscle or peripheral vascular system. In static working increases the maximum blood pressure significantly, but are also being increased and the minimum blood pressure, pulse pressure showed no significant increase.

At muscular work cardiovascular activity passes through two stages: the period of inworking and a period of steady state. The period of inworking of the cardiovascular system is short - from 20-30 s to 2-3 min. We must distinguish between the period of inworking at physical load and the period of inworking at the beginning of the work shift, which is considerably longer and its mechanism lies the principle of formation of dynamic stereotype, and adaptation to the complex working conditions.

Especially important is the recovery period after the termination of the load. We distinguish early stage of recovery and stage of actual recovery. Early recovery takes 30-60 s and is characterized by a steep drop in the pulse rate and the maximum blood pressure. In the second stage of the recovery, the parameters of the cardiovascular system is closer to the initials. The development of recovery has exponential curve and depends on load. In samples with dosed exercise at the end of the third minute to reach full recovery. In static work immediately after switching, pulse rate remains unchanged and the recovery process proceeds more slowly.

In well-trained individuals the response of the cardiovascular system to a moderate load is characterized by a smaller amplitude changes - pulse rate is rising less, and the recovery is faster compared to the untrained individuals **(Fig. 1)**. At maximum load (denial) untrained persons meet with greater frequency pulse.

2). ECG changes

Physical work needs increased myocardial oxygen and blood flow in the coronary vessels. This can lead to a inconformity between the demands of the myocardium of oxygen and entering in coronary vessels blood. Electrocardiography allowed to express themselves inadequate in terms of myocardial loading. ECG changes also reflect the modified speed and direction of blood, changes in neuro-vegetative tone. These changes can be visualized primarily through two approaches - classic when ECG is recorded at the time of load (although difficult using no standard - leads) and is registered in the first 15 s, after loading, of the first, second and sixth minute of

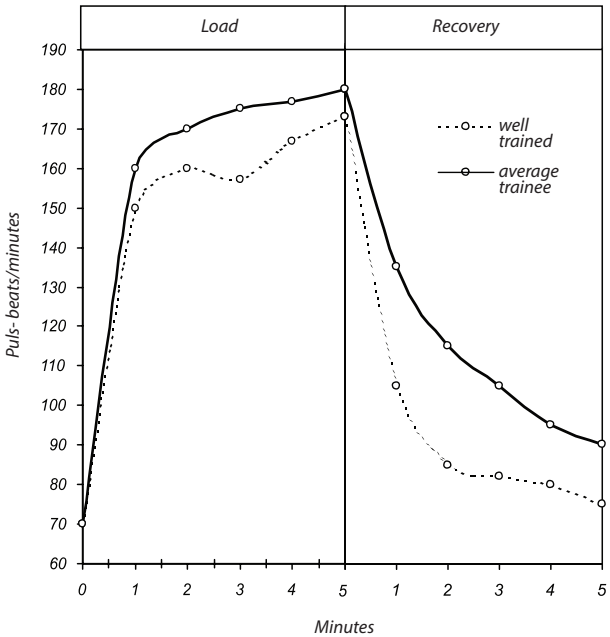

Fig. 1. Effect of training (on Brua)

recovery, or at conditions of employment and recovery, by recording of Holter systems.

Important for assessing the state of the myocardium is the application of samples of dosed exercise intensity and duration, as submaximal and maximal loads are achieved gradually or stepwise with continuous monitoring of changes in pulse rate, blood pressure and ECG. Ergometry bicycle allows to accurately implement dose and standard load and be quickly terminated at the appearance of indications. It is generally accepted that in order to provoke the cardiovascular system and manifest ECG changes, pulse rate with loads should reach 160-170 beats/min. Most often and best manifest ischemic changes in left precordial leads (V5). They are expressed by lowering ST-segment associated mostly with reduction or inversion of the T wave. Decreases in ST below 2 mm horizontaly and/or descending, sure evidence of myocardial ischemia, occurred as a result of a discrepancy between physical activity and opportunities of coronary vessels.

Close by informative are ECG recordings immediately after the implementation of the two-staged test steppe Master or other standard workloads that do not require sophisticated equipment. Step Test Master successfully used both in dynamics during the working day, and in assessing the degree of reduction of physical efficiency in workers spent some somatic, infectious or diseases of the cardiovascular system. This is particularly important for workers engaged in moderate or hard physical activity. As an expression of pathology accepted criteria: 1). Occurrence of dyspnea, cyanosis, cardiac pain; 2). Sinus tachycardia with heart rate above 120 beats/min, paroxysmal tachycardia or arrhythmia, extrasystole; tachycardia after 3 minute break; 3). ECG changes in the conduction system; 4). ECG changes in the interval ST and T wave.

3). Changes in muscle

One of the important conditions for effective work and out of fatigue is good blood flow to the working muscle. If the muscle has good conditions for the supply of oxygen, anaerobic decomposition processes may even before the formation of lactic acid being switched to aerobic cycle of tricarboxylic acids. Thus, if observe to specific conditions, such as the supply of carbohydrates, the work can be performed for a long time. The maximum flow rate of muscle depends on vasodilatation and the minimum of force of muscle contractions. Maximum flow of muscles in a dynamic work is determined by 140-200 ml/min per 100 g muscle against 2-3 ml/min at rest. In static operation, if the force of maintenance does not exceed 20% of the maximum capacity of the muscle, increase blood flow can reach 15-30 ml/min per 100 g muscle mass in which work can continue for a long time. Above 70% of maximum opportunities blood supply is extremely limited and the work can last only a short time.

4.) Changes to the respiratory system.

The respiratory volume increases linearly with the increase of the load and of 500-600 ml/min can reach up to 2000 ml/min. The effectiveness of the increase is limited by the increase of respiratory dead space. Minute respiratory volume of 6-8 l/min can grow up to 100 l/min, as its limitation comes from increased respiratory dead space with shortness and frequency of breath. At that oxygen consumption increases linearly with breathing minute volume to certain values - up to 2 l/min, after which the growth of minute respiratory volume is ahead of that of oxygen consumption. Maximum oxygen consumption in well-trained men can reach up to 3.5 l/min, which is limited by the cardiovascular system.

Reduced physical activity

In a number of modern professions (operators of control desks, apparatuses, working on a video display, workers in offices, management staff and many others, are increasingly present adverse effects of factors **hypokinesia** (reduced physical activity). In another large group of professions carried out a large number of manual operations, mainly from small muscle groups of the arms - **hypodynamia** (occupation of the conveyors, installers, workers from the electronics, textiles and clothing, etc.). Because the consequences of hypodynamia and hypokinesia of the body slightly differ, used the general term "reduced mobility". At the professions reduced physical activity is added and the deficit of the social-mode of life physical activity.

The changes in the body as a result of reduced physical activity in less movable and less dynamic professions often arise on background of the prolonged static exertion of muscles, providing forced standing or sitting posture, resulting in changes in performance due to both factors.

The main factor in the mechanism of hypokinetic and hypodinamic changes is to minimize the load on the cardiovascular and musculoskeletal system. This led to a decrease in the level of functional status of these systems and their detraining and reduced capacity. Detraining of cardiovascular system is exacerbated by postural fluid redistribution in the body where the blood circulating in vessels lost their normal tone. Under these conditions humoral regulation of the cardiovascular system is not able to provide a influx of the required amount of blood (oxygen) to the brain, which is one of the main preconditions for the reduction of mental performance and development of fatigue. By a number of authors found that the immobilization results in a reduction of the activation process and from there to considerable disturbances in the central nervous system, the hormonal changes, water-salt and energy metabolism.

Detraining in conditions of reduced physical activity leads to sharply reduce the possibilities of functional systems. Very common are inadequate reactions - increased intensity of muscle work irrelevant to the physical load, unproportion movements. Violations in coordination functions of locomotorium in turn make it difficult to adapt to working conditions and workload. In these professions it is hard to staff at the beginning of the working day and slowly reaching steady working condition.

Decreased physical activity in modern society leads to so-called **hypokinetic disease**. In it are characteristic changes in the central nervous system (reduced activity of excitation processes, anxiety, drowsiness, emotional instability), disorder in the metabolism, disorders of the gastrointestinal tract and the like. Decreased physical activity and neuro-psychological tension in contemporary professions are and etiology of cardiovascular diseases.

Most important for the prevention of adverse effects of reduced physical activity are events aimed at creating a rational regime of work and rest in which the principal is the implementation of active intershifts rests. Lengthy period of inworking gives base to propose the introduction of a regulated rest at the beginning of the work. This is so-called opening or introductory active recreation. The complex of exercises performed before or 10-15 min. after starting work, contributes to more rapid introduction into the labour rhythm. Later during the work shift active rests must comply with the specificity of the work and the dynamics of the performance.

In addition of intersfifts prevention of the effects of reduced physical activity it is particularly important and conducting activities outside working hours, aimed at improving the physical training of the body.

2.2. NON-PHYSICAL OR MENTAL WORK

Mainly issues related to determining the weight and classification of non-physical or mental work are much more complex than in the physical. More complex are the problems of mental fatigue and exhaustion and health consequences of them. If physical work can accurately determine by the energy expended and on this basis or on the basis of the working pulse can be categorized and normalized labour, it is impossible in mental work. **First**, it follows from the fact that as the increasing intensity of metabolic processes in the brain, due to the relatively small volume to the body, they insignificantly affect the total energy expense. **Secondly** this "working" organ is incredibly complex and many sides of its operations are still in the area of hypothetical explanations. On the other hand mostly mental work, because of the different requirements for the basic neural processes, analysts, psychomotor, varying degrees of involvement of hormonal mechanisms, and emotional sphere is very diverse. Therefore, one of the approaches for studying the workload and strain in this kind of work is it to be classified based on mainly involved in its work processes and on this basis to analyze and evaluate the intension of labour.

2.2.1. CLASSIFICATION OF MAINLY INTELLECTUAL WORK

1. Work with predominantly sensorineural intension.

In this type of work they are loaded mainly sensory organs, mainly the visual and auditory. These are so called observer occupations such as operators, equipment operators, proofreaders and others. Of course, observation activity involved and central parts of the analysts, basic neural processes and especially processes of attention, but if the work is not complicated, the load is mainly on visual or respectively auditory analyst. Usually when evaluating this type of work are examined changes in hearing sensitivity of the analyzer, fatigue accomodation mechanisms, changes in the electrical sensitivity of the eye (ocular reobasis), critical frequency signal mergence and others. From the degree of change at the end of the day assessing the intension of labour. Further-

more, in the physiology of labour there are classifications of the intension of the labour on the basis of number and size of observed objects, changing their perimeters per unit time, the relationship between relevant and "noise" signals etc.

2. Mostly sensoryneural and psychomotor work.

In this type of work, to the reflex arc includes motor reply. In practice very often a signal requires a fast motor response. The most typical example of this kind of work is the work of drivers, crane operators, dispatchers, machine-operators. Besides intension of sensing systems and load on the same kinematic chains, these professions require rapid differentiation of signals and decision, brought great demands on attention processes and more specifically to its two main qualities - distribution and sustainability. The assessment of load is based on a changes, in the course of the day occurred in the sensitivity of sensory organs, in time for the implementation of simple and complex sense-motor reactions changes in attention and working frequency pulse.

3. Work with mostly emotional stress.

Emotional stress is most pronounced in occupations with greater responsibility. It is believed that the responsibility is least if the worker is responsible only for products providing his salary. It is greater if he is responsible for ensuring the work of many people, even more if mistakes can cause accidents or loss of life. The greatest degree of responsibility in occupations that are responsible for many lives. As an example of working with great responsibility may indicate the work of drivers of motor vehicles, airline pilots, air traffic, drivers of locomotives, operating personnel in nuclear plants and others.

In professions and activities with greater responsibility very often require rapid decisions, decisions on choice of several alternatives, which implies knowledge and experience, flexible memory and thinking processes. Making decisions in conditions of deficiency of time makes this work particularly stressed. Significant emotional intension exists in professions that require contact with many people - teachers, managers, working with clients. The evaluation of the intension is performed by assaying the level of excitation processes in the CNS, the amount of excretion of stress hormones - adrenaline, noradrenaline, corticosteroid hormones, evaluation of the reaction of the stress of the person, the dynamics of the working pulse rate and the like.

4. Work with mostly informational load.

In this type of work load is mainly in terms of information and the memory processes. Work in a variety of occupations such as bank clerks, accountants, cashiers and others such processing, requires a large amount of information during the workday. Here goes the work of students, researchers. In these professions is engaged more and visual analyzer, and also processes of directed attention. The evaluation of the intension of work is done on the basis of changes occurring in the course of the working day in the visual analyzer, the maximum amount of processed information per unit of time, changes in the sustainability of attention, pulse rate and more.

5. Mostly creative work requires the involvement of higher thought and associative processes, a large amount of stored information and mobility of memory processes. This type of work related professions of architects, engineers, doctors and others. The basis of creative work lies formation of dominant. This creates a selective and purposeful mental activity. All mental creation, all associations and knowledge are directed to a particular site and the stream of thoughts is not diverted to adverse events. Moreover, in permanent employment, dominant thought process can even be supported by adverse stimuli. On this occasion Uhtomsky wrote: "The mind burdened with an idea suddenly finds mechanism for resolving its, among side impressions on a trip in the mountains, on a sunny day or walk in the street crowd."

In this type of work, there is another very characteristic. This is the fact that a strong dominant peculiarity can not be easily extinguished. Working day in many professions is far from finished with the completion of work time. These features make it difficult to apply the regimes of work and rest in creative professions. Fatigue and stress often arising in solving difficult and important tasks are important health and social problem here.

2.1.2. CHANGES IN ORGANS AND SYSTEMS IN MENTAL WORK

Main characteristic of the condition of the body in mental work is low gas exchange. In intensive neuro-psychological work maximum oxygen consumption increases about 6% and the evolved carbon dioxide - by 2%. These relatively small changes in total gas exchange is not respond, however, for low intensity of metabolic processes in the brain (the intensity of metabolism per 100 g of brain substance is much higher than that of 100 g muscle).

Changes in the frequency of the working pulse and blood pressure depend on the type of mental work. The pulse rate increases significantly at neuro emotional intension. Conversely, the difficult mental tasks run optimally at lower levels of activation, so that the pulse rate even showed a downward trend. In many professions that require a focus, inclusion

of memory and thought processes, pulse rate is low adapted indicator of labour tension **(Fig. 2)**. The same is largely true for changes in blood pressure.

Undoubtedly the most significant changes occur in the nervous system. First are the changes in the activation process. The effective implementation of each task requires a certain level of activation. Above and below this level efficiency is reduced. The low level of tonic nonspecific activation during night shift work is a major cause for difficulty maintaining attention. Furthermore, the inclusion of specific physiological activation processes as a result of external stimuli, depends on what condition they found the nerve centers in relation to activation. In actively functioning system of self-regulation (by way of descending projection system) achieves necessary, according to the task, level of activation. Where tasks or relevant signals occur rarely, such as when working on some control desks, the level of activation decreases, develops so-called monotonous due to deficiency of information.

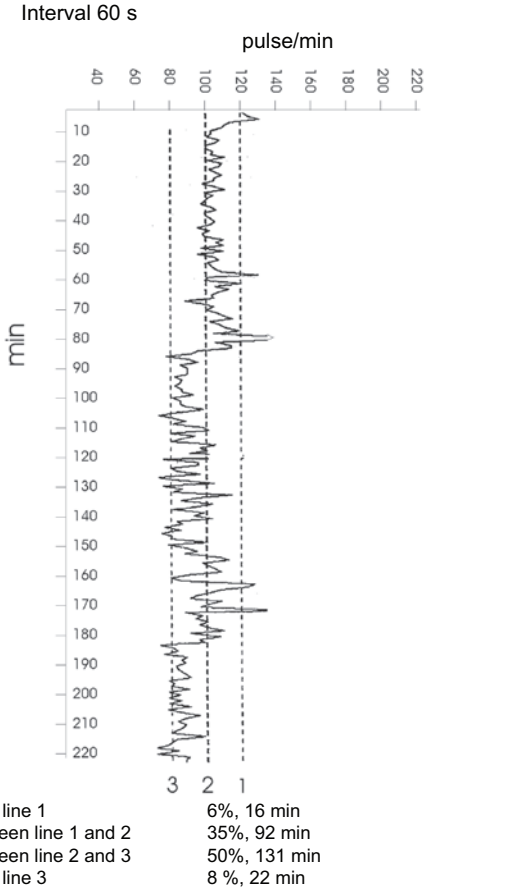

Fig. 2. Changes in pulse rate during student exam:
- More frequent pulse rate when towing a ticket (80/min)
- Reducing the pulse rate frequency pulcovata between thinking (90-150/min)
- Increase the frequency in responses (150-180/min)

The frequency response of the background EEG activity gives a real idea of the level of excitation processes in the central nervous system. The picture of EEG activity is very complex, it becomes even more complex with active mental work and to assess the changes required complex machining analysis. Therefore, this method has not yet been entered in the broad practice of the physiology of labour. The same can be said of methods of induced brain activity - induced in response to a stimulus or task potentials (evoked potentials). In practice, to assess the changes in the nervous system successfully apply a range of behavioural methods such as time of simple and complex sense-motor tests, threshold sensuosness of sensory analysts, tests to assess the quality of attention and others.

2.3. WORKING CAPACITY

The definition of efficiency performance, as the ability of man to maintain longer a level of productivity without occurrence of pathological changes in organs and systems is valid for both physical and mental performance. The factors which determine performance are also valid (of varying severity) for one or the an other kind of work. Efficiency can be determined by indicators of productivity (qualitative and quantitative), or by changes in the functional state of organs or systems. As a result of the work, performance varies over the course of the working day and working week, as well as during the length of service.

The factors, that determine the efficiency can be included in the following groups: 1. factors associated with severity and organization of work - duration, mode of work and rest, shift work, working movements, posture; 2. factors in the workplace and environment - microclimate, noise, toxic factors, lighting, etc .; 3. individual qualities of man - age, gender, length of service, experience, motivation, health status, etc .; 4. psychosocial factors, associated with working team.

The dynamics of the performance during the workday depends primarily on the type and level of work performed. In moderate physical work, manipulative or instalation work for example, usually in the dynamics of performance detect three phases. The first phase is the period of the inworking, over who is restored or develop dynamic stereotype, productivity increases and operating frequency pulse gradually decreases. The period of the inworking may continue until 2 h. The second phase is the period of steady performance, which in a rational organization of labour is the longest and is characterized by stable production indicators and pulse rate.

The third phase is set at the end of the working day when performance begins to fall and pulse rate as a result of coming fatigue increases **(Fig. 3)**.

In this classic curve of performance, some authors distinguish 7 phases or periods: 1. Period of the inworking; 2. Period of maximum performance; 3. Period of full compensation when there are already tired but it is offset by willful effort or high motivation; 4. Period of unsteady compensation - sometimes performance increases, then decreased; 5. Period of final revival - efficiency decreases, but productivity can be increased; 7. Interruption of work and development of fatigue.

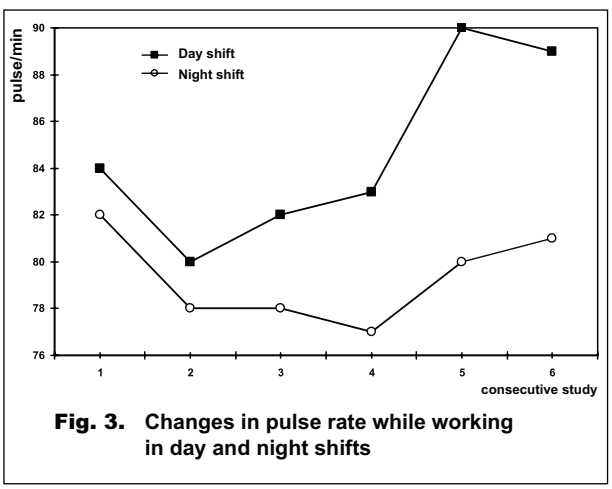

Fig. 3. Changes in pulse rate while working in day and night shifts

In many modern forms of work, the classic curve of performance has not been established. For example, in many automated activities, basic intension develops in the beginning of work, followed by an extended period of monitoring. The period of intension can be delivered before the work commences. The so-called anticypatic stress (or intension) is associated with thinking, planning, making important decisions related to the work ahead.

Regularity in changes in performance are identified and during the working week. The first day somewhat conform to the inworking. The highest performance is in the middle of the week and at the end already feels fatigue.

Physical performance

The level of physical performance depends on the functional state of the respiratory and cardiovascular system, the morphological and functional properties of the muscle, the adaptation capabilities of the muscle metabolism, i.e. of efficiency performance or physical capacity. Second rank factors of work environment such as microclimate, dust, toxicochemical impacts, motivation, psychosocial factors.

To preserve the health and performance, it is essential workload to match the capacity of the worker. This even more largely true for persons with reduced capacity due to chronic illness or period of rekovalestsentsiya. To determine the physical ability to apply different functional samples loaded the body in varying degrees. There are two main groups function tests - with moderate load with submaximal or maximum load.

1. The tests with moderate load refer orthostatic test, Martynez test, step test Master, Harvard step test. To them - before, during exercise and during the recovery account the reaction of the cardiovascular system (pulse rate, blood pressure, ECG changes). The results of the reaction of the body are more favorable as the changes are less marked.

2. Tests with submaximal and maximal loads are aimed at determining the maximum aerobic capacity and should be applied only in healthy people. Determination relates to the application of loads to fail, and as the capacity is greater, the higher values reaching oxygen consumption, heart rate, maximum blood pressure. Even with well-trained persons maximum oxygen consumption rarely exceeds 3 l/min, while during the same time through the lungs can pass 6 l/min oxygen. It is believed that the load is optimal when the person works with 30% of their maximum capability.

Physical performance can be significantly increased as a result of training. During the training, the body goes a number of morphological, physiological and biochemical changes that lead to better adapt to the work and increase efficiency. The changes affect the whole organism, the most significant are expressed in respect of muscular and cardiovascular system.

As a result of system loads the size of the working muscles increases, a to account of thickening of the muscle fiber. However, in the muscle grow more capillaries and blood circulation improves, which is a prerequisite for improving metabolic processes in muscle contraction. Transmission of nerve impulses in the motor plate also improved, while each pulse triggering more fiber. All of this leads to increase strength, endurance and speed of muscle. Due to the unevenness in the pass of processes these properties can be achieved before the apparent increase in muscle mass.

Significant changes in the process of training occur in the heart muscle. In an untrained organism increasing needs of oxygen to working organs are satisfied mainly by accelerates the heart rate. In trained individuals, sending more blood to the muscles to account of increasing stroke volume of the heart. It is also affected and blood pressure. In untrained persons continued work leads to progressive decline in systolic blood pressure, which is an expression of exhaustion, while trained persons that effect recedes.

During the training participate most actively and nervous system. Repeated exercises lead to proper interaction between the CNS receptors, motor organs, respiratory and cardiovascular system. Based on the improvement of basic neural processes and conditional - reflector links, being set up and dynamic stereotype, resulting in improved coordination and automation of movements.

On the basis of the new features of the trained body, are and significant biochemical changes. Increases energy potential (macroergic phosphate groups, liver glycogen), changing the amount of myoglobin. Increase in the enzymatic activity, the destruction and the formation of glycogen. The increased buffering properties of the blood lead to a better toleration of acidosis. The training becomes by special programs that rely on such basic principles as repeatability, gradually increasing the loads, complexity, consistently, etc. These principles are basic in the manufacturing training.

Mental performance

Though the basic principles of efficiency are generally valid for mental performance, it has some important features compared to physical. Firstly it is pronounced interindividual differences in the course of major psychiatric processes and activities, and hence the different capabilities of individuals for the performance of one or another kind of task. Secondly this is its strong influence on the motivation, which often leads to masking advancing fatigue. In mental performance, there is another important feature, this is the great flexibility and the ability to change the strategy in a change in functional status, which often mask fatigue.

Taking a slightly different aspect of mental performance is reliability. It is seen as probability to readiness for flawlessly, faultlessly and timely execution of a task and in extreme conditions, as and opportunities for fast recovery. The reliability of the human unit in the system "man-machine" in conditions of a large production risk is essential. Factors that lead to reduced mental performance and development of fatigue, adversely affect reliability.

At the level of perception mental performance depends on the functional state of auditory and visual system. Functional state of the visual system in the first place is determined by visual acuity. Visual acuity for near is particularly important parameter, since many manipulative professions, assembly services, working with texts, the distance from the object to the eye is 30-40 cm. For occupations requiring monitoring moving objects (eg. monitoring radar screens by flight head) is important kinetic visual acuity. Uncorrected hypermetropia and presbyopia lead to rapid fatigue. For performance of the visual system are also important adaptation options to light and to different brightness, color vision, disturbances in accommodation.

Reduction in auditory sensitivity to noise exposure, deteriorated speech clearness, difficult the adoption of useful information and leads to faster nervous fatigue.

Optimal tonus of the brain cortex is essential for optimal functioning of mental activity. Above and below this optimum effectiveness is reduced **(Fig. 4)**.

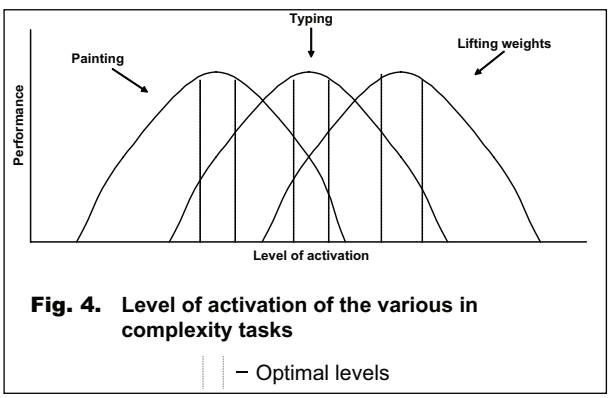

Fig. 4. Level of activation of the various in complexity tasks

In active functioning of the system of self-regulation (the path of the descending reticular formation) is achieved requested, according to the task level of activation. It is considered that the effect of self-regulation underlies at U-shaped relationship between the level of activation and performance - and at low and at very high levels of activation performance deteriorate. In this, complex and complicated task requiring a lower level of activation as compared with the simplest task.

Circadian rhythm is a major factor that affects the activation processes. In connection with the existing two main types of curves in body temperature - a peak in the early afternoon and peak in late afternoon hours, shaped and so-called evening and morning types of people. Evening (introvert) type is unable to work in the morning and morning (extraverted) - in the afternoon. Introvert persons starting day with a higher level of activation as compared with their morning extraverts and their performance in the same task is higher **(Fig. 5)**.

Mental performance also depends on a number of properties of the nervous system such as strength and agility of the main nerve processes, capabilities of supporting sustained attention. The rapid switch over of attention from one object to another is particularly important in some professions such as operators, equipment operators, dispatchers, drivers of motor vehicles and others. For mental performance are important the memory capabilities of person,

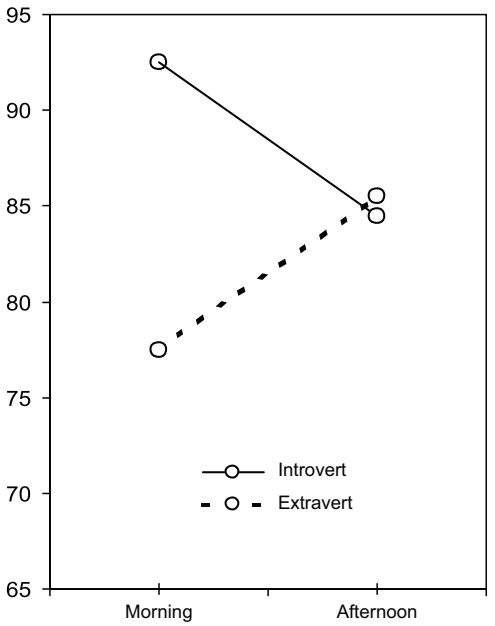

Fig. 5. **Daily dynamics of performance at intro- and extraverts**

profound mental and associative processes. High performance especially in creative tasks are based on the principles of building dominant. This largely explains why in many modern forms of work performance curve is different from the classic.

2.4. FATIGUE

For many authors fatigue is a major protective phenomenon that exists in all living beings and from this perspective should be seen as an important link in the system of self-regulation. One of the classic definitions assume that fatigue is temporary and reversible decrease the ability of the body to carry out physical, mental or psychological activity as a result of previous work. Unlike fatigue, over work regarded as prepathological condition that occurs after the strong loads, or when the work is started again, without being resolved phenomena of fatigue and has reached a state of complete recovery.

Usually classical theories to explain the fatigue can be divided into two large groups. To the first belong humoral-localistic that explain fatigue by exhaustion of energy resources, accumulation of catabolic products. Another group of theories based on the principles of neurism and consider fatigue as a condition that is associated primarily with CNS - imbalance of processes of excitation and detention, violation of regulatory mechanisms and more.

2.4.1. CLASSIFICATION OF FATIGUE

Depending on the nature of the work fatigue is usually: physical (local and general), sensory (visual or auditory) and mental fatigue, which can also be several subspecies.

1. Physical fatigue

General physical fatigue occurs at the inclusion in the work of the large muscle groups of 3, 4 and 5th class of movement. In modern forms of work this type of fatigue occurs rarely, than local physical fatigue, which is very common in manual work and at load the same muscle groups and joints. This fatigue is mostly associated with the depletion of macroergic compounds and failure of anaerobic reactions. It is generally believed that the accumulated catabolic products (lactate, pyruvate), stimulate nerve endings and generate specific sense of diffuse pain in the muscle tissue.

Incorrect performance of the actions of tired muscles most likely is the result of wrong afferent information from muscle receptors, which in turn hampers the control of the central nervous system and leads to a distortion of the dynamic stereotype

2. Sensory fatigue

For practice is important fatigue of visual and auditory system. Visual fatigue meet primarily in activities requiring intension and concentration of eyes when working at close distance, in frequent changes of focal distances (eg. installation of small details, correction of texts, entering data into a computer, etc.), activities that require intension of the muscles involved in the process of accommodation, convergence, reduce the effects of spherical and chromatic aberration. Very often at the end of the working day workers complain of headaches, flushing, itching, watery eyes. These complaints are more frequent and more degree at hypermetrops, and persons after age 40. Some of the symptoms, such as headache, dizziness and general fatigue not due, of course, only by fatigue of eye muscles, and are one integral effect of overload of exceptionally complex mechanisms involved in visual perception. Fatigue may be a result of excessive psychosensorial work or forced maintaining wakefulness.

Auditory fatigue develops as a result of high noise levels - 70-90 dB, depending on the nature of work (degree of sensorineural load). The most characteristic manifestation of auditory fatigue is temporary deterioration of auditory sensation. In this connection, it must first be distinguished from the adaptation, which is also a protective reaction of the body. It is assumed that for adaptation can speak in raising of hearing threshold by 10-15 dB and recovery period to 3-10 min. Changes in hearing sensitivity and

time to recover above these values are regarded as fatigue. Again, as mainly visual fatigue, are appear other signs and symptoms of "general fatigue".

3. Nervous fatigue

Nervous fatigue can be mostly sensorineural, psychomotor, intellectual, depending on which mainly units of the CNS are engaged in labour activity. Much of the features of neuro-sensory covered with ocular and auditory fatigue. Psychomotor fatigue develops in work that besides the load on the sensor system requires quick decisions and psycho-motor effects (drivers of motor vehicles for example).

Mental fatigue in the broader sense or intellectual fatigue are obtained in actions demanding processing and storage of information (the memory processes), requiring solving creative problems by engaging the higher mental activity. The most characteristic feature of mental fatigue is that develops slowly and imperceptibly and signals for rest come later. The strong positive motivation to complete a task, for a long time can mask fatigue. On the other hand what makes mental fatigue less favorable than physical is that physical work after its termination process of recovery begin immediately, while at the mental work extinction of the dominant excited focis can not start immediately. Burdened with solving a task brains "still working" long after the formal working hours. Strong dominant focis do not go out and through the night sleep. Therefore the recovery process run more slowly, and breaks especially if are shorter, are ineffective.

Mental fatigue is manifested by general weakness, headache, often appears tinnitus due to contraction of the peripheral vessels and invasion of blood to the head. In severe mental fatigue appears palpitations and hyperesthesia, appears irritability, weakened self-control. Sleep disorders are also indicative symptom of nervous fatigue.

Mental fatigue should be distinguished from adverse psychic states of boredom and monotony. They arise as a result of specific working conditions, monotony, lack of stimulus or deficit of information, repetition of activities with little difficulty and complexity and are an expression of the decrease in activation processes. Characterized by lethargy, decreased alertness, drowsiness. Monotony can develop into mental satiation, leading to a reduction in productivity, emotional disorders and others.

Reduced capacity may not be the result of work, and also can expressed as fatigue by decrease the functionality of an organ, system, or whole organism - in condition of an irrational diet, lack of oxygen, strong emotional agitation and other adverse factors.

2.4.2. OVERWORK

Overwork is seen as prepathological condition that occurs after very high load or as a result of operating in an incomplete recovery of the body. In this connection distinguish: hyper overwork, acute overwork and chronic overwork.

The classic example of hyper overwork is the historical fact of the death of the Greek warrior run a 40 km by Athens to announce the victory of the Greeks over the Persians in the village of Marathon (490 to J.C). There is also in the pursuit of hunting. The animal stopped, it has an extremely strong dyspnea, tachycardia, fever. Death occurs in a few minutes, followed by a post-mortem numbness. Establish hypoglycemia, increased lactate in the blood, hyperthermia.

Acute overwork develops in hard loads or untrained body. Typical is vibrate weakness. Accompanied with anorexia, insomnia, agitation. Can be monitored and other unwell-defined conditions, and also renal disorders. In healthy people, who do not have any kidney disease is established albumin in urine, can have hematuria or cylinders. This requires close monitoring of individuals. Eg. in sporting activities with hard and very long loads or at short, but with maximum intensity exercises often appears albuminuria. It is believed that it is due to the permeability of the capsule of Bowman, result of lactic acid in blood and hypoxia, as a result of reduced blood flow. Sports albuminuria can last up to 24 hours.

Chronic overwork, occurred as a result of system load after incomplete recovery, is characterized by somatophysical exhaustion and a number of disorders, such as severe sleep disorders, developments neuroses, neurasthenic symptoms, weight loss, loss of appetite. If you do not take serious measures could lead to the development of psychosomatic diseases. So to chronic fatigue should also be treated as a prepathological state that needs of prophylactic and therapeutic means.

2.4.3. SIGNS AND SYMPTOMS OF FATIGUE

Is generally accepted, the changes in fatigue can be divided into **objective** and **subjective**. Besides the subjective feeling of tiredness, fatigue and depression, other symptoms specific to the type of fatigue were described above. Objective criteria can be divided into 3 groups: production indicators; physiological indices and psychological indicators.

1. Deterioration production quality is the earliest manifestation of the objective manufacturing indicators. Reduced coordination of movements and

violations dynamic stereotype, led to an extension of time for individual working operations, of micropauses and reducing the quantity and quality of production. Increased errors and accidents. One explanation of this fact is that the conflict that plays out between the subjective symptoms of fatigue and obligations which the worker must perform on behalf of overcoming fatigue leads to anxiety and nervousness, haste and wrong actions. Moreover nervous fatigue, leads to deterioration of attention (concentration and distribution of attention), to reduced vigilance, which also increases the risk of accidents and deterioration.

2. Physiological indicators. The assessment of the level of development of fatigue is done by assessing the functional status of the involved organs or systems or changes in the functional state of the systems, ensuring their work and, in particular, these are changes that occur in the cardiovascular and respiratory system.

The first group performance related reduction in muscle strength and endurance (muscle strength in the arms, or waist muscle strength), raising the threshold of auditory sensitivity, changes in eye reobasis or frequency of critical fusion of signal, changes in accommodative and others. Of course, these indicators provide information not only for the working body, but also for functional state of the CNS.

The second group of indicators related changes in the cardiovascular system - increase in pulse rate and longer recovery time, increase the variability of heart rate, blood pressure changes, indicators of external breathing, increased metabolism energy. It also covers a s of indicators that provide information about the functional state of the endocrine system.

3. Psychological indicators. This group includes a series of tests and methods that provide information about the various neuro-psychological processes - time of perception, time of simple and complex neuro-sensory reactions, pass of complex thought and decision-making processes, processes of attention.

2.4.4. PREVENTION OF FATIGUE AND OVERWORK

When considering means to combat fatigue must be considered, as some common general performances and also specific measures depending on the type of fatigue and its stage of development. Measures to combat tiredness and overwork, can generally be divided into 3 groups:

1. Prevention of fatigue

2. Means of increasing the efficiency

3. Treatment of overwork.

1. Measures of the first group placed first in fighting fatigue. They are always specific, depending on the nature of work and the characteristics of the particular professional group. The events may relate to solving these important problems.

a) Improvement of the working environment and furnishings. These include measures to reduce the intensity and exposure factors such as noise, vibration, adverse microclimate, dust, toxic hazards, insurance as required by the work, required illumination of the workplace, heating, ventilation, personal protective equipment, etc.

b) Rational organization of the labour process, which is particularly important proper organization of the working movements and work to the maxial and optimal work area, posture, appropriate mode of work and rest. These include also ergonomic requirements at the workplace, working furniture, machinery and equipment.

c) Optimizing workload - optimal regime of work and rest. Optimal workload means not to cross the line behind which quickly comes "exhaustion" and occurs fatigue, but also on the other hand it must not be below a certain lower limit beyond which adaptation to the labour are difficult, develop adverse psychic conditions such as monotony, due to deficiency of information, boredom, dissatisfaction with the job.

2. Means to increase physical and neuro-psychological capacity refer to targeting specific to employee events. An important condition is the physical and mental work to respond of the physical capacity and neuropsychic features of the person.

Adopted in our country physiological norms and rules for manual handling of loads (Decree № 16/ 31.05.1999) state that:

2.1. The distance for regular manual weight carrying is limited to 2 m in women and 30 m in men (when moving on stairs - 1 step = 1 m distance);

– In carrying 2 m distance allowed weight up to 15 kg for women and 50 kg for men for single load and consequently to 4 t for women and 10 t for men - a total shift;

– When carrying up to 30 m (only for men) - 30 kg single load and 6 t total at shift.

2.2. Indicate also the requirements for permissible laden weight for group carrying - less than the sum of those for individual loads as well as the intervals at repetitive operations - not less than 30 s.

2.3. Prohibit regular manual work of loads, including also vehicles, by young people from 15-16 years of age. In a single lifting the load is limited to 8 kg for women and 15 kg for men (age 16-18 years values are 10 kg or 20 kg, and regular work is forbidden).

For increasing the efficiency are widely used:

a). Exercises and training. They are particularly important for physical work and manipulative activity. In the process of training whose basic principle is the gradual increase in load achieved the desired changes in the structure of muscles and metabolism of body. By improving coordination of movements and build a dynamic stereotype movements become more efficient and require less energy consumption. Of great importance is to increase the capacity of the cardiovascular system.

A number of neuro-psychological processes are also subject to training - memorable processes, processes of attention, increases sensitivity to the perception of the relevant signals. Upon exercise increases lability of CNS, conditional reflex connections become more sustainable, improving relationships between I and II-signal system.

b). Healthy nutrition, in certain cases and protective-professional nutrition, are also important to recede fatigue.

c). Raising the general resistance of the organism by hardening and gymnastics is also one of the means to recede fatigue.

3. Means for removal and treatment of overwork.

The best is the removal of overwork going through physiological means - providing adequate rest, good sleep, rational nutrition. Recently widely discussed and application of chemicals to enhance performance and prevent overwork. Chemical stimulants are generally divided into two groups: metabolic and neurogenic.

a). Metabolic stimulants included in intensive metabolic processes that accompany muscular work and reduced adverse humoral changes that lead to fatigue. To this group stimulators belong, glucose phosphate, vitamins that affect oxidation processes - vitamins of group B, vitamin C, as well as trace elements necessary for the enzyme system and coenzymes. These and similar means are physiological and can be used in practice at an appropriate dosage and individual control.

b). Neurogenic stimulants act primarily the nervous system. These include fenamine, caffeine, ginseng (Eleutheroccus senticosus, Panaxia ponicum), echynopsine al., whose effect is manifested primarily through increased efficiency of cortical cells and weakening processes of inhibition. Regardless of the effect of these substances to enhance performance they recede protective effect of inhibition and operations realized at account of the exclusive consumption of energy resources. They are not harmless and should be applied only in rare cases.

c). Depending on the symptoms of overwork are selected and other means of treatment such as agents improving existing anemia, or treatment of gastrointestinal complaints, agents for improving sleep, means for increasing activation processes in the central nervous system, or vice versa means of inhibiting these processes in case of agitation and anxiety.

2.5. PROFESSIONAL STRESS

Stress is characterized by a set of physiological, psychological, and endocrine responses. Hans Selye explains stress by the theory of general adaptation syndrome, including obligatory non-specific response of the body as a result of any external irritation. The reaction of stress is regulated by simpatico-adrenal and hypothalamic-pituitary-suprarenal system, through complex feed-back. In an open state reaction takes place in three basic stages: 1). Alarm stage, characterized by shock and anti-shock phase; 2). Stage of resistance associated with the activation of regulatory processes, which performs the recovery of the imbalance and increases resistance of the organism: 3). Stage of exhaustion and depletion of adaptation options as a result of strong or long-acting stressors. In this connection, where it will expand the response to stress, is distinguished of two qualitatively different effects identifying such as: 1). **"Eustress"** - a condition in which there is adaptation and mobilization of defenses and 2). **"Distress"** - a condition characterized by disadvantageous reactions, exhaustion and development of pathological conditions.

Within the general concept of stress, a number of theories, etc. theories of interaction underline the importance of the mode of interaction between the person and the environment.

The model of professional stress, called **Model matching personality - environment** (French et al. 1982), perceived stress as an imbalance between work environment factors and work tasks on the one hand and individual capabilities to perform work activities. This theory rests on two basic parameters and their mismatched. The first is the objective reality (Environment - OR) and the second perception by the individual (Personal perception - P). Variables OR and P may be subjectively or objectively presented and accepted. Based on these 2 by 2 combinations can be obtained no sufficiently matching, at the result of that develop stress responses. Individual possibilities bring elements of subjective perception of the whole complex of working conditions and they become a very important factor in occupational stress.

In 1979, Karasek, developed a model of professional stress whose basis is the notion that the characteristics of labour may not be in linear relation to

the health of workers and that they can combine and interact differently. Based on a meta-analysis of results from studies of workers from the US and Sweden, found that workers with high job requirements (complicated and responsible work) and low decision-making have definitely worse health. Lowest rate of disease and death was found among groups of workers engaged in labour with moderate intension combined with a high possibility of decision-making and control of tasks and working conditions. The combined effect of these two characteristics of working conditions is called Model **"Control over decisions - Requirements of operation" (Fig. 6)**.

In 1991, Johanson et al. expand the model by adding a third dimension and formulated as **"Control over decisions - Requirements of work - Social support ".** Dimension "Social support" comes from the attitude of colleagues and managers. In exceptional mental requirements (eg greater responsibility in decision making) it serves as a buffer in the development of distress. Conversely, high demanding of the task, low control over the situation and low social support (or social exclusion) are accompanied by internal anxiety, psychosomatic complaints and high cardiovascular risk.

The interaction between the three dimensions in the model and the output of this interaction depends and on the individual characteristics. Also "impaired relaxing ability" (impaired relaxation) can model the health effects at the scheme of the model. For example. "Impaired ability relaxing" very often found in conditions of very hard work, combined with homework (less time for rest and sleep, no enough time for personal needs), leading to negative health effects.

2.5.1. WORK-RELATED STRESSORS

In conditions of employment, there are numerous stressors, which generally can be grouped as follows:

1. Stressful work environment factors.

The physical factors of work environment, along with its specific action could lead to the development and response to stress. For example, in addition to the specific hearing impairments in excessive noise levels often diagnosed sleep disorders, hypertension, headache and others. Other physical factors such as vibrations, high or low temperatures may also cause a disease-specific, but in no high intensity to lead to discomfort that can result in mental stress.

Exposure to chemicals can also lead to stress through a variety of mechanisms - direct stimulant effect on the central nervous system, operating in a high risk of poisoning and explosions, unpleasant

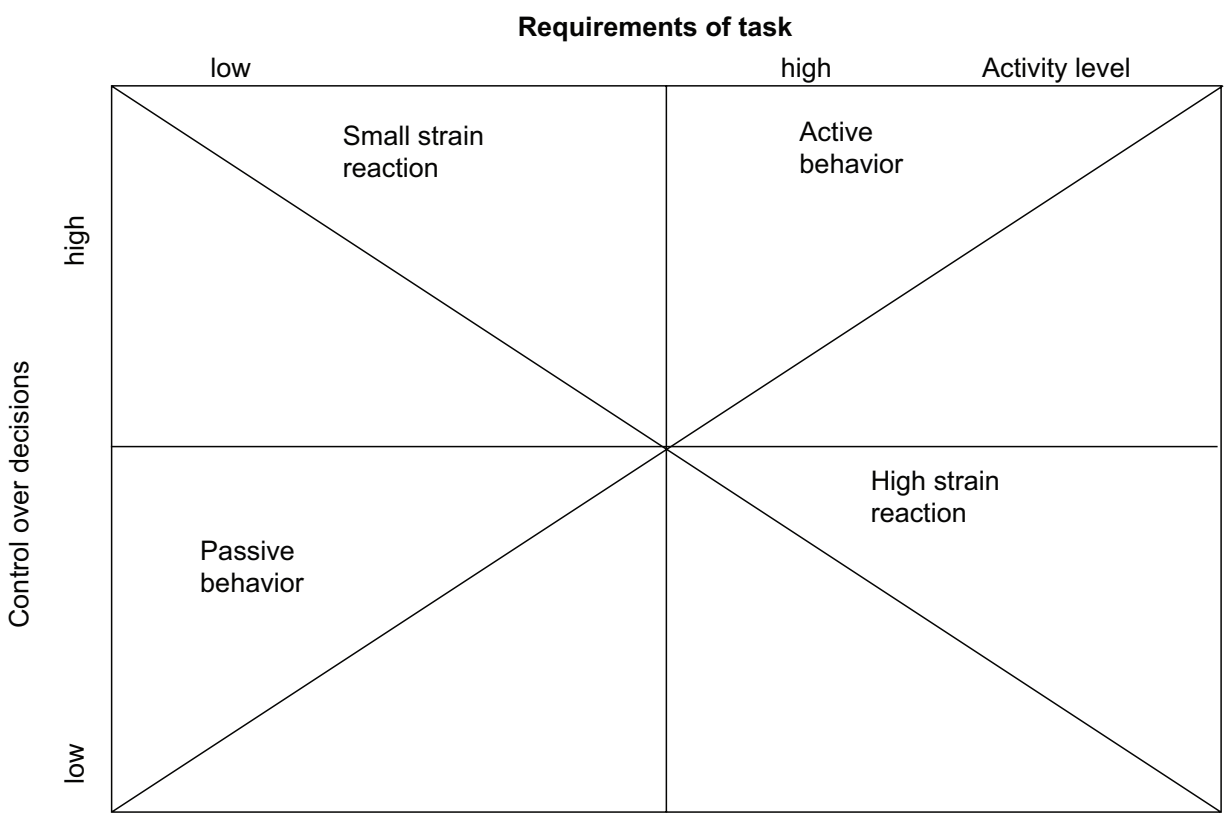

Fig. 6. Model "Control over decisions/requirement of the task" (Karasek, 1979); strain - intension

odor and others. The possibility of infections in contact with biological factors can also lead to mental tension and stress. Work environment factors can act as stressors alone or acts combined effect (potentiated or additive).

2. Organizational factors and relationships.

Currently many people in industrial and even more so in post-industrial society are engaged in organizational or managerial activities. These activities can be stressful factors as for managerial teams, and in performers. Factors such as political conflicts, authoritarian imposition of decisions, transfer of responsibilities, etc. may be strong stressors. Interpersonal and intergroup conflicts, conflicts with managers also reinforce dissatisfaction of work and lead to stress. Conversely, support from colleagues, family and especially by the heads can prevent the development of distress. Here may refer a number of stressful situations as badly defined task, not well defined responsibility, failure qualifications and skills of performers, inability to training and advancement in the hierarchy etc.

3. Labour task

Overload and under-load, the ability to make decisions, the level of responsibility are factors of particular relevance to occupational stress.

Load in both qualitative and quantitative terms is pointed out as a reason for stress-related illnesses. In quantitative overloads worker carried a lot of work, and in qualitative overloads functional requirements are high, ie tasks are very difficult. Quantitative overload often associated with cardiovascular disease, and quality - of adverse psycho-physiological changes.

Insufficient quantitative and qualitative load also adversely affect the functional state. Simple, elementary tasks and activities, especially if performed by qualified and skilled employees, leading to frustration and stress.

Responsibility at work is also a factor for stress. The highest degree of responsibility when it comes to responsibility for human life or the lives of many people - surgeons, guiding of flights, pilots, dispatchers, etc.

4. Shift work

Shift and night work is widespread stressor that is impossible to avoid in enterprises with a continuous cycle of work, transport, rescue teams and others. Health problems are the result of disturbances in circadian rhythms of the body and frequently changing dynamic stereotype. They are most pronounced in subjects with continuous sequence of night shifts and transcontinental flights.

5. Individual, group and social factors.

It is known that among workers exposed to the same stressors, stress reaction reaches different degree and distress reach a limited number of persons. Moderators of stress response are different sides of the individual characteristics, social protection and social class (group) associated with a different perception of the stress factors and possibilities to suppress stress.

Over the past 30 years, much attention was paid to the **individual vulnerability** in relation to coronary heart disease and the role of psychological and behavioural factor in the development and suppress stress. The concept of type A mode of behaviour was originally developed by Friedman and Rosenman. It defines the type A as a major risk factor for cardiovascular disease. According to a large-scale studies, people with high risk for coronary heart disease defined as type A (Friedman, Rosenman) are: strongly associated with work and dedicated to the profession; with a strong sense for urgency of tasks; with a strong sense of competition and the tendency for aggressiveness.

Indicators that assess the state of hostility and angrylity of persons, are also referred to as predictors of cardiovascular disease.

The relationship between type A behaviour or hostility and angrylity of persons on the one side and cardiovascular risk than another is moderated by several factors such as age, sex, **socio-economic and educational status**.

Group differences and the creation of vulnerable groups, reflects the effect of individual differences and social status, which are common and characteristic of certain groups and/or affect the general model of reaction in response to exposure to stressors. A number of studies identify various vulnerable groups, such as young workers, older workers, women, immigrants, incapacitated.

2.5.2. SYMPTOMS OF STRESS. OTHER STRESS-SIMILAR STATES.

Subjective complaints depend on a number of factors and conditions. First except intensity and exposure to the stress factors, to consider is how stressors is new, unexpected or expected, quick-changed, etc. Subjective symptoms depend on the state of development of stress reaction - at a sense of mild tension to complaints related to the development of distress and stress-related illnesses. **Emotional reaction** to stress is characterized by anxiety, intension, stuttering speech, impaired motor coordination, errors in performing the work. **Vegetative changes** that occur as a result of the activation of the hypothalamic-pituitary-suprarenal and simpatico-adrenal system is reflected in an increase in blood

pressure, shortness of heart rate, increase cerebral blood flow, flushing, mydriatic pupil, sweating of the hands, muscle weakness. The excretion of stress hormones can repeatedly be increased. The state of distress should be distinguished first of nervous fatigue and of some adverse psychic states.

The problems concerning the quality and duration of sleep may be also be related to occupational stress. Sleep disturbances are frequent in early rising, which reduces REM sleep and in later bedtime, which reduces the duration of slow wave sleep. Difficulty falling asleep or frequent awakenings, are accompanied by anxiety, the sense of no relaxing and can lead to neuro-psychical disorders. At stress can be seen opposite effect - sleep is extended to 9-10 hours or more as a means to overcome stress - ie. "withdrawal by sleep."

Stress-similar states. Although there are many common features between stress and **nervous fatigue**, it is considered that they are completely different states. Stress is associated primarily with vegetative changes and mental fatigue - changes in the CNS. When stress the fundamental experiences are threats, fear, uncertainty, excitement, anxiety. In the nervous fatigue person feels tiredness and weakness. Fatigue develops at the end of the working day until symptoms of stress are not directly related to the timing of the work.

Stress has some common characteristics with other adverse mental states among the most important is **burnout (burnout - burning) or syndrome of professional exhausting.** This condition develops as a result of continuous professional stress or distress and is regarded as one of the most important consequences. Characterized by:

- **Physical exhaustion.** *Going back to the model H. Seley, this is the third stage of the general adaptation syndrome, or state of distress. People complain about reduced energy, weakness, severe fatigue, which often leads to frustration.*

- **Emotional exhaustion.** *Workers in the state of burnout have a sense of helplessness and hopelessness. Satisfaction that previously had in their spare time or when are with their family or among friends disappears. Disappears and general satisfaction of life and people may fall into a deep depression.*

- **Dehumanization and cynicism.** *Negative assessment of the work has dehumanizing effect on worker and he often can not respond to the feelings of others. This negative attitude and dehumanization can be extended to family and friends. Other adverse mental states, which must be distinguished from stress and occur frequently in modern forms of work are* **mental satiation and monotony. (See section 3.5).**

2.5.3. STRESS AND ILLNESS

Impact of stress on **mental health ranges from slight changes to the express mental illness**. Subjective changes in terms of self-esteem are some of early manifestations of stress - very often symptoms are anxiety, intension, anger, irritability. These complaints are often a precursor to more serious illnesses.

A number of behavioural changes also often occur in occupational stress. The reduced participation in family and community life, apathy or unreasonably aggressive behaviour towards others are common symptoms. Individuals in high-stress state and dissatisfaction with work often resort to excessive use of alcohol and high consumption of cigarettes. Other behavioural changes relate to variations in appetite - from very large to the complete disappearance of desire for eating; assessment of risk situations that most often develops a tendency to take unreasonable risks; decreased interest in recreational activity.

The next level changes are prominent psychological dysfunctions. These include prominent clinical depression, anxiety disorders, psychosomatic disorders (hypochondria, for example), and exacerbation of pre-existing illnesses. Established a statistically significant link between these illnesses and stressors such as job loss, lack of opportunity to improve the social situation and others.

So-called post traumatic stress disorders is a consequence of significant traumatic episode in working conditions affected colleague. The patient consistently "experiencing" the traumatic event and developed symptoms of reduced ability to respond adequately to the outside world. Fear anxiety, depression, avoid actions that cause memory of the tragic incident, emotional lability are often accompanying symptoms.

Mass psychogenic illness (mass psychosis) is a collective manifestation of symptoms that affect two or more persons sometimes covering the entire team. Symptoms are always subjective such as headache, dizziness, vertigo, heaviness, fatigue or weakness. There is a small or absent objective symptoms. The disease is more common among women's groups in offices of various kinds and spread by auditory or visual chain. Factors and conditions such as boredom, monotonous and unsatisfying work, mismanagement, isolation and poor communication are often in this disease.

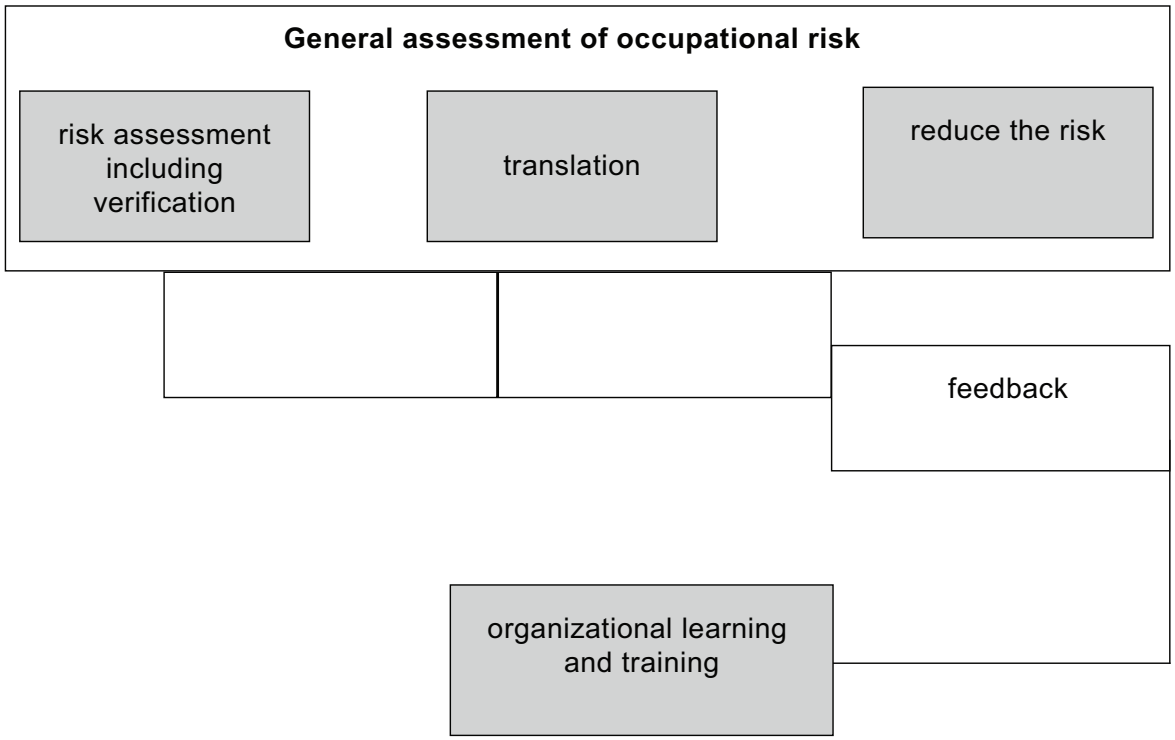

Fig. 7. A structural model for managing work-related risks (in Cox, T. et al., Work related stress, 2000).

Stress control programs	Stress-reducing programs
Autogenous training Biofeedback Deep breaths Physical exercise Free activity Massages Meditation Progressive relaxation Social support Yogism	Defending workers' rights Resolving conflicts Ability to make decisions and resolve conflicts Interpersonal skills Work planning and time management Psychotherapy Psychotherapeutic means

Tab.3. Programs for prevention and stress management

Professional stress is a risk factor for **cardiovascular disease**. It is known that atherosclerosis, coronary artery disease and myocardial infarction, insult, in large part, the result of such risk factors like dyslipidemia, hypertension, smoking, unhealthy lifestyle. Stress contributes to heart disease in two ways - either individually or through potentiation of other risk factors. Very high workload associated with increased incidence of myocardial infarction. When workers engaged in mental work establishes a close link between job satisfaction, conflict and coronary disease. In strong stress response was an increase in serum cholesterol. And increases the excretion of stress hormones - adrenaline, noradrenaline and cortisol, androgens, thyroid function, increases and blood pressure.

Diseases of the digestive system, especially ulcer disease long associated with stress. Epidemiological data show an increased incidence of gastric and duodenal ulcer among executive directors, air traffic controllers, dispatchers and other professions with high responsibility. The relationship between stress and peptic ulcer can be explained in that the autonomic nervous system has essential role for stomach secretion. Stomach secretion also increases and as a result of the catecholamine excretion.

Other gastroinestinalni problems, such as diseases related to appetite (bulimia and anorexia), gastritis, ulcerative colitis, and enterocolitis, functional biliary dyskinesia, endocrine diseases (eg. thyrotoxicosis) and immune systems, also often associated with stress.

2.5.4. PREVENTION AND STRESS MANAGEMENT

Prevention and management of occupational stress is one of the biggest challenges of modern industrial and post-industrial society. This stems from the fact that more and more growing stresogenic

professions and activities, and the difficulties associated with the disclosure of the stress factors and diagnosis manifestations of distress. Available models (Cox T. et al; 2000), which emphasizes that stage "reduction stress" ("risk reduction") includes not only prevention of the stress factors, but also activities aimed at health and well-being of the individual **(Fig. 7).**

Prevention of stress begins with disclosure and assessment of the stress factors, passes through the understanding by the leaders of the need to implement measures for their removal and understanding of the worker by developing its own strategy, behaviour and lifestyles aimed at overcoming stress. The doctor is obliged to seek targeted and reveals the earliest symptoms of distress, to establish as far as possible the causes and to offer workers means and methods to reduce stress. In the prevention and management of occupational stress using two basic approaches - individual and organizational.

Most of stress managing programs are aimed at the individual and aim to develop in him the knowledge and skills of management and reduction of stress. These programs can be offered to all employees or to a particular group or individual. Skills can be classified as managing stress and stress reducing techniques **(Table. 3)**.

Managing techniques are directed to methods to help individuals cope with the psychological and physiological effects of stress. Stress reduction program developed interpersonal skills and strategies that aim to reduce the stressful nature of work-related psychosocial environment and the stress factors arising from specific labour organization and those arising from factors in the work environment and employment. In the prevention of stress in some cases can lead to proposals and developing health and hygiene regulations, industry regulations or initiating legislative activity.

Zhel. Hristov, B.Tsenova

2.6 MODERN APPROACHES AND METHODS TO ASSESS THE STATE OF MENTAL STRESS AND INTERVENTION

Modern approaches and methods to assess the state of mental stress and stress intervention continues to be defined in different ways and at different levels of analysis - and as a stimulus and as response, and as recapitulate the interaction and the imbalance between the individual and the environment.

2.6.1. MODERN PSYCHOLOGICAL THEORIES OF OCCUPATIONAL STRESS

Transactional theories and models. These theories about stress are based on the conceptual structure of the *interactive* models, with a focus on the structural characterization of the interaction between the individual and the surrounding work environment. It, however, puts more emphasis on the *psychological mechanisms* underlying this interaction - emotional reactions, cognitive assessment and reassessment, and stress management (coping)

– Model imbalance "effort-reward" (Siegrist J. et al., 1990). According to this model the survival of chronic stress can better be explained in terms of the ratio of high cost - low gain. Conditioned by labour stress is a consequence of the discrepancy between the magnitude of the *effort*, with sources of external demands and internal motivation and the amount of the consideration *received*, either monetary, socio-emotional payoff or position control, related to career prospects and security at work. Adverse health outcomes such as cardiovascular risk prevalent in professions where situational constraints do not allow workers to reduce the imbalance between effort and reward.

– Cognitive - phenomenological transaction models for assessing and coping (Lazarus R.S. et al., 1981), according to which stress is a negative *mental state* with cognitive and emotional sides. The *primary assessment* involves constant checking interactions personality-environment in terms of requirements, possibilities, capabilities, limitations and support (the question is: "Do I have a problem?"). Establishing a problematic situation is usually accompanied by unpleasant experiences and a general feeling of discomfort. The *secondary assessment* after the establishment of the problem involves a thorough analysis to determine the *coping strategies* ("What do I do?"). Stress occurs when a person, considers that it can not cope with the requirements set him or something that threatens his well-being, ie stress with unpleasant state of intension that occurs when a situation is assessed as hostile, deflecting of habitual and a threat to self. The arguments in favor of the *negative definition* of stress are numerous. Only negatively rated events can be expected harmful consequences, and only when the intensity and duration exceeds a critical limit. Typical of stress that is **associated with the individual's psyche**, as decisive is the *assessment* of the individual, not only its physiological reactions, movements or behaviour (although for it is often judged on the basis and of these effects).

Stressors are events that increase the probability of stress conditions. Potential stressors are different loads of work: from labour task, of job role, of the physical and social environment, of the conditions of behaviour - isolated jobs or "overpopulation, of professional (non) availability, inefficient style of action, lack of experience. These effects cause **stress - response**, including uncontrolled and uncontrollable physiological, psychological and behavioural changes. It can be short or long with negative consequences for both work performance and health.

– Burnout is an individual experience, specifically in the context of work. The worker is no longer able to engage in normal work. Exhaustion causes of action for withdrawal (emotionally and mentally) of their work as a way to deal with overload.

Burnout is determined with questionnaires, the most widely used of which is the Maslach Burnout Inventory (MBI, 1981), including 22 questions grouped into subscales: "Emotional exhaustion" (EE) - emotional overload and exhaustion of their work; "Dehumanization" (DH) - indifference and cynicism regarding dependent of professional people; "Employability" (E) - a subjective sense of competence and efficiency in their work.

Contemporary theoretical framework of burnout syndrome clearly integrates individual and situational factors based on the model "work-person" with a focus on the correlation between worker and six key areas of work environment: workload, control, reward, community, justice and conflict between valuables. These six areas of life in an organization are frame preceding the development of

burnout organizational characteristics. The greater is the *chronic* disparity, the greater the likelihood of burnout. Understanding the specifics of burnout syndrome requires distinguishing it from similar and related states such as frustration from work, stress, depression, poor mental health, mental violence, absorption of the work **(Table. 1)**.

2.6.2. PRACTICE FOR RESEARCH ON WORKPLACE STRESS

As indicators of long-term effects of stress on health most often studied markedness of psychosomatic complaints and depressive tendencies, cardiovascular diseases (CVD) and digestive system, back pain (low back pain).

Numerous recent studies clearly show that high *demands* and intension of the job are the main risk factor for CVD. As etiological links, *psychosocial and behavioural* configurations (responsibility, dedication to work, chronic fatigue) play a role, as well as conventional risk factors (hypertension, smoking) and changes in autonomous and neuroendocrine mechanisms. High demands of work associated with a 7-times increased risk of emotional exhaustion and low social support at work - 2 times higher risk of problems and pain in the back and shoulders.

Type of work and freedom of decision-making are important for the formation of the risk of cardiovascular, gastrointestinal, skin, allergic and other diseases. There are sufficient evidence that the major risk factors for these diseases are psychosocial and their action is limited to: ● Depletion of adaptation mechanisms; ● An imbalance in terms of "Requirements-Control" in the workplace ● Imbalance "Efforts-reward."

The presence of *psychosomatic complaints* is an important indicator of overall prosperity. In an analysis of registered complaints should be taken into account personal circumstances and factors related to motivation as they changed causal relationships. Complaints include various neurological and vege-

Table 1. Burnout and related conditions - similarity and distinction

Related conditions		Burnout
Depression	● all areas of life ● emotional, physical and behavioral symptoms, weakness ● predisposed to depression (with high scores in neuroticism scale) are more vulnerable to develop burnout	● related to the specific situation of work ● decrease in efficiency due to negative attitudes ● emphasis on the emotional and behavioral symptoms rather than physical ● under normal people without previous psychopathology
Impaired mental health	● in all professions and people ● linked not only to work ● more pronounced pathological profile	● clinically healthy people ● form of poor mental health, closely linked to the work performed, "work-related neurasthenia" (ICD 10)
Mental violence at work	● all jobs ● extreme psychosocial *stressor* ● with victims and affected organizations similar state as in burnout	● in social professions, emotionally demanding work with people ● consequence of unresolved chronic stress
Stress	● in all professions ● prerequisite for burnout	● social professions, emotionally loading ● result of chronic no overcome stress
Connectivity with work	● *positive* emotional and motivational state of satisfaction and fulfillment, *positive* antithesis of burnout ● high activation and pleasure associated more with the *resources* of the work (job control, feedback, learning opportunities) ● main components: - Energy, without fatigue put effort, stabity - Devoted - dedication, involvement, enthusiasm, pride - Pleasant absorption - the operating time passes imperceptibly	● *negative* wealth with *low levels* of independent dimensions - activation and pleasure ● more connected with the demands of work – load, emotional demands ● main components: - exhaustion - cynicism and alienation - reduced efficiency
Dissatisfaction with work (DFW)	● in all professions ● extent that work is a means and source of satisfying the needs and exemption from creating displeasure factors ● does not include **personal** connection with the work itself (content and meaning)	● social professions ● connected to itself performed work ● closely related to DFW, but causation has not yet been clarified ● contagious and self-powered through informal interactions at work

tative symptoms and disorders, leading to depression, isolation, alienation.

As an indicator of the general health being used individual number of symptomatic response and the comparison of different subgroups and the percentage of persons with an increased number of complaints. The distribution of responses to individual questions, as well as their configuration depending on individual criteria for forming subgroups to compare, sex, occupation, experience, etc. - is a basis for comparison.

Special attention should be paid *depressive* tendencies, because although the psychological price of such problems is mainly at the expense of the individual and his family, society pays an enormous price in the form of loss of work time, inefficient interpersonal contacts and their multiple organizational implications and other mutualities effects.

According to the hypothetical model for risk factors and pathogenic mechanisms for the development of diseases hypotheses about the emergence of the CVD, diseases of the digestive and the musculoskeletal system should integrate clinical methods with labour - social and psychological causes and physiological mechanisms. Synthetic model of stress, various events and consequences that cause in their dynamics is presented in **Figure 1**.

In analyzing the etiopathogenetic relationship between experiences stress and disease, must always be in mind that the relationship is *probabilistic* rather than strictly deterministic. Professional work is only one of the possible areas of life, which can cause stress and illness respectively. Much of the stressors associated with family, unfulfilled expectations for tomorrow, insecurity, poor ratio expectations-realistic reality, leading to so-called "stress of wear." It is essential the influence of family history of a disease.

Appropriate is division of investigated stressors according to their origin to several factorial areas:

- Factors of **socio-economic** environment - economic development and stability, standard of living, unemployment, crime and aggression, structural reforms, ethnic problems, etc .;
- Factors of **family environment** - financial problems, unemployed in the family, difficulty in caring for parents and children, health problems, relationship and support between family members and others.
- Factors related to the **profession** as complexity, responsibility, qualifications, workload and rhythm and more.
- Factors of working environment, including:
- **Physical, chemical and biological** factors;
- **Psychological factors** - social support from management, opportunities for communication and expression;
- Factors of **work organization** - working hours, variable schedule and mode of work and rest, staff shortages, emergencies.
- For determination using validated questionnaires and special questionnaires containing different numbers of questions with possible answers.

Each **stressor** is assessed regarding 1) the existence and 2) by a factor of weighted average potency. The potency is determined in degrees awarded by the respective grading value. For each type and group of stressors determine the types and group factor potency (0.1-1 odds). The ratios of potency can be calculated and depending on the individual and personal characteristics of the respondents - experience, gender, age, position, stress stability, resources for coping.

Depending on **availability**, duration and transience of the **stress state**, the respondents refer to:

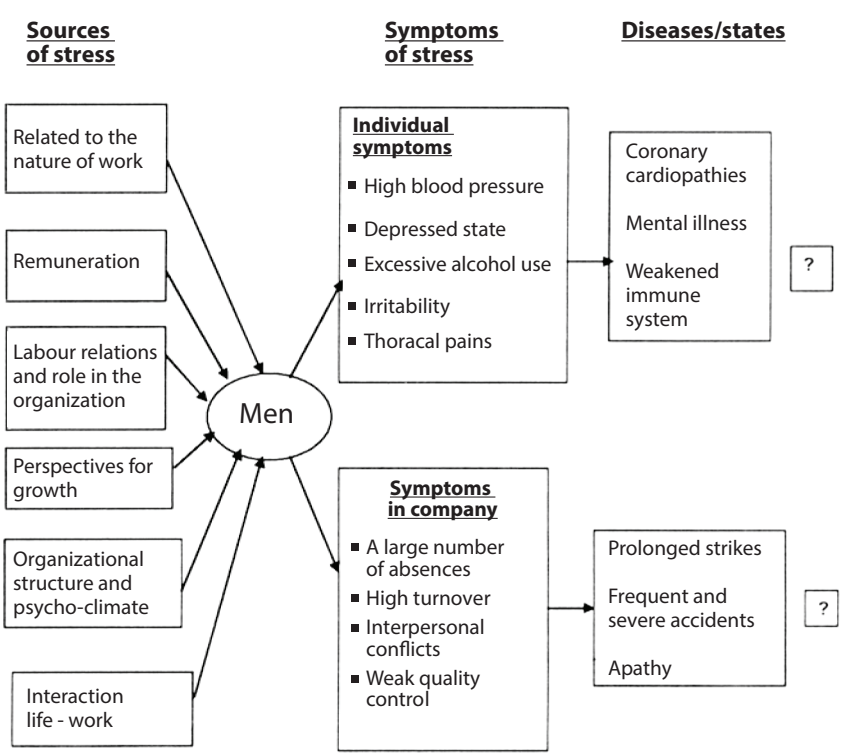

Fig. 1. Model Cooper for dynamics of the labour stress (adapted from Cooper & Marshall, 1976)

- The state of constant stress - provided the "permanent" presence of not less than two or more symptoms
- State of intermediate stress - not less three symptoms, referred to "often" with a break
- Fast transitional state of stress - "rarely" (1-2 times) indicates the presence of symptoms, regardless of their number.
- It is important data for these factors to be as *specific* as possible and be focused on the **performance** of the work (absenteeism, delays, decrease efficiency, omissions, causeless accidents), or **abnormal repetitive behaviours** (alienation, inappropriate humor, superficiality, big mood swings, more and more careless appearance, alcohol, etc.) for a longer period of time.
- The strength of the existing **relationship** between the listed diseases and studied stressors is estimated by calculating the **odds ratio** (OR) of the studied factors or relative risk (RR) by method of single- and multi logistic regression.

2.6.3. STUDIES IN BULGARIA

Studies of stress in teachers, doctors, social workers show that in our country they are at increased risk of stress-related diseases. Changes in the basic principles, rules and norms of the device in society, related to socio-economic transition create new stressful situation that causes serious changes in the psycho-emotional state of people and their health. This situation is characterized by: 1) Increase the sources of stressful impacts, 2) Diversity in the structure of factors and the appearance of new, 3) Increase the intensity and duration of action of different stressors, 4) Reduce the stress stability of the population.

Stressors and state of stress for doctors and teachers. With a starting hypothesis that the profession and the work of doctors and teachers in conditions of socio-economic transition are subjected to numerous and with greater intensity stressful factors, leading to the occurrence of psycho-emotional reactions that adversely affect their health has made epidemiological characteristics.

A comparison of different groups stressors shows that psychological factors generally have highly stressful effect (second only to socio-economic factors in both professions). Foremost among them is "bad balance in the estimates of official capacity and the work done", i.e. non-conformity personality - environment, followed by "lack of support and cooperation from the leaders."

The ratios of the potency of socio-economic factors are significantly higher connected with profession and reflect the attitude towards public events (economic development, crime and violence, reforms in education, health, etc.) from a professional point of view. Highest stressful action has low living standard.

Profession and gender of individuals influences the perception of various factors and perceived strength of their action - in teachers is a higher relative part of highly effected factors in comparison with doctors.

They studied two main forms of stress - affective and depressive. Affective symptoms of stress were observed in 98% of investigated, and by high levels of this form of stress amounted to 31% doctors and 37% for teachers.

Depressive symptoms are common to more than one-fifth of investigated, and by the high level of these are 8 to 14% in different professions - doctors, respectively teachers.

Nearly a quarter of doctors and teachers are in a state of *constant* stress and *fast transitional* stress is distributed to a third of them. When teachers, registered a greater frequency and greater part of the constant and often presence of stress-related psychosomatic symptoms. Women in both profesions have a greater number of psychosomatic events compared to men.

Digestive system diseases and stress. The study of the probability of occurrence of such diseases due to various stressors at doctors and teachers shows that the highest prevalence among cases are the stress factors:

- Poor sanitary conditions;
- Unemployed in the household;
- Work associated with monotony, non-ergonomic requirements, forced posture;
- Eating habits-, number of meals throughout the day, malnutrition, opportunities for supplying quality food.

Much of stressors in which the risk of diseases of the digestive system increases statistically significantly associated with family and social environment, the influence of family history, the problems in the health and educational systems. They increase the risk of developing these diseases from 1.4 to 3.5 times.

They are identified and specific, increasing the risk occupational factors - for doctors - work and contact with specific contingent and contagious patients, in teachers - uniformity and monotony of work and poor sanitation.

Burnout. Research on the prevalence of burnout in recent years have shown that early and middle

stages of burnout are widespread among teachers, medical and other support staff. Different profile and markedness of the dimensions of burnout in professional groups connected with the specific conditions and load of work. Analysing the correlation of burnout symptoms and indicators for common intensity and frequency of load and social support by the head, displayed on the base on of established stressors indicates that:

– the total amount of stress, frequency and load, determine the development of emotional exhaustion;

– the frequency of experienced of work stressors, especially the lack of support from the head, associated with the development of indifference, cynical attitudes dehumanization. Dehumanization reliably correlated with burnout indicators for the situation of working, which in turn have associated with marital status;

- the sense of reduced efficiency and performance is related to the intensity of the load;

- the greater the frequency and experienced significance of individual stressors, as of the stronger is expressed burnout symptoms, emotional distress and dehumanization.

2.6.4. STRATEGIES AND POLICIES TO LIMIT STRESS - MODERN PRACTICES

2.6.4.1. MONITORING OF STRESS

Main reasons why you should be paying special attention to work-related stress, to monitor and supervise the extent and spreading him are:

– stress is a problem both for the *user* and the *enterprise*. Stress-related disorders and diseases lead to decreased productivity, absenteeism, disability, poor psychological climate;

– related employment dependent stress *problems are growing* - the nature of work is changing in recent years due to increased requirements for mental labour, technological changes, flexibility of production and services, constant reorganizations, leading to a growing need for retraining and increasing insecurity;

– *legal obligations.* Framework Directive of the European Community for health and safety, requires an employer to recognize and correct the existing stress factors. It stipulates that the employer is obliged to ensure the safety and health of workers in relation to any aspect related to work, following general principles of prevention - risk avoidance, risk assessment (which can not be avoided), anti-source of risk, adaptation of work to the individual, particularly in terms of workplace design, equipment selection and choice of work methods and technologies, with a view, in particular, to alleviate monotonous work and work with a given rate and reduce their unfavorable health effects;

– Harmful stress can be *avoided*.

Monitoring of stress requires the development of sufficiently reliable, informative and efficient methods for identifying and evaluating its, applicable to various professional communities.

2.6.4.2. TYPES AND LEVELS OF INTERVENTION IN COMPANIES AND ORGANIZATIONS

Any organization that seeks to establish and maintain the best state of physical, mental and social health of employees, needs a rate policies and procedures that comprehensively cover health and safety. This company policy includes procedures for stress management based on the needs of the company and its collaborators. There are many choices that need to be taken into account in the prevention of stress - primary level of prevention (eg. Reducing stressors), secondary (eg. Stress Management) and tertiary (eg. Programs to help contributors-consultation) and are aimed at different phases of the stress process.

Primary prevention deals with the organization of activities to modify or eliminate sources of stress present at the work environment. Possible strategies are:

- *re-design* of task;
- *re-design* of the work environment;
- establishing *flexible work* arrangements (schedules/time-table);
- stimulating the management with broad *participation of all* employees;
- *inclusion* of employees in the planning of professional development;
- analysis of the roles of work and setting *goals*;
- providing *social* support and feedback;
- building cohesive *teams*;
- promote *fair conditions* and relationships at work - a policy of the employer;
- *distribution of salaries* and incentives.

The organization's activities in the primary intervention include changes in communication, meetings and discussion processes, *changes in the methods and style of work organization*, changes in corporate culture.

Any intervention should be guided by a previous diagnosis or test of identification specific workplace stressors, interviews and questionnaires to detect stressors associated with fatigue and burnout.

Secondary prevention is mainly engaged in the *discovery* and *management* of experienced stress by

raise *awareness and improve skills* for stress management of the *individual through* training and education. Key factors that influence individual susceptibility to stress are: individual personality, individual coping strategies, age, gender, attitudes, training, past experience, level of social support from family, friends, colleagues.

Secondary prevention can focus on learning different *relaxation techniques*. Activities in health promotion and programs to change *lifestyles* relate to primary prevention. *Education on the problem of stress and learning* for stress management help individuals: to recognize symptoms of stress; build skills to change their lifestyle or work (eg. training for management and distribution of time or self-affirmation); acquisition of resources to cope, as with both inherent in the working environment stressors, which can not be removed (e. uncertainty of operation), as well as other in extraprofessional sphere.

The role of *secondary prevention* is inherently primarily *limit the damage* (*often* directed more at the consequences, rather than the sources of stress). The main objective is to improve the adaptation of the individual to stress.

Tertiary prevention is concerned with the care, *rehabilitation* and *recovery process of those individuals* who have suffered or are suffering from serious health damage due to stress. Interventions include specialized psychological counseling on the issues of employee in professional and personal spheres. They are effective in helping workers to cope with stressors in the workplace, which can not be changed, and with stressors outside the professional sphere, which spread and affect activity in the profession. Such services and consultations are strictly protected, they are subject to medical confidentiality.

Psychological consultation (therapy) is possibly effective in terms of improving mental well-being of workers, and in terms of economic impact. There are data showing significant economizing in such investment - on the order of 3: 1 to 15: 1.

2.6.4.3. FIGHTING BURNOUT SYNDROME

Most effective approach is a combination of changes in management practices and training. Interventions in the leadership needed to change in any of the six areas of working life, but not sufficient, as long as no changed the consumed individual skills and attitudes.

Common strategies for preventing or reducing already occurred burnout vary according to the *level* and type of four *goals* of the intervention:

- *individual level:* helping individuals to operate more effectively with occupational stress;
- *interpersonal:* colleagues are mutually reinforcing and help to cope with stress, creative supervision, submitting feedback, listening, group training, communication training;
- *workplace:* changes in the workplace to eliminate or reduce stressors.
- *organizational level:* long-term political and structural changes in/on whole organizations or institutions, rules and regulations.

Activities at each level can have different general objectives:

- *identification* of the existence of burnout, causes and its consequences at every level;
- *preventing* on any level;
- *intervention:* reduce burnout or reversed development if the process is started;
- *crisis intervention:* therapy for persons, who are in an advanced stage of burnout.

Specific measures to deal with stress loads aim to:

- implement active strategies to overcome stress, such as changing the situation itself or its individual aspects, personally or together with other colleagues;
- expand own capabilities and competence to respond more adequately to the stress;
- providing social support where together with other colleagues seek a solution and understanding of experienced professional difficulties.

Main in preventing burnout is the attention and care to preserve the health of staff, awareness of the existence of the problem, definition of responsibilities in prevention of burnout, improving cooperation and mutual support and the support of the head.

2.6.4.4. PREVENTION OF MENTAL VIOLENCE (MOBBING)

Depending on the phase of escalation of the conflict and the reasons apply different measures: changes in the design of labour, changes in the behaviour of the head, protect the social position of the individual and raising the level of ethics and standards in working group and the company as a whole.

An appropriate strategy for the prevention of workplace violence involves four steps:

- policy for prevention of workplace violence - supported by management, with annual training and complete (if necessary);
- realization of the first weak manifestations and potentially violent behaviour in the workplace;

- intervention demonstrating the importance of the problem, encourage early notification of potential violence, which is a better outcome in a timely care;
- networking - an interdisciplinary team analyze signals of violence, which contributes to maximum benefit from interventions in low potential risk.

Justify its effectiveness in cases of psychological harassment in the workplace are the promotion of knowledge on the problem mobbing through periodicals and scientific-popular publications, radio and television broadcasts.

2.6.4.5. PROACTIVE APPROACH - AN OVERALL POLICY TO LIMIT AND REDUCE STRESS

There is a change to the old style, where experts react to already arising problem of management, providing for the possibility of problems and finding solutions through a proactive approach. According to EU directive 1996 y. for consideration of stress as a process, for analyzing and managing risk and protecting workers, recommended **cycle of action,** running through several phases: identifying injuries, assessment of risk, implement appropriate control strategies, monitoring of their effectiveness, reassessment of risk, analysis for needs of information and education at risk.

Perspective is spreading throughout the world since 2002 SOLVE methodology of ILO for management of psychosocial questions in the workplace and related health problems through *integration into overall company policy*.

The International Labour Organisation, the World Health Organisation or by joint working groups and specialized agencies and commissions of the European Union, develop programs to prevent the harmful consequences of the development of mental stress and workplace violence. In our country this base model is built on prevention and limitation of stress with the strategic goal of reducing morbidity and mortality from diseases associated with stress and improve health in a variety of professional groups in the country **(Fig. 2)**.

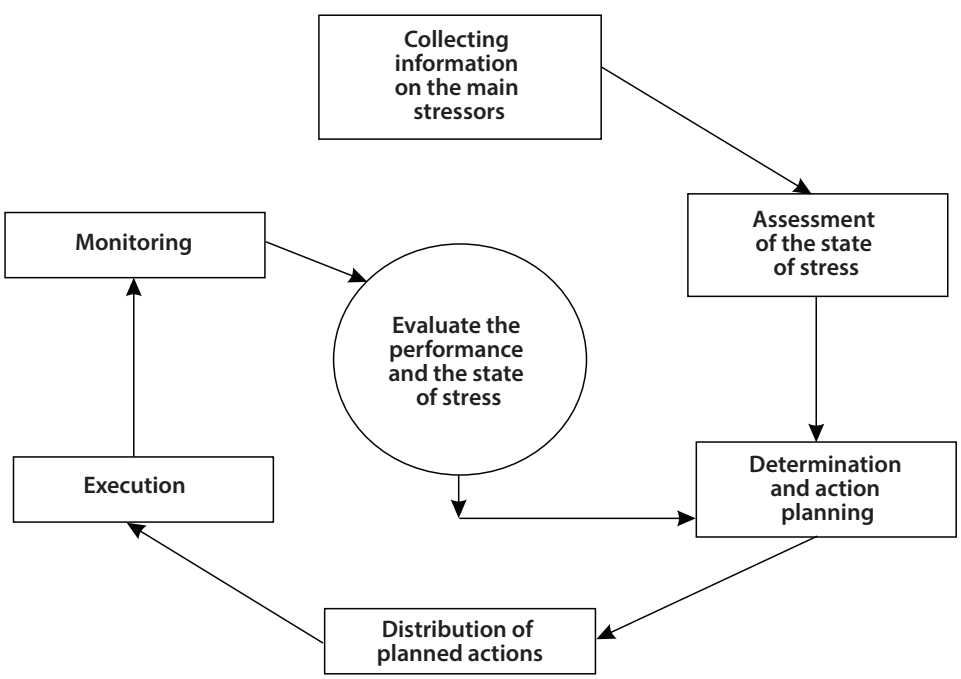

Fig. 2. The algorithm of model to limit stress (Zhel. Hristov, 2004)

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3 PSYCHOLOGY OF LABOUR

Psychology of labour study the experiences and attitudes of people in the labour process, psychological conformities, mental processes and properties of the individual in their relationship with the subject and work tools, with physical and social environment, i.e. mental regulated employment of individuals with regard to its conditions and consequences. It covers these achievements and methods of the various psychological disciplines, that are relevant to the analysis and optimal design of employment.

The main objectives of the psychology of labour at applied research are the humanization of labour and increasing its productivity. Under the humanization of labour is of course prevention of fatigue, occupational diseases, prevention of industrial accidents and professional deformation of the person, raising the thoughts of labour, creating conditions for multilateral personality development and deployment of its abilities.

To solve these tasks using a variety of measures: technical, technological, sanitary, organizational. Psychology solves tasks for humanization of labour with its own means taking into account the psychological characteristics of employees. All these events come into the structure of scientific organization of labour.

General psychology of labour study basic properties of psychic structure and regulation of work involved in solving any single task in any type of work activity (Hacker, 1998). It deals with particular processes of aim-formation and motivation, the creation of an indicative basis, the design of the programs of action, of deciding on the modalities of implementation and controlling it using a relatively stable operating forms, resulting in the execution of specific activities. Its subject are the process of activation and psychic automation that alter mental regulation of work. Special psychology of labour takes into account the characteristics of different types of work and prepare the psychological foundation for solving several specific tasks that can be attributed to the following areas:

1) Psychological design of activities, i.e. improving *working conditions*, covering design and evaluation of regulating psychic structure of work in shaping the articles, especially the means of labour, decision-making on the organization of work and also for general working conditions such as work environment, regime of work and rest.

2) Improve the *individual conditions and opportunities* for successful work, covering the opening and development of the psychological foundations of professional training, including diagnostics of individual prerequisites for achievement as a basis for/or a result of the training.

3) A separate group tasks of the psychology of labour associated with *the development and evaluation of methods* for labour psychological research. These are the methods of assessment and analysis of the activity, for diagnosis and prediction capabilities, methods for studying social structure and psychological climate in the team, methods for assessing work-related mental conditions.

3.1. STAGES OF DEVELOPMENT BY PSYCHOLOGY OF LABOUR

Psychology of labour is a relatively young science whose formation began in the late nineteenth century. The main stages of development of work-psychological theories differ in the definition of man's place in the production process - "economic", "social", "own-developing" and "complex" human, understanding for the organization and the organizational structures, the purpose of changing labour activities and the criteria for their evaluation. The main directions of its development - **teylorism, psychological aptitude, the movement of human relations, business, industrial and engineering psychology** - are associated with the development of economic life, industry and public relations. Based on the study of individual elements of the movement in the late nineteenth century american engineer **F. Taylor** developed a system for rationalization of movement and attempted to justify scientific organization of labour. The recommended its system of strict professional selection boils down to choosing the fast-

est and best candidates on the basis of work tests to maximum performance, regardless of the needs and interests of workers and the consequences for their health.

During the first three decades of the XX century especially became known **psycho-technique**. Its occurrence is associated with the names of famous german psychologists-psychotechniques William Stern, introduced the concept in 1903 and Hugo Myunsterberg worked mainly in the US. Psycho-technique, understood as practical use of psychology in different areas of life and culture, brings to the fore issues of professional selection and orientation, studying individual differences in professional ability and demand for appropriate methods for the detection capabilities (with orientation mainly to test method).

The study of the process for implementation of work and determinants for achievement is linked to the development of **experimental psychology and psychophysiology**. Current problems are fatigue and efficiency and strive to fully cover the employment process. Differentiated by its specific tasks **industrial or production psychology**, which studies the forms, methods and organization of work, selection of personnel in industrial production, the psychological issues of furniture and shaping the work environment. Since this area is a separate **business psychology** with typical tasks - socio-psychological and economic-psychological issues of market, supply and demand, the relationship between people and production. In the 60s it was formed **engineering psychology** dealing with the complicated interrelationships between man and machine in modern terms of technical progress and automated production. It gives psychological foundations of programming, design and management of automated systems. It is also known under the name of "human engineering" (Human Factors engineering). The need for coordination of a number of biological, technical, economic, social and psychological parameters in the optimization of labour in the system "man-machine" (SHM) led to the development of **ergonomics**.

3.2. CRITERIA FOR PSYCHOLOGICAL ASSESSMENT OF WORK ACTIVITIES

In determining the purpose of changing labour activities essential issue is the system of criteria by

Tab. 1. Hierarchical system for psychological assessment of work activities (Hacker, 1998)

Level of assessment	Degrees	Screening methods	Possible criteria (examples)
4. Encouraging personal development	♦ development of selected **A**bilities and **A**ttitudes ♦ preservation of certain **A.** and **A.** ♦ reduction of certain **A.** and **A.**	⇩ TBS* on Hacker (1989) ⇩ Satisfaction of labor with control of covarianted values also possible: ⇩ Labour subjective analysis (SAA Udris & Alioth, 1980) ⇩ Diagnostic examination of the activity (JDS, Hackman & Oldham, 1974)	• time for independent action • time for creative activities • necessity of learning
3. Preservation of violations of the mental condition	♦ no or addmissible violations ♦ tolerable conditionally eligible violations ♦ malfunctions	⇩ Screening of mental intension with cards for CEAL ** ⇩ Scales to assess subjective health complaints also possible: ⇩ Scale to assess own status (Nitsch, 1976) ⇩ AET *** (Rohmert et al., 1978) ⇩ Comprehensive analysis of the workplace and mental stress ⇩ Ergonomic assessment by Schmidtke (1976)	• negative changes in psychophysiological indicators (ECG, EEG, catecholamines) • disorders of mental condition, deterioration of self-confidence • dynamics of achievement
2. Safety	Damage to health ♦ excluded ♦ possible ♦ they are very probable	⇩ Brief methods for assessing working conditions and workload, represented a health risk from CEAL ** complemented by ⇩ Analysis of morbidity and separation of covarianted variables ⇩ Psychoneurologic questionnaire for toxic effects ⇩ General analysis of labor ⇩ Ergonomic assessment of labor	• values of threshold and a maximal concentration of harmful substances • occupational diseases • accidents
1. Implementation	♦ unlimited ♦ partially restricted ♦ reliable implementation no exists	⇩ General ergonomic analysis of work (AET *** by Schmidtke)	• anthropometric standards • psychophysical parameters of CNS and perception - labor - hygienic standards • psychophysiological limit values for processing of information

* TBS (Tätigkeitsbewertungssystem) - evaluation system for activity
** CEAL - complex ergonomic analysis of labor
*** AET - Arbeitswissenschaftliches Erhebungsverfahren zur Tätigkeitsanalyse Rohmert, Landau, 1979) - scientific research method for analysis of work

which they are measured. One of the most commonly used systems today to assess the level of psychological shape (design) of labour activities developed by Hacker W. It contains four hierarchical levels of evaluation **(Table. 1)**.

Hierarchy means that: 1) the implementation of the minimum requirements of the first level is a prerequisite for the transition to the next higher level of assessment of work and 2) higher levels provide possibility for further and fuller realization of lower. Eg. the possibility of individual work style and use of intellectual abilities may prevent the onset of fatigue, to prevent violations of the mental condition.

Assessment in terms of **safety** is primarily occupational health and hygiene problem with (or not) damage to health.

Fluctuations and deficiencies in the **implementation** of the action may be general information about the existence of problems with its realization. Identification of such problems is possible by the analysis of the performance of the particular task. Restrictions on the performance of an activity can be determined by:

1) the design of tools and working environment that does not meet the anthropometric and physiological properties of man.

2) cognitive feasibility of actions, ie overload capacity for processing information of the person or difficulty mastering of leading for activity signals due to lack of feedback or unclear purpose.

In the assessment of work in terms of **preservation of violations of mental health** problems associated with load, tested primarily negative effects of stress. They depend primarily on the informational characteristics of activity and occur as reversible deterioration of mental preconditions for achievement, for ex. fatigue and similar conditions such monotony, satiation, etc. The emergence of negative consequences of stress involves a change in the level of claims (personal goals for a specified quantity and quality of performance change), or by change of put in efforts. During prolonged and intensive load compensation by raising efforts it becomes impossible and leads to distortion in the structure of activity and dysregulation. In psychomotor regulated activities occur violations of coordination, increase is additional and hidden breaks and interruptions. In mainly perceptual regulated activities, eg. tasks of monitoring, the effects of load are pass signals and prolonged reaction. In intellectual regulated as processing information, fatigue worsened short-term and operative memory, changing and defining them motivation. For the assessment of the potential negative consequences of load, associated with a particular activity, it is necessary to follow its implementation and changes in personality over the course of one shift. Essential in this assessment are changes in the term structure of the work shift, the organization and the combination of different activities. Also important is the assessment of the physical and mental work correlation, which has no standards. It is known that the purpose of the design work process is balanced participation of physical and mental work, for reasons of health and optimum degree of activation.

Under the *potential of employment* to ***boost development of personality*** understand the opportunities for further development of the usual activity regulating psychological conditions: attitudes and abilities.

The main conditions to encourage personal development in employment, which are evaluated in the analysis of activity are:

- Providing sufficient activity.
- Providing opportunities for application and storage of the acquired premises for work.

The concept of *overall employment activities by* Hacker is based on the consideration of this problems. The characteristics of the overall labour activities are: ◆ self *goal setting*, ◆ independent *training* of the actions taking functions of planning, ◆ independent *choice of the means of action* and the necessary interactions to achieve the goals, ◆ perform activities by providing *feedback* its passing, ◆ *control*, incl. possibility to verify the compliance of the results of their actions for posed goals. In *partial* activities absent any of these characteristics, mainly the possibility of self goal setting and decision-making or for individual courses of action. Integrity of work activities has two sides: *cyclic integrity* associated with the consistent performance of all business functions and *hierarchical integrity* associated with the use of various levels of psychic regulation of the activity. Optimal structures are covering preparatory, performing and controlling phase with changing of different levels of psychic regulation - from sensmotor to the level of solving intellectual problems in the activity.

- Provide opportunities for application and expansion of the variety of conditions for activities by learning.
- Provide self goal setting, decision making and selection methods.
- Provide creative potential opportunities to change working methods.
- Provide opportunities for collaboration with others.
- Provide assessment of the significant achievements of the performers.

Three components - complete structure of actions, learning opportunity and partly jointly per-

formed work, provided the activity of people at work, which in turn is a prerequisite for adequate activity and success.

Measurable effects on the developing potential of the work is established in two areas: a) maintaining and developing intellectual abilities through continuous employability with higher cognitive demands **(Fig. 1)** and b) motivation for own work task and work for public purposes and problems.

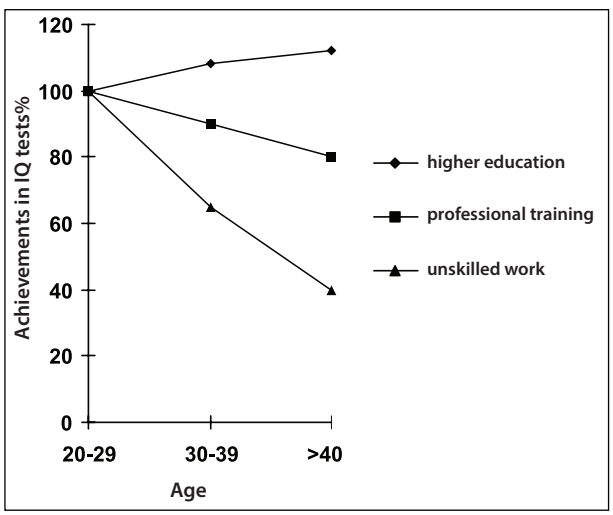


Fig. 1. Age changes in the performance of the IQ tests depending on the qualification level of activity performed *

3.3. METHODS FOR PSYCHOLOGICAL ANALYSIS OF THE CONTENT OF THE WORK AND ITS IMPACT ON HUMANS

Most commonly used methods of analyzing the perception of objective employment situation and analysis of the impact of work on the general state and self- confidence. In psychological analysis of activity uniting different methods, allowing reliable and valid conclusions in one way amending various parameters set by them.

Numerous areas of application of the results of labour - psychological studies, that range from the design of the work system to professional selection and professional guidance, explain the variety of methods used.

1) Psychological analysis of tasks assigned, the conditions of implementation and operation in order to detect their determining employment factors. These methods are analytical procedures that are modified depending on the class of studied business and target (rationalization and design of business or screening for specific industry). "Objective" method with a similar goal is method for detecting regulatory *requirements* in employment (MURI, VERA of Volpert et al., 1983), ie the requirements in terms of planning, thinking and decision making. "Subjective" method with such goal is the method by Ulich (1981). Another example is a system to evaluate the performance TBS by Hacker (1989), which is a differentiated system of sorting and separating meaningful for assessment of work *characteristics*: organizational and technical conditions that determine the completeness of the activity; cooperation and communication; arising from job responsibility; required cognitive performance; requirements for qualification and training. Another method of psychological analysis of the *labour process* in a workplace is a methodology for analyzing workloads as a result of regulatory obstacles (AWRO, RHIA of Leitner et al. 1987).

2) Expert assessment of the task and activity. For this purpose there are standardized universally applicable methods, regardless of the activity, of which the most widespread is the questionnaire for the analysis of labour McCormick et al. (PAQ, Position Analysis Questionnaire, 1969). They are more suitable job classification and definition of requirements for employability than for the justification of the proposals for activities related to the design of the activity and qualification requirements. PAQ questionnaire consists of 6 modules: receiving information, information processing, work performance, significant work for connections to other individuals, environmental influences and additional conditions.

3) Appraisal in attitude of global dimensions. This category methods analyze the perception of objective work situation in order to explain and forecast the impact of events on the work design. The most widely known and distributed is a questionnaire for diagnostic examination of the activities by Hackman & Oldham (Job Diagnostic Survey - JDS, 1974). It revealed scores of workers objective characteristics of their work, and emotional reactions to the work and its conditions, and readiness for positive motivation to work **(Table. 2)**.

According to the authors the emergence of internal motivation for work is related to the experience of "critical psychological states," which are defined by the five dimensions of activity. Intrinsic motivation is determined by human needs of personal development. People with high need for personal development react more positively to the changes related to increasing the degree of markedness of the five characteristics of the activity, ie better quality performance, higher satisfaction of employment by reducing absenteeism, higher intrinsic motivation.

* Amthauer test for structure of intelligence IST (Schleicher, 1973). Special studies have shown that by the same starting level of education, differences in mental performance increase with age and length of service, depending on the cognitive demands of the activity.

Tab. 2. The design and structure of the questionnaire for diagnostic study activity by Hackman & Oldham

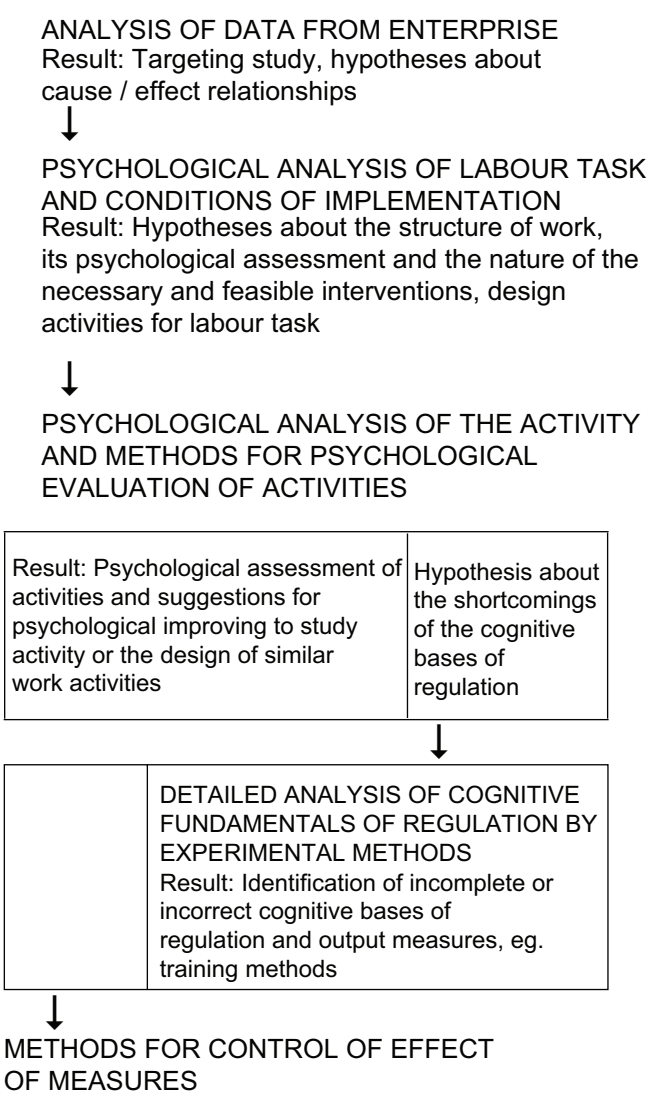
Dimensions of activity	Psychological states	Effects for work
• A variety of activities • Integrity task • Importance of the task of one's own life and for others • Autonomy (independence) • Feedback on performance inherent in business	♦ Experienced importance of work ♦ Experienced responsibility for performance ♦ Knowledge of current performance, the quality of own work ♦ Need for personal development	* High motivation to work * High quality employment achievement * High satisfaction with labor * Less absenteeism and low fluidity of labour

Widely known is the method of subjective analysis of the activity of Ulich, E. (STA, 1981) to stimulate the motivation of workers, where they are regarded as "experts" in relation of their activities.

4) Estimation of work related to required professional qualities. In this type of expertise differ two approaches: with an emphasis on the necessary for implementation of the action properties and capabilities, and focusing on the required cognitive processes. For a description of activities by data on cognitive processes Miller (1971) compile a list of terms such as detection, search, identification, coding, interpretation, categorization, transmission. Another group of methods described business with the necessary behaviours, like Job Activities Checklist of Palmer, McCormick (1961).

5) Analysis of the effect of work on the self confidence of workers. Labour situation works by reflecting it and processing it, in relation with the experience, goals and own claims. As confirmed by the results of research into stress, the process is three stage - perception, assessment, action. The processes of perception and evaluation are available by revealing experience, consisting of: motivation; various aspects of job satisfaction and its conditions; use and raising of qualification; different dimensions of intension; difficulties in implementation of labour. For their study used various questionnaires. Some of the most commonly used are the scale Bartenwerfer (1969), screening of intension by Meister (1976), scale of states by Nitsch (1976), a questionnaire for determining the intension, monotony and satiation by Plath, Richter (BMS, 1978), polar profile by Mathesius (1972).

Fig. 2. Stages of psychological analysis of labour.



3.4. METHODOLOGICAL APPROACHES TO PSYCHOLOGICAL ANALYSIS OF LABOUR

Psychological analysis of work is carried out successively in stages **(Fig. 2)**:

1) Preliminary analysis of the documentation of enterprise in terms of interpersonal differences and dynamics of individual annual performance, qualifications required for various types of activities in jobs, error analysis, an analysis of the inworking of newcomers, morbidity, fluidity of labour and accidents. These data allow quick orientation and are necessary for the preparation of labour psychological analysis and activities, for two reasons: a) The implementation of anthropometric, labour-hygienic and medical requirements is a prerequisite for employment-psychological designing of activity. This means that an activity must be feasible and does

not lead to disabilities and to optimize in relation of the consequences of mental load and opportunities for personal development. b) The effectiveness of the work depends not only on external factors (distribution of functions, means and labour conditions) but also by personal characteristics. Cards for complex ergonomic analysis of the workplace, allow for a differentiated assessment of the state of labour hygiene and many other characteristics of work. Furthermore, there are specific methods of analysis and evaluation of occupational hygiene and anthropometric characteristics of the workplace (ergonomic assessment of the labour system by Schmidtke, 1976, and the system AET by Rohmert et al., 1975, that reveal and evaluate all factors associated with feasibility and safety of work process).

2) Psychological analysis of work activity and conditions of employment on the basis of technological documentation, description of operating systems and facilities, interviews with experts from the enterprise and individual workers, observations in the workplace and, if necessary, - monitoring the passing of the activity. The analysis of the working activity comprises the following steps:

a). technological division of the manufacturing process and separation of humans tasks.

b). identifying the work process, within the production process: for the entire enterprise.

c). description the qualities of the product to be treated or the process that must be managed with reporting capabilities for impact by workers;

d). analysis of the distribution of labour between workers and labour conditioned cooperation and communication, based on documentation, interviewing the immediate managers and workers;

e). description of the rough structure of individual work activity (in relation of stability, and complexity); determining the sequence of individual operations, identifying the key points for decision-making and operations in different work situations, determine the number of signals and managing impacts, of listing rules and criteria that must be respected;

f). establishing objective possibilities for choice between different ways of working. Choices that have worker (= degrees of freedom), refer to the sequence of individual operations, goals and objectives, means and ways to achieve them.

g). Characteristics of the individual activity in relation of the time factor - duration, frequency (repeatability) during a working day/shift. Similar objective activity can lead to various activity (redefinition of activity) depending on the subjective perception of the individual and the qualities of the individual.

3) Psychological analysis of the activities and methods of its psychological assessment.

Structure of employment is characterized by a) the content of labour, expressed in the needs, motivations and targets and determined by the means and conditions of work and b) the hierarchical elements of activity - actions, operations and movements. The *action* is an essential element of the work and is characterized by relative autonomy.

The psychological analysis of activity is always going according to the particular job. Labour activities, and actions for their designing and improving, assessed according to how well they execute the requirements in relation of their enforceability and safety, eligibility of load and conditions for progress of personality (Hacker, 1998). Depending on this, is defined, as subject of study variables and parameters, and also methods for their determination.

4) Planning of psychological analysis of work.

To remove possible sources of error in collecting necessary to evaluate the performance data, needed to ensure their representation in several respects:

a) representation on work assignments for the workplace (hampered at varying tasks, eg. the production of small series).

b) representation in relation of time conditions in the workplace, which may change depending on the mode of shift work.

c) representation in relation of the groups of workers (level of qualification, experience, operating mode).

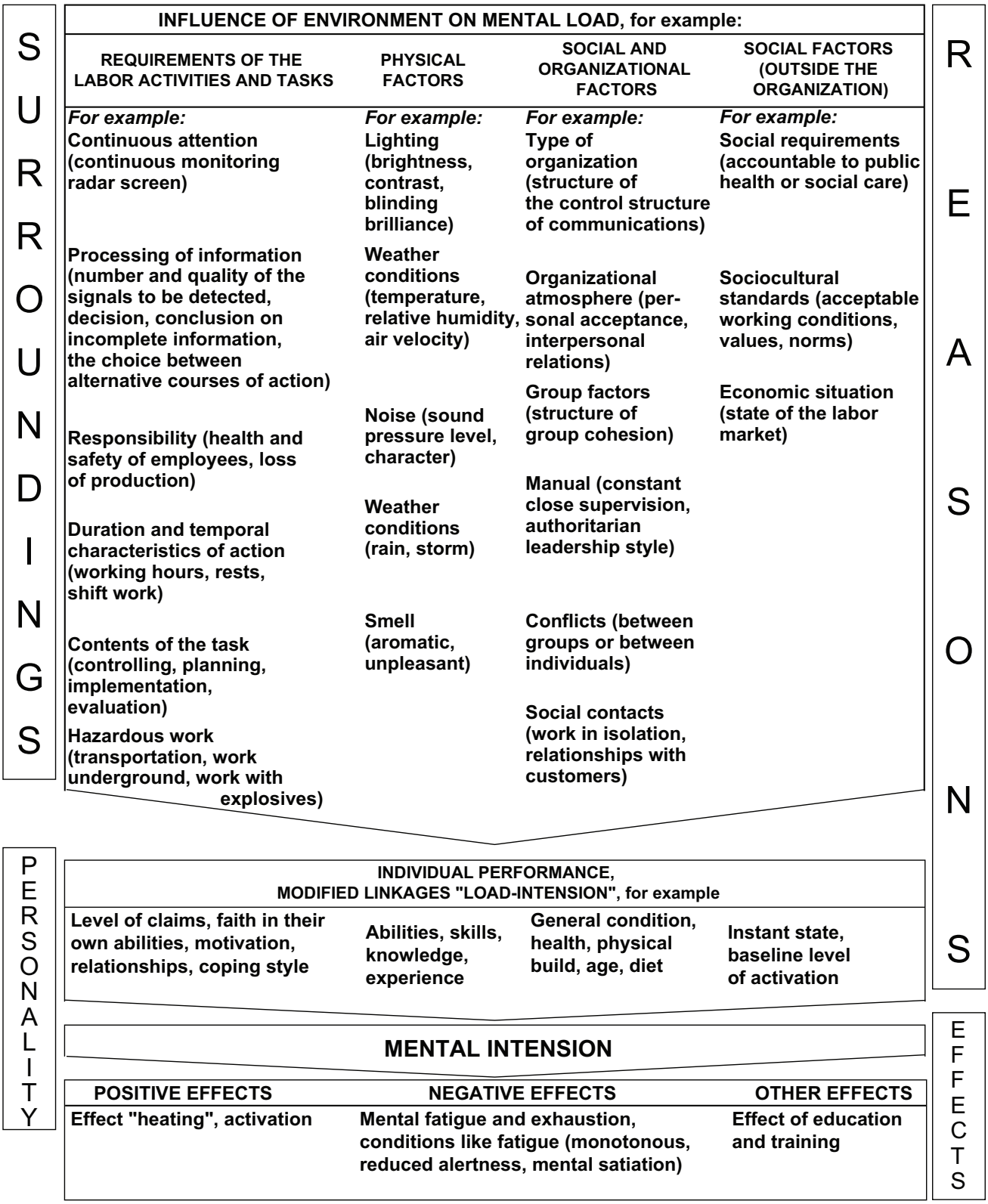
In all studies of human behaviour must be taken into account effects associated with conscious or unconscious influence of behaviour. Errors of this kind due to the "conscious participation in the experiment" called *Houtorn effect*. The possibilities for reducing such reactive effects are few:

- awareness lectures and providing assistance to workers for their participation in the experiment,
- testing methods and approaches in pilot studies,
- opportunity to control and verify the stability of the routine individual ways of work.

3.5. PSYCHOLOGICAL STATES AT WORK

Mental state of man is relatively stable structural organization of all components of the psyche and serves as an active human interactions with the external environment presented at a time of particular situations. "Psychic" in this sense refers to the emotional and cognitive processes in humans associated with the acceptance and processing of information. The characteristics of a mental state caused by the same stimulus may be different depending on the individual characteristics of personality and its assessment in relation to the stimulus.

Fig. 3. Scheme of the "mental load - mental intension"



Emerging in the labour process mental states of people can be classified (staged) on several counts: duration, a leading component, level the overall tone, degree of active awareness, dominant in the structure properties of personality, etc. In any psychological state there is a certain level of activity of an organism which can be detected by change of psychophysiological parameters.

Most important for the psychology of labour classification of states by level of intension, as this feature is most significant in relation of their impact on business performance. Moderate intension is normal operating condition, occurring in mobilizing impact on employment. It is necessary for the

normal implementation of the action and is accompanied by moderate modification of physiological reactions manifested in good spirits, stable and moderate implementation of the action. Increased intension accompanies the activities taking place in extreme conditions.

1. Psychological states and working conditions

Each activity, even primarily physical work can lead to mental stress. According to the European standard (EN ISO 10075-1, 2000) *mental stress* is a term to describe the set of all external influences on people who influence him mentally and can be evaluated. Mental load causes an increase or decrease in mental intension in humans. Depending on your previous activity or baseline and the qualities of the individual, the same situations can constitute mental load leading to increased or decreased mental intension.

Mental intension is a direct nolong-lasting effect of psychological load on people. The consequences of mental intension can vary in shape and strength depending on the individual characteristics and situation. Adverse factors that increase the intension, refer to the following groups: physiological discomfort or non-compliance of the conditions of living and regulatory requirements; biological fear and danger to life; lack of time; increased difficulty of tasks; increased significance of potential improper actions; presence of significant side factors; failure due to objective circumstances; deficit of information for decision making; sensory deprivation; information overload; conflict conditions and others. The degree of mental intension, which is carried out, depends also on motivation and assessment of the objective. The high assessment of the purpose of the action, requires more energy and effort to implement its, mobilization.

The links between mental intension, individual characteristics, experienced mental intensiont and its immediate effects are shown in **Fig. 3.**

The immediate effects of mental intension are positive or negative. *The positive effects* of mental stress are:

Effect of "heat" - a common consequence of mental intension that soon after the commencement of operations reduces the effort to carry it out.

Activation - inner state with varying degrees of mental and physical functional capacity. There are areas where activation is optimal and provides the best functional capacity. The sudden increase of intension can lead to undesired hyperactivation and worsening of the results.

The negative effects of mental stress should be differentiated depending on the time needed for recovery and ways to achieve this. These include:

Mental fatigue - a temporary disorder of the effectiveness of mental and physical functional capacity, depending on the strength, duration and course of the prior mental intension. Reduced functional capacity occurs eg. in feelings of fatigue, a more unfavorable ratio between achievement and put in effort, type and frequency of errors, etc. The components of tiredness as a mental state are:

- Feeling weak, oppressive intension and uncertainty;
- Attention deficit disorder characterized by instability, low mobility or chaotic;
- Disorders in the sensory area of receptors involved in such work as ex. "smearing" of the text after prolonged reading or weakening of tactile sensitivity after prolonged manual work;
- Disorders in the fields of motility, resulting in delay or unorganized movements, rhythm disorder, reducing accuracy, coordination and automation of movements;
- Defects in thinking and memory in the field which is related to work, lowering their wits;
- Loss of will, determination, persistence and self-control;
- Drowsiness as an expression of the development of protective inhibition.

Tagged psychological indicators of fatigue occur depending on its extent. Low fatigue (first stage), without significant changes in the psyche, signaled the need for measures to avoid a decrease in performance. The second stage of the fatigue is characterized by a marked and enhancing deterioration of productivity, often only in relation of quality. The third stage is characterized by an acute experience of fatigue in the form of strain at which sharply decreases the efficiency.

***Similar fatigue states* - monotony, reduced vigilance and mental satiation.**

- *Monotony* is slowly evolving state of reduced activity, which may occur during prolonged, uniform, repetitive tasks or activities and which is mainly characterized by drowsiness, fatigue, decrease and fluctuations in productivity, a reduction in the adaptability and reactivity.
- *Reduced vigilance* is slowly developing condition characterized by reduced signal detection in uniform tasks of monitoring (eg. monitoring of screens or indicator devices).

To overcome the negative impact of boring and monotonous work is very important motivation.

Mental satiation is a state of nervous, periodically Increasing, strongly emotional rejection of repetitive task or situation that is experienced as "not get-

ting anywhere" or "leading to a dead end." Additional symptoms of mental satiation are anger, reduced performance and / or sensations of fatigue and a tendency to withdraw and business interruption. Mental satiation in contrast to the monotony and reduced vigilance is characterized by unchanged or even increased level of activity, but associated with negative emotions. It is caused primarily by repeating the operation, while monotony are needed and other objective data - narrow of irritants, uniformity, narrowness of the observed field.

As an indirect consequence of mental satiation may occur effects of training related to repeated overcome the mental strain.

Other effects are boredom, feelings of overload where there is great interpersonal differences. The same applies for possible *adverse long-term effects* of frequent, repeated very high or very low mental stress: psychovegetative disorders, increased irritability, depression, psychosomatic complaints and diseases, permanent disability, *stress*, burnout "burnout" (Tsenova B., 1993). According to modern concepts of *stress* he was uncomfortable state of intension arising from the assessment of a given situation as unfavorable for the individual and the resulting need to remove this imbalance individual- environment in which he can not cope. Richter & Hacker (1998) propose a system of nine criteria to distinguish most researched forms of negative effects of mental stress **(Table. 3)**.

The state of negative emotional intension that occurs in humans at the meeting of a substantial barrier on the way to achieving a significant goal for him is described as **frustration**. It is usually accom-

Table 3. Criteria for distinguishing the different forms of the negative effects of mental stress (Richter & Hacker 1998)

Criterion	Negative effects of mental intension			
	Mental fatigue	**Monotonous**	**Mental satiation**	**Stress**
Configuration **triggers**	Prolonged demands on the prerequisites for achievement (PA); accelerated when exhausted and overloaded PA	Insufficient use of PA as a necessity in narrow range of observation	Experienced a lack of sense in readiness for realization of the task	Objectively overload without the possibility of avoiding and/or by experiencing overload congestion of caused negative emotions; frustration of goals
Leading signs of experience, phenomenal	Exhaustion, tiredness without boredom	disinterest, boredom, accompanied by a sense of fatigue	marked irritability and discontent, disgust	excitatory troubling intension, anxiety, concern the feasibility of the task
Time to business	Occurs after a continuous activity; constant process	occurs after a continuous activity; undulating course	it is also possible in advance and during the operation	it is also possible in advance and during the operation
Change activities - effect	small	improving achievement	?	probably permanently lower achievement
Depending **on assessments**	small	small	high	high
Activation	Initially compensatory increased, later downgraded	downgraded	increased	increased
Individual **dispositions**	Missing abilities and skills strengthen mental fatigue	Missing PA and a limited variety of tasks favourable for usual hypoactivation	none	increased vulnerability to emotional lability
Restore passing	Constantly, requires time	possible intermittent	?	persistent violations of recovery
Precautions	Design of labor, distribution of functions man-technique, training regulatory PA, regime of work and rest	enrichment of tasks, reducing the requirements for continuous attention	individual sense, pricing, goals, changing activity	reduce emotional stress by expanding the field of activity, treatment of emotional lability

panied by negative emotions - fear, anger, insecurity and the formation of a negative attitude towards the cause. In frustration behaviour manifests as aggression, marked hostility, rudeness, malevolence or directed at himself aggressiveness, or on the contrary - depression and lower activity.

Human behaviour in extreme conditions of activity is an expression and a result of his *psychological readiness for activity*. The total and situational psychological readiness includes the following components:

a) motivational (need for achievement, interest in the operation); **b)** cognitive (understanding the obligations, task, assessing its relevance, knowledge of the means to achieve the objective); **c)** emotional (sense of responsibility, enthusiasm, confidence in success); **d)** volitional (self-control, focus on the task, overcoming doubts).

Rationalization of working conditions in the broadest sense is a means to combat the negative mental states.

Some examples of solutions and approaches to prevent the negative effects of mental stress at work at different levels are: distribution of breaks for rest; avoiding monotonous noise; shift work; periodic replacement of the operations carried out; combination of tasks of varying complexity; distribution of tasks; avoid fragmentation of the operating time and continuous attention; variety of tasks; identification of signals; changes in the modality of signal; lighting; temperature; coloring; the possibility of individual approach in carrying out tasks etc.

2. Methods for assessment of mental states

To evaluate the effectiveness of rationalization in certain working conditions using different methods to study the states. In many cases, only the combination of more methods may allow diagnosis of mental state. To determine its use monitoring of human behaviour, natural and laboratory experiment. Monitoring results can be supplemented with the objective data as: timing, photographs, camerawork, measuring some physiological functions (heart rate, breathing, blood pressure, sweating, skin-galvanic reaction, etc.). This approach is called a methodology for visual assessment of the situation in external signs and somatic indices.

Mental state of fatigue is a complex phenomenon which depends not so much on the severity of labour but also by a number of subjective conditions: increased excitability, asthenic emotions, lack of planning. **Used today methods can be divided into two groups: methods oriented to physiology and to psychology. In physiological methods investigate** the physiology of the muscles (electro-myography), central nervous system (EEG), autonomic nervous system (pulse rate measurement, skin-galvanic reaction, reflex of the pupil). **To psychological methods belong** tests measuring mental functions in humans, volume, distribution and switching of attention: the tests on Kraepelin-Schulze, Nechaev, Schulte-Platonov, Bernstein, tachyometrical survey and others. Only using various methods, including subjective report can give an accurate description of the state of fatigue.

3.6. PSYCHOLOGICAL STUDY OF OCCUPATIONS

1) Professiography

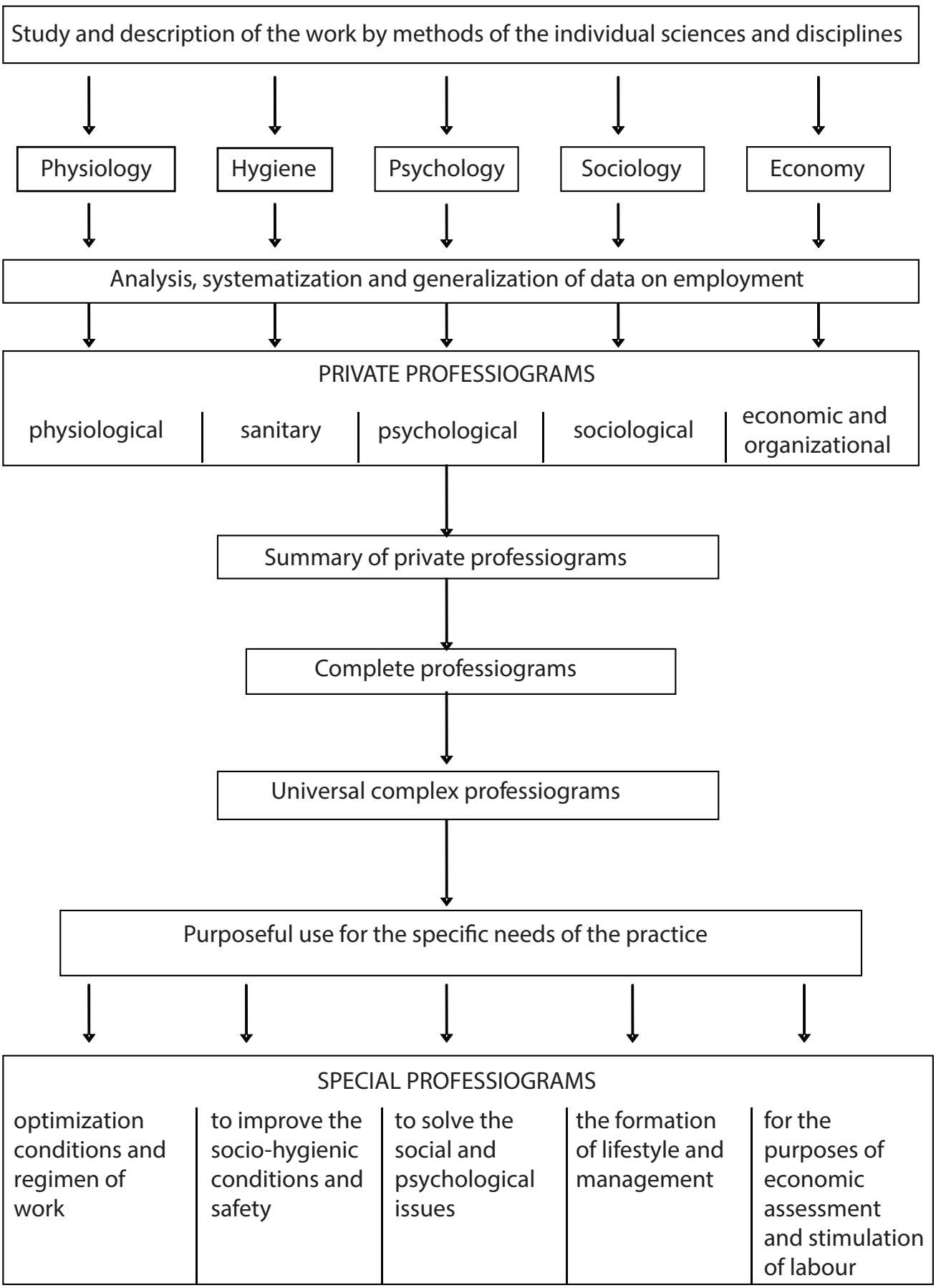
For many professions are developed professiograms - description of socio-economic, industrial and technical, hygiene, psychological and other characteristics of the profession. An important part of professiograms is psychogram - feature the requirements of the profession to the human psyche. Development professiograms and psychograms first descriptive stage of psychological study of activity.

Professiography *principles* are: complexity, purposefulness; principle of social activity, suggesting inclusion in professiograms elements of profpropaganda; principle of the personal approach; principle of reliability (concerning stability of a person under unfavorable conditions); principle of differentiation, providing different disciplines entering a profession and principle of typification requiring aggregation of occupations in certain groups; and finally the principles of perspectivity and reality.

In accordance with the first of these principles, the complex analysis of the profession suggests: - production characteristics of the profession and its specialties; - data on the economic importance of the profession; - sociological and socio-psychological characteristics (incl. features of interpersonal relationships, the collective, the social prestige of the profession); - listing of knowledge and skills required for successful professional activity with the release of those defining professional mastery, involving and terms of training and perspectives for career development; - hygienic characteristics of working conditions with the release of so-called "occupational hazards"; - review of requirements of human, point of view of physiology and medical contraindications to exercise a profession; - Compiling psychogram.

The sequence of the research in the use of profesiographycal method is presented in **Fig. 4.** Depending on his appointment professiograms are divided into universal, containing information and specifications apply to all types of work, and special containing specialized features needed to solve specific labour tasks.

Fig. 4. Continuity of research of work using professiographycal method



The content and volume of psychogram depend on the purpose for which studied the profession. Such an objective can be professional selection and orientation, occupational training, rationalization of the regime and working conditions, optimization of labour, increased safety, standardization and evaluation the hardness of labour, etc.

2) Methods of psychological study of the professions

On the psychological study of professions methods are used to gather information about the peculiarities of the professional environment and the resulting demands on people, properties of the subject of labour and its demands on the business environment, as well as methods for processing and systematization of information received.

Analysis of the documentation.

Of technological documentation, manuals, instructions, periodicals can obtain initial information about the structure of the workplace and the circumstances of: basic professional tasks, operations, performance for mode of work. Knowing these characteristics of the business and professional terminology is a prerequisite for the development of monitoring plan and talk scheme with specialists.

Talk and survey methods.

These are aids, but can be used at the provisional stage of familiarization with the profession or the study of refractory for monitoring and experimental verification parts of professional activity. The study can be in the form of verbal discourse or using the inquiry forms.

The survey - receipt of written answers to questions - can have a "free response" and type "selected response".

The reliability of the information received in the lecture and the survey depends on the belief of respondents, that reported their data will not be used to the detriment of themselves and their colleagues.

Method of monitoring.

Monitoring work-process and behaviour of specialists (operating with tools, materials, communication) enables a more accurate description of the actions and movements of workers in accordance with production tasks and the results of labour and enable the detection of critical moments.

Monitoring is usually combined with timing. Timing and photography of the working day is convenient to use specially prepared blanks with vertical scale of time on the left side. The main elements of the activity coded in advance. The monitoring data is usually complemented with objective data obtained with the help of measuring methods.

Introspection and own account

In the case of own account workers are instructed *to think aloud* on the job, to explain to the viewer as a student all necessary actions and operations.

In the second option of using introspection in professiography himself psychologist became a student and studied the profession by participating directly in the work and gradually acquire the required skill and habits. Such an approach called *working method* is applicable only for relatively simple jobs, whose adopt does not require much time. In other professions, this approach can be applied in the form of labour tests when adopt only some, the most important or accessible areas of activity.

Experimental method

In professiography experiment serves to verify the already constructed psychogram. When enough match (high correlation coefficients) between experimental evaluations and practical performance data of professiographical analysis can be recognized as correct and justified.

The main experimental productions in the study and analysis of the occupations are:

- finding experiment aimed at ascertaining whether the respondents have certain qualities (sens-motorial, intellectual, personality),
- production experiment, which involves the introduction of changes in the organization and execution of work in aim to studying their influence and also role of individual psychological factors to cope with the requirements of professional activities,
- modeling experiment in which models are made, reproducing complex conditions and labour operations by analogy with the real.

We should'nt, instead careful preliminary analysis of the profession, at the outset, to try an experimental evaluation of by chanse taked mental properties, associated with a profession, because they can produce false conclusions.

Personality methods

The properties and relationships of personality can be assessed from the examined person as in most personality questionnaires, or colleagues who know him well - a method of independent characteristics. Widespread personality questionnaires are of Eysenck questionnaire to determine neurotism, extroversion-introversion and psychotism; sixteen factorial questionnaire Cattell (16 PFI); MMPI (Minnesota multifactor personality questionnaire). Expression of a factor in a person is determined by 10-15 questions whose answers in implicit form characterize these factors (properties).

Using these questionnaires assessing personality, suggests their adaptation (taking into account socio-cultural differences) and standards for the

population. It should be taken into account that obtained using these questionnaires "diagnosis of personality" is *unsustainable* and change significantly by environment and lifestyle of the individual.

In the *method of independent characteristics by* Platonov a trait of personality evaluated by the immediate supervisor and workers who know better research specialist (of 5-7 people) on a 5-point scale. The received average assessment show formed in the working collective opinion about a person.

3.7. PROFESSIONAL SUCCESS AND MOTIVATION TO WORK

1) Definition and criteria for professional success

Concept of "success" is not unambiguous. It includes primarily labour productivity but also product quality, making mistakes, the necessary neuro-psychical efforts to achieve a result (its "cost"), and employee satisfaction.

When the degree of markedness of any individual-mental property corresponds to the high level of professional success, this property can be regarded as professionally significant, ie ability. Abilities can be as individual properties (peculiarities of his cognitive, emotional, volitional activity) and the relationship of personality (interest, passion, responsibility, debt). A prerequisite for success in a profession not just a collection of the necessary properties and a certain structure - "functional group" capabilities (combination of various components). The level of the capability development depends on the relationship between the possibilities of man and his motivation and interest in the profession.

Individual sustainable system of psychological means, personal approaches and methods of work that one uses consciously or spontaneously to adapt to objective conditions and requirements, called individual style of activity.

Strong motivation and the existence of a conflict between the properties of the individual and the relationship of personality capabilities to structure can be removed through the development of under-expressed significant professional property.

The second option to overcome this contradiction is expressed compensation of insufficient property or set of properties with other habits and skills. In real terms it is difficult to determine whether the basis of professional success are certain abilities or new forming experts skills.

This contradiction can not always be overcome even partially. In this case, the outcome is the formation of positive attitudes towards other activities. Consultant for professional orientation can greatly contribute to the creation of proper motivation and peaceful reorientation.

2) Motivation to work

There are many studies and attempts to classify individual needs that motivate people. A. Maslow releases 5 basic levels of human needs: 1) basic physiological needs, ensuring survival (hunger, thirst, sleep, etc.). 2) need for safety and security; 3) need for love, friendship and social activity; 4) need for respect, recognition and self-esteem; 5) need for self-fulfillment, achievement and personal growth. Only after satisfying the needs of lower-level, person strives to meet the needs of a higher level. Maslow believes that the need for realization of their opportunities, continuous self-improvement and creativity in a broad sense, dominate the little people.

The motivation for work is influenced by how work contributes to addressing the needs of workers. Obviously, unmotivated behaviour is harmful to an organization, such individuals are absent often, do not comply with working time, distinguished by neglect, cynicism, apathy, lack of interest, sometimes aggressive attitudes and reactions to his colleagues. They themselves are in a state of stress and provoke the occurrence of stress in others. Conversely, it has been proven that highly motivated workers come in regularly to work, work diligently, with more readily and are more flexible. Motivation is a critical element for any organization.

Specific opportunities to increase motivation vary, but generally it can be said that: To provide the best possible payment and job security - at least on average somewhere existing payment and favours for similar work. To bring style and practice of supervision that encourage motivation to work and set up working groups (team) of compatible personalities. Endeavour in the selection, recruitment, distribution and evaluation of staff reaching out to better match between individual capabilities and offer job opportunities; periodically to check whether people continue to feel that they are treated fairly and are assigned a suitable job and responsibility. To use all means possible to maintain good and open relations with all contractors. These include: friendly supervision of consultation character; regular joint consultations with senior executive staff; regular discussions with employee representatives / unions; effective assessment of individual performance and achievement based on disclosed properly justified and validated criteria; briefing groups; periodic examination/screening perceptions and attitudes towards work.

3) Predicting the success of the activity

Developing a system to predict the success of the activity involves the following stages:

- Psychological study of activity. The psychological structure of activity gives an idea of the necessary structure for capabilities of personality for this activity;
- Selection and development of methods for collecting baseline information. Optimal design of diagnostic methodology combines analytical - study of individual mental properties, and synthesis approach, i.e. imitation, creation of simplified models of the activity;
- Developing criteria, objective measure of the success of the activity. Used for this indicators must comply with the basic principles: adequacy purposes of forecasting; use primarily on objective indicators; using several indicators with separation of the lead or generalizeted evaluation of success;
- Conversion of output information in an integrated diagnostic evaluation. Used in systems for predicting the success of the activity statistical methods are: comparative analysis of final groups, a method of correlation pleiads, factor analysis, sequential analysis, regression analysis;
- Definition of validity of the system for forecasting and assessment of its acceptability for practical use. Comparing formed on the basis of informative indicators forecast with the objective characteristics of the success of the work of the same sample of persons is called **verify the prediction**.

If the system is valid, it is formed as a guide.

Used in procedure for professional selection methods are evaluated according to the criteria: predictive value (informative) - probability of correctly predicting the professional efficiency of the studied, measured by the coefficient of correlation between the survey results and success of operations; reliability - stability of the results obtained with a method in repeated studies of the same persons; differentiation (specificity) - orientation of each methodology for assessing the specific function or set of functions.

Diagnosis and prediction capabilities, assessment of employability, must be carried out only by persons with special psychological training.

Privacy, prevention of psychological disability and discrimination, require full information of the evaluated candidate and ensure agreement prior to the dissemination of information, data or diagnoses for him to others.

3.8. PSYCHOSOCIAL FACTORS OF LABOUR AND HEALTH

Related health and mental well-being issues include psychosocial stressors at work, in family and social life. They affect health and self-esteem through 4 types of closely interrelated mechanisms: emotional, cognitive, behavioural and physiological.

Psychosocial factors are associated with social structures and processes are mediated by perception and experience and include: 1) structures and processes in the environment (incl. and work) eg. rapid social change, destruction of cultural traditions that may cause pathogenic or, on the contrary, improving health effects; 2) individual determinants of human capabilities to respond to such stimuli (personality characteristics, habits, attitudes); 3) psychosocial conditioned physiological, psychological and social mechanisms (functional disorders in the excretion of hormones, anxiety, increased willingness to risk); 4) psychosocial conditioned mental or physical illness; 5) lowering of self-esteem, satisfaction, quality of life; 6) various aspects of the healthcare system, including events promoting health and efficiency of health care (Levi, 1979).

The common forms of psychosocial determined ill health and self-esteem is: a mismatch between individual needs and offer environmental opportunities for their satisfaction, mismatch between the capabilities of human and environmental demands, discrepancy between individual expectations and perceived situation.

One of the most significant psychosocial stressors in employment are: insufficient or too high load and pace of work, monotony, role conflict, limited opportunity for social contacts in the workplace, autocratic style of leadership, lack of social support from the supervisor, poor interpersonal relations and workplace violence (psychological terror), insufficient motivation to work and achievement, shift work, insufficient participation of workers in decision-making at work, lack of autonomy and control capability of the work performed, job insecurity and redirection, risk of unemployment.

Can be distinguished two groups work characteristics affecting mental health:

a) a permanent effect (absence deteriorating health, but their presence over the required not improve it): physical security and safety; recognition of social status; have the necessary financial resources;

b) the effect of further deterioration, when there is a negative relationship between high doses and health: control options; opportunity to use skills; foreign targets; diversity; clarity regarding the environment; the possibility of interpersonal contacts.

The negative effects of labour activities affecting fundamental and diverse areas of human life, so in literature speaks of "the long arm of the work." These include: short-term disturbances of psychosomatic condition and subsequent long-term deterioration of physical and mental health of workers; reduce intellectual capacity, especially the mobility of thought; passivity leisure and social sphere; education of children in a spirit of obedience and passivity; changing valuable orientations of the individual.

Adverse effects of the different characteristics of the work for the *mental well-being* is expressed in: anxiety associated with the subjective assessment of events and demands as a threat; depression, as an indicator of loss of control over the situation; low self-esteem, resulting from constant comparisons with others; psychosomatic complaints as a sign of poor coordination between body and psyche; irritability, as an indicator of depletion of mental resources, a common frustration.

The model of P. Warr related to labour welfare and *mental health* includes five components: emotional well-being, with basic dimensions "pleasure (joy)" and "activation"; subjective competence, which is seen as the capacity to cope with external and internal requirements and overcome emerging difficulties; autonomy as an individual feature to counteract the effects of environment and independently determine their own beliefs and actions taken; aspirations and claims reflected in motivated behaviour in search of new opportunities and efforts to meet the significant challenges; integrally functioning of the individual as a whole, relating to the complex interrelationships and balance between the first four components.

New directions in research and practical work in the field of psychosocial factors are **organizational health, mental violence in the workplace, psychological contract**.

Organizational health

Improper organization and management of the work require that the creation of unnecessary tension and stress, which is a breeding ground for the development of burnout and the emergence of psychological torment in the workplace. Organizational health is defined as the quality (general condition) the structure and function of the organization, its system of governance and its culture. Healthy are these organizations, "in which the differently components that determine their general condition, make them able to fulfill its goal, successful and adaptable and which are perceived positively by its workers" (Cox T., 2000). Therefore, preventing its important task falls to the executives who are responsible for the "health of the organization."

Mental violence in the workplace - mobbing

Mental violence in the workplace, reaching down to psychological terror, is an extreme form of psychosocial stressors at work. It involves systematically targeted, objectively unreasonable maltreating, hostile and unethical behaviour by one or more persons in respect of an individual who behaves in a position of helpless and defenseless with consistent repeat (at least weekly) discriminatory actions for several months. The term is indicated severe harassment horizontally (by colleagues) and vertical (head, bullying) to workers in different organizations with negative consequences for affected (increased irritability, psychosomatic complaints, reduced ability to complete nervous breakdown and diseases) and the organization as a whole (worsening labour morale and motivation, poor production, decline in productivity and waste, turnover) and society (reduced competitiveness, higher costs for health care, loss of work-day).

Main causes of mental violence are factors related to labour organization and management of work. Usually starts with a conflict, that has long been obscure or no clarify.

A quarter of workers in Western Europe have been subjected to systematic humiliation and psychological harassment in recent years. Mental harassment in the workplace is increasing rapidly and is a problem in all EU countries. The consequences of this phenomenon is expected to become the most widespread occupational disease of the future.

New guideline regarding "organizational characteristics - stress and burnout" is the transition from situational context **to organizational and managerial environment** (in which works) to the importance of the valuables that form the emotional and cognitive connection of people with their work. They are defined as a **psychological contract**, ie belief, what is required to give the employer, based on the perceived promise of reciprocal exchange. Today, employees are expected and required to give more with respect of time, effort, skill, flexibility, while in return they receive less in relations of opportunities for career development, job security, etc. . Violation of the psychological contract leads to burnout, because it destroys the idea of reciprocity, decisive to maintain prosperity. For this particular focus of development programs and boost staff, motivation and finding meaning in work performed at any level of emotional intelligence of managers.

Management of psychosocial problems. Work-related psychosocial factors are managed through control programs and reduce mental intension and stress in the workplace, including improved ergonomic working conditions, psychological design of

work, reorganization of tasks, improve communication within the organization, professional selection and staff training, incl. managers, raise individual abilities of employees to cope with potential stressors through various techniques for psycho-vegetative unloading and training in social skills.

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4 ERGONOMICS

The rapid development of modern production, automation and centralization his ubiquitous use of information and computing equipment, complication and increase the speed of technological processes, the emergence of more and more professions, associated with dynamic social progress, created the need for new interdisciplinary science of labour. She should summarize all the more valuable of the theory and methodology of various disciplines for human work, while immediately decided purely practical problems arising in specific employment situations. This interdisciplinary science is ergonomics (from Greek: Ergon - work and nomos - law) - **Fig. 4.1.** Its aim is the adaptation of labour to the physiological and psychological possibilities of man, with a view to securing the most efficient operation, without creating danger to his health, at minimal cost to biological resources. Ergonomics is divided into correction (or classical) and projective (or ergonomics systems). **Classical ergonomics** studies already created systems - a man - machine - working environment and considering mainly certain elements of the line of interaction between man and machine (mainly means for control - MC and means for presenting information - MPI) strive to adapt to the characteristics of operator. As a result formulate separate one-dimensional models, respectively requirements for workb instruments, working furniture, environment, etc. Ultimately corrective ergonomics as a result represents the sum of one-dimensional models (requirements) of the individual labour sciences (eg. hygiene of laboour, psychology of labour, etc.). Often these requirements are contradictory and it is almost impossible truly optimize the human work.

Projective ergonomics participate more in the development of a system man - machine - working environment and implement multifactor synthesis of joint system - i.e. - recognize interrelated impact on human activity of psychological, anthropometric and hygienic factors, defined by the corresponding parameters of the machines and work environment. In narrow aspect since its creation in the 40s of the twentieth century (also called **human engeneering** or **human factors** in the US) ergonomics deals with the correct adaptation of the machines and work place to man.

4.1. WORKPLACE AND ANTHROPOMETRIC CHARACTERISTICS OF MAN.

Crucial element in ergonomic design or correct workplace is complying with basic anthropometric characteristics of human - operator. It should be taken into account: **a)** linear (static) anthropometric data, **b)** the maximum amplitudes of movement in the joints, **c)** the maximum area the reach of hands **d)** the maximum muscle strength.

a) The anthropometric data according to BS 14386-77 regarding 5%, 50% and 95% of the population (i.e. men and women fifth, fiftieth and ninety-fifth percentiles). Eg. man fiftieth percentile P 50 is a typical representative of the average individual who has values of performance equal to the corresponding P 50 and P 5 man size is typical for the small person. For Bulgarian population basic anthropometric indicators are presented in **Table. 4.1.1.**

b) The maximum amplitude of movement joints are required in the ergonomic design and control necessary to optimize working space and freedom of movement in employment. On **Table. 4.1.2**. data are presented for upper limb (by Dempster).

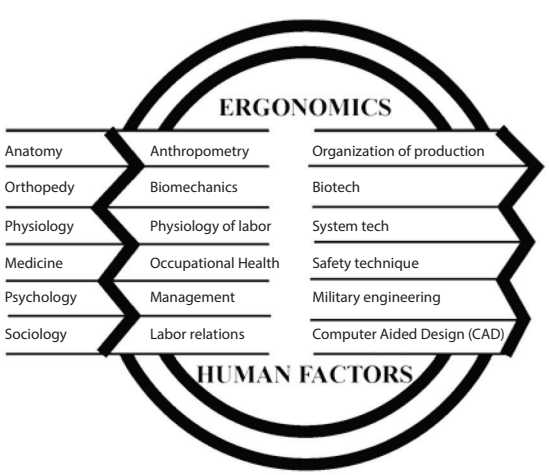


Fig. 4.1. Sources and applications of ergonomics

Tab. 4.1.1. Basic anthropometric indicators

Name of index	Female			Male		
	P 5	P 50	P 95	P 5	P 50	P 95
Indicators standing posture - in mm	(except	№2 in kg	and №49	to 54 in °)		
1. Growth	1488	1586	1675	1602	1707	1810
2. Body weight - kg	49	64	83	57	72	90
3. Height - pupils	1383	1478	1567	1488	1591	1697
4. Height - shoulders	1203	1294	1381	1300	1398	1495
5. Height - elbow	927	1000	1071	996	1081	1165
6. Height - clenched fist	645	702	761	683	747	814
7. Height to the fingers of the hand	547	601	657	574	637	698
8. Maximum width of the shoulders in arms	383	425	472	418	460	499
9. Maximum sagittal diameter of the chest	162	188	219	186	216	251
10. Maximum width of the hips	316	352	394	306	337	369
11. Length - upper limb	636	694	752	700	760	822
12. Length, armpit	258	294	328	282	316	353
13. Length, forearm	189	219	256	209	245	282
14. Length of palm with fingers	159	180	204	177	198	223
15. Maximum width of the palm	86	93	102	98	106	116
16. Thickness of palm	20	24	29	24	28	33
17. Length - foot	818	890	960	880	960	1035
18. Length - hip	408	459	513	436	493	547
19. Length - shank	308	347	390	329	376	425
20. Sole - height	73	82	96	76	90	105
21. Sole - length	224	242	261	245	265	286
22. Sole - width	89	97	107	96	107	118
23. Maximum reach forward	722	799	878	793	869	949
24. Maximum span of arms	1481	1598	1709	1625	1746	1876
25. Maximum span elbow	796	868	941	862	935	1020
26. Maximum reach up	1863	1994	2120	2032	2179	2324
27. Maximum reach up with bent fingers	1767	1898	2018	1928	2068	2212
Indicators sitting posture						
28. Popliteal height	369	402	439	407	450	494
29. Growth in sitting posture	788	838	890	843	899	953
30. Height of pupils	646	700	749	682	740	796
31. Height shoulders	522	564	607	557	602	646
32. Height - elbow	218	252	288	226	262	295
33. Height of the third lumbar vertebra	168	198	227	178	204	234
34. Depth of sitting	436	474	518	444	481	524
35. Distance between the plane of recline and top of the knee	526	570	621	545	592	641
36. Length of straight leg	858	942	1020	910	1000	1080
37. Maximum reach forward	737	802	874	801	870	950
38. Maximum reach up	1174	1259	1336	1279	1364	1451
39. Maximum reach aside	728	794	865	801	868	938
40. Maximum width of elbows	359	422	496	388	454	519
41. Maximum width of the seat	314	368	424	302	341	383
42. Maximum width of the hips	328	374	429	299	333	375
43. Maximum width of the knees	158	188	233	164	188	222
Indicators head						
44. Length	168	178	189	176	188	201
45. Maximum width	139	148	159	144	155	166
46. Front head height	192	212	232	207	229	253
47. Length of the ear	53	60	66	57	64	72
48. Width ear	28	32	39	30	35	41
Indicators of angular deflection - in plain degree						
49. Maximum angle of deflection of the foot - up	16	28	45	15	27	39
50. Maximum angle of deflection of the foot - down	22	37	52	29	39	51
51. Maximum turning angle of the hand - external	116	149	185	117	149	183
52. Maximum turning angle of the hand - internal	23	50	77	20	42	72
53. Maximum angle of rotation of the head - right	50	67	83	52	71	84
54. Maximum angle of rotation of the head - left	51	68	84	54	71	84

Tab. 4.1.2. Amplitude of the joint movement of the upper limb in degrees

Type joint motion	Average P 50	For percentile P 5	For percentile P 95
Wrist:			
flexion	90	114	66
extension	99	125	73
total	189	231	147
abduction	27	45	9
adduction	47	61	33
total	74	100	48
Forearm:			
supination	113	157	69
pronation	77	125	29
total	190	250	130
Elbow:			
flexion	142	162	122
Shoulder:			
flexion	188	212	164
extension	61	89	33
total	249	287	211
Horizontal:			
abduction	134	168	100
adduction	48	66	30
total	182	222	142
Rotations			
internal	34	60	8
total	131	179	83

c) The maximum area of reach, hands are different for sitting posture (BS 15371-81) and working right (BS 15262-81). **When sitting posture** for the basic of the cylindrical coordinate system which have maximum working areas of the hands is chosen point of sitting posture - the intersection of the plane of the seat, backrest and the symmetry of the human body - **Fig. 4.1.1.**

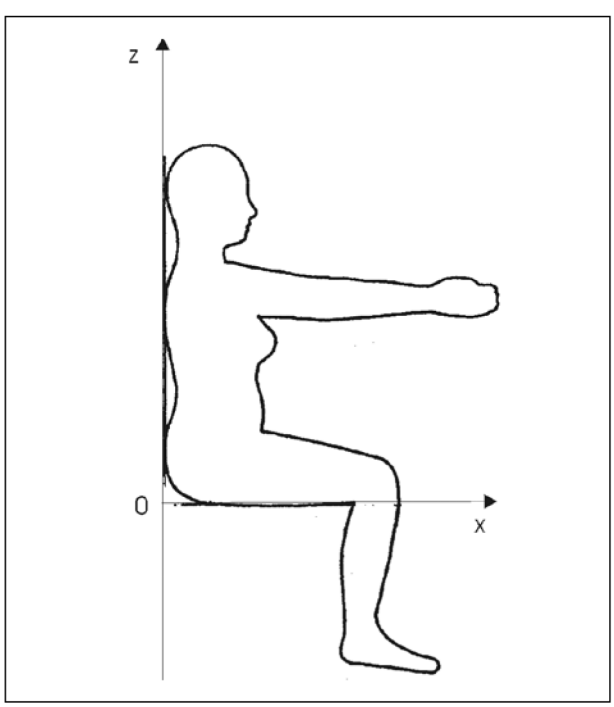


Fig. 4.1.1. Basic planes for ergonomic design of maximum areas to the reach of hands in a sitting posture

In ergonomic design or correct workplace maximum work areas comply with the dimensions of a woman P5, separately for right and left hand. This provides convenience for 95% of likely working (in other words, MC for example should not be placed beyond the maximum working area woman P5). For sitting position provided horizontal sections of maximum working areas at different levels of 0 (the plane of the seat) to the level of 1100 or upwards through 100 mm - **Table. 4.1.3.** and **4.1.4.** For posture straight reach zones in vertical and horizontal planes for a man of medium size they are determined by figures **4.1.2.** and **4.1.3.**

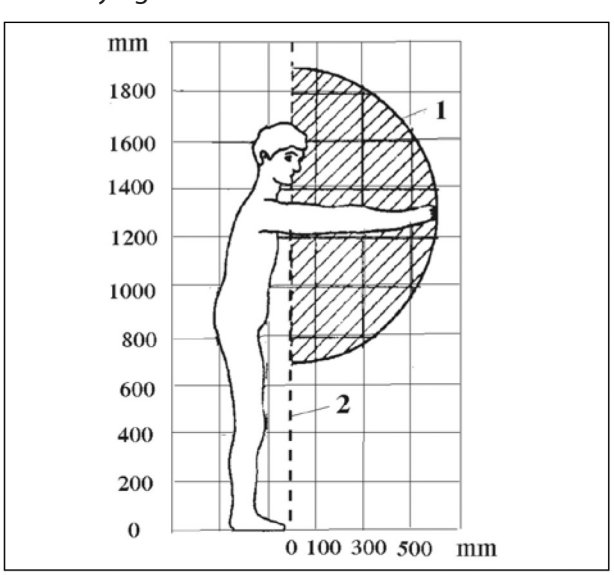


Fig. 4.1.2. Area of reach, in a vertical plane

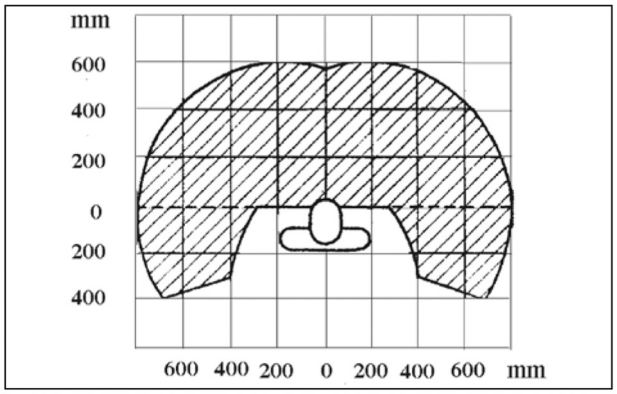
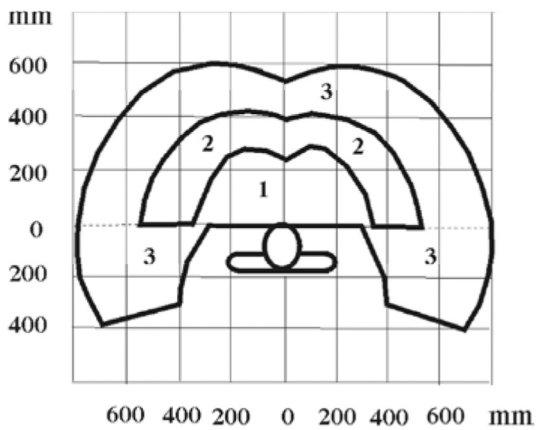
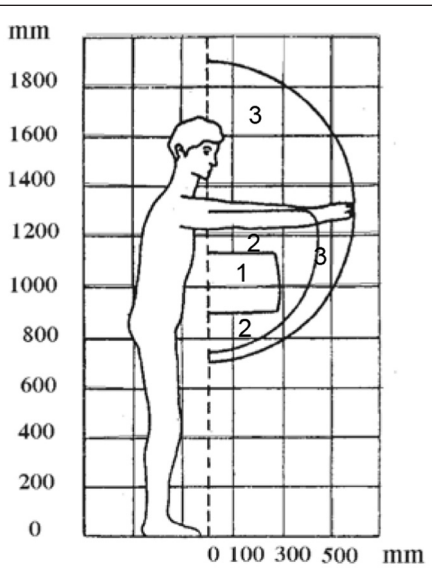


Fig. 4.1.3. Area of reach horizontal plane



1 - area available to the most important and many frequently used controls (optimal workforce)
2 - area available to frequently used controls (zone normal reach)
3 - area available to the rarely used controls (zone reach)

Fig. 4.1.4. Areas for the implementation of manual operations and positioning of controls in a horizontal plane



1 - area available to the most important and very frequently used controls (optimal workforce)
2 - area available to frequently used controls (zone normal reach)
3 - area available to the rarely used controls (zone reach)

Fig. 4.1.5. Areas for the implementation of manual operations and positioning of controls in a vertical plane

In all cases the aim is the implementation of manual operations, especially those that occur very frequently and frequently is within the optimal operating zone or within the normal reach - figures **4.1.4.** and **4.1.5.**

d). Maximum muscle strength is determined by the maximum effort that can support an individual for a few - 5-10, seconds.

On **Table 4.1.5.** are the values of maximum force exerted by hand at different angles at the elbow and different types of muscle actions. It should be added that the maximum muscular strength varies depending on: the relative position of the parts of the active limb, muscular effort type and stability of posture, and is facilitated by the use of various types of supports.

4.2. WORKPLACE AND WORKING POSTURE.

Working posture (WP) is an essential element of human work, respectively in the workplace. Fixing the body in a certain position enables hands, feet and eyes to perform certain movements on the implementation of an action. Maintaining appropriate posture leads to an increase in static muscle work. The smaller is the proportion of static muscle tension, the more rational is WP. The main positions in the labour process are listed in **Table 4.2.1.**

The aim is to seek optimal WP for the specific work. Since in practice distinguish two basic postures - standing and sitting, we will look at the basic features. In terms of biomechanics and physiology of labour sitting posture has many advantages: greater stability, small muscular efforts, low energy expense, optimum conditions for fixing the body, the shoulder girdle and upper parts of the arms, which allow for precise operation. Its disadvantages are mainly: limited motor freedom, static tension of the muscles (in unpleasing ergonomic work furniture) and stagnation of blood in the legs and pelvis (especially at constant sitting position - to the fifth, sixth hour of work).

Ergonomic optimization to sitting requires WP:

A. The implementation of operating procedures very often and often in optimal work area, or within a zone of normal reach - **Fig. 4.2.1.**

B. The construction of production equipment to create an opportunity to reduce static muscle work by regulating the height of the work surface, seat, backrest, space and footrests.

The adjustable parameters are determined by nomograms, and if this is impossible - according to the requirements, set out in **Tables 4.2.2.** and **4.2.3.** and **Fig. 4.2.2.**

Table 4.2.1. Types of job positions

Free		Coercive	
	1. Sitting normal		1. Sitting
	2. Standing normal		1.1. Leaning
	3. Sitting-standing		1.2. Hands up
			2. Standing
			2.1. Hands forward
			2.2. Hands up
			2.3. Inclined (to 30°)*
			2.4. Strong inclined (up to 30°)*
			3. On knees
			3.1. Normal
			3.2. Inclined (to 30°)*
			3.3. Strong inclined (to 30°)*
			3.4. Hands up
			4. Squatting
			4.1. Normal
			4.2. Hands up
			5. Lying with his hands up
			* Tilt the body from the vertical

Table 4.1.3. Maximum work areas - right-handed women 5 P in mm

Polar angle *	Level											
	0	100	200	300	400	500	600	700	800	900	1000	1100
-90	-	290	303	307	312	310	310	300	295	280	266	246
-80	-	300	320	399	343	341	340	334	318	299	267	253
-70	290	325	356	372	381	385	385	377	358	339	307	284
-60	313	352	391	421	439	445	441	432	406	378	341	304
-50	348	399	444	478	500	510	510	500	473	438	396	345
-40	399	448	498	530	557	568	570	558	585	500	455	395
-30	451	500	545	582	607	618	618	607	581	542	500	440
-20	296	543	588	626	658	658	653	638	618	585	542	480
-10	520	568	616	656	682	695	632	675	643	607	561	518
0	541	595	643	685	710	718	715	708	772	630	578	525
10	547	617	668	708	723	730	728	715	680	639	582	522
20	559	632	680	715	732	738	738	722	698	650	585	518
30	568	639	690	720	738	740	740	732	710	667	600	510
40	570	640	690	720	742	745	745	735	719	673	600	510
50	558	638	687	720	735	740	740	737	718	667	598	506
60	540	628	678	709	727	738	740	730	708	660	598	506
70	590	620	670	700	720	730	730	722	700	660	595	495
80	507	598	650	682	702	710	710	705	690	648	579	490
90	480	575	628	659	675	680	680	678	665	636	570	490
* Divergence angle of the hand in front position: (-) on the other hand, (+) to the same hand												

Table 4.1.4. Maximum work areas - left hand for women 5 P in mm

Polar angle *	Level											
	0	100	200	300	400	500	600	700	800	900	1000	1100
-90	480	575	628	659	675	680	680	678	665	635	570	490
-80	507	598	650	682	702	710	710	705	690	648	579	490
-70	530	620	670	700	720	730	730	722	700	660	595	495
-60	540	628	678	709	737	739	740	730	708	660	598	506
-50	558	638	687	720	735	740	740	737	718	667	598	506
-40	570	640	690	720	742	745	745	738	719	673	598	506
-30	568	639	690	720	738	740	770	732	708	667	600	510
-20	559	632	680	715	732	738	738	722	698	650	585	518
-10	547	617	668	708	723	730	728	715	680	639	582	522
0	541	595	643	685	710	718	715	703	672	630	578	525
10	520	568	616	656	682	695	692	675	643	607	561	518
20	496	542	588	626	653	658	658	638	618	585	544	480
30	541	500	545	582	607	618	618	607	581	542	500	400
40	399	448	493	530	557	568	570	558	535	500	455	395
50	348	399	444	478	500	510	510	500	473	438	392	345
60	312	352	391	421	439	445	441	432	406	378	341	304
70	290	325	356	372	381	385	385	377	358	333	309	284
80	–	300	320	339	343	341	340	334	318	299	267	253
90	–	290	305	307	312	310	310	300	295	280	264	246

* Divergence angle of the hand in front position: (-) on the other hand, (+) to the same hand

C. To assure optimal angle of comfort for body parts. For example, for a job of truck driver comfort angles that provide minimal static tension of the body are presented in **Fig. 4.2.3.**

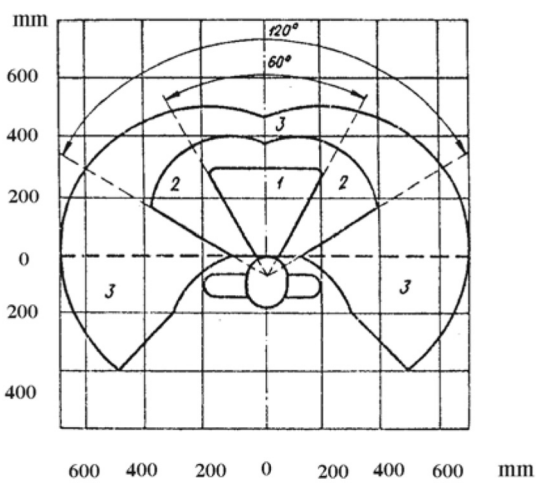
D. When work allows it to provide support arms by armrests installed on the operating chair or work surface.

Table 4.1.5: Maximum muscle strength at different angles at the elbow and different muscle actions

Type of action	Angle at the elbow - degrees	Percentiles (kg) P 5	Percentiles (kg) P 50	Percentiles (kg) P 95
Propulsion	60	15.4	41.7	68.0
	90	16.3	39.0	69.8
	120	16.3	46.7	78.0
	150	19.0	55.8	88.0
	180	22.7	62.6	95.3
Tow	60	10.9	28.6	33.6
	90	16.8	39.9	61.2
	120	19.0	47.2	69.8
	150	25.4	55.3	85.7
	180	23.6	54.4	77.6
Lifting	60	9.1	22.2	37.2
	90	9.1	25.4	48.1
	120	10.9	27.2	56.2
	150	8.2	25.4	53.5
	180	6.3	19.5	39.9
Descent	60	9.1	23.1	40.4
	90	11.8	24.0	39.9
	120	11.8	26.3	44.4
	150	9.1	21.3	36.3
	180	7.7	18.6	37.2
External rotation	60	9.1	23.6	39.5
	90	8.2	22.7	44.0
	120	10.0	24.0	45.4
	150	9.1	24.5	47.2
	180	9.1	22.7	47.2
Internal rotation	60	7.7	19.0	37.2
	90	7.3	16.8	30.8
	120	6.8	15.4	28.1
	150	6.8	15.0	29.0
	180	6.3	15.4	28.1

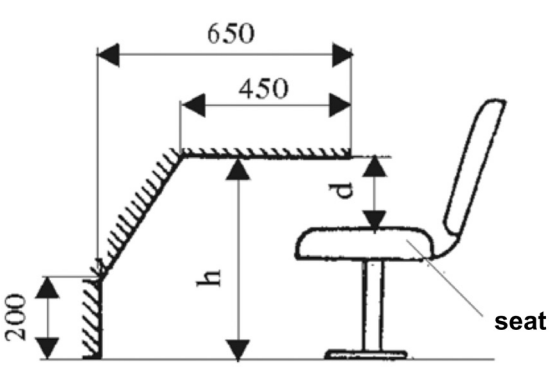
Table 4.2.2: Recommended parameters for the height of the work surface in mm

Types of work	Height work surface		
	female	male	female and male
1. Many fine visual work (assembly of watches, engraving, cartography installation of small details)	930	1020	975
2. Fine work (assembly of small parts machining operations requiring high precision, etc.).	835	905	870
3. Light work (installation of medium machine parts, activities no requiring high accuracy, etc.).	700	750	725
4. Rough work (light assembly work at a large parts, printing machine, topographic machines, etc.).	630	680	655



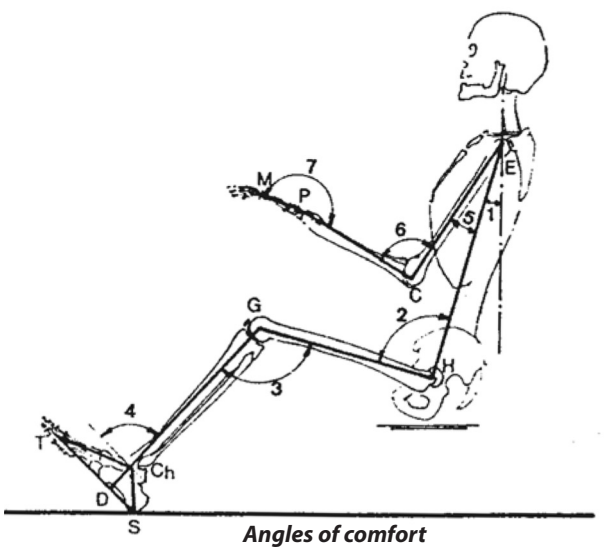
1 - area available to the most important and frequently used controls (optimal workforce)
2 - area available to frequently used controls (zone normal Reach)
3 - area available to the rarely used controls (zone Reach)

Fig. 4.2.1. Areas for the implementation of manual operations and positioning of controls in a horizontal plane



d - *distance from the seat to the lower edge of the work surface, not less than 150 mm*
h - *height leg area*

Fig. 4.2.2. Recommended parameters of the workplace in the course of work sitting



Angles of comfort

1 - between 10 ° and 20 °
2 - between 90 ° and 110 °
3 - between 95 ° and 120 °
4 - between 90 ° and 110 °
*5 - 15 ° (extension) and 45 ° (flexion) **
*6 - between 80 ° and 160 ° **
*7 - between 180 ° (radial inclination) and 190 ° (cubital inclination) **
7 - between 170 ° and 190 ° (flexion-extension)
*** With support arm*

Fig. 4.2.3. Recommended angles of comfort

WP standing preferred: when performing hard and medium physical work; technological conditioned sizes are large manufacturing equipment; serve several machines. It has the following advantages: provides greater motive freedom and force; the worker can move into the work area allowing to increase the working field of arms and legs, while expands and the visual field. Its disadvantages are mainly: increased energy consumption (an average of 10-12%, while bending forward up to 47%); increased muscle tension especially the pelvic girdle,

legs, spine and neck; increase local muscular fatigue of the legs and in the waist; difficulties in the blood circulation of the lower limbs; deformations of the sole arch and possibly knee and intervertebral joints - with longer standing; ptosis female genital under similar working conditions, especially combined with the individual characteristics of the female body; reducing the threshold of external stimuli, the speed of reflexes and level of attention - as a consequence of general fatigue.

Ergonomic optimization of the WP standing requires:

A. Ensuring the standings and freedoms position of working with tilt forward up to 0.2618 rad (15 degrees).

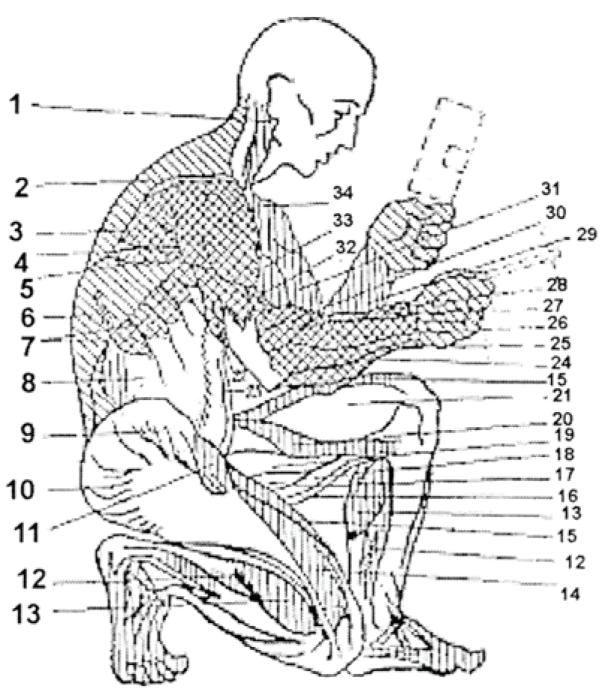
B. Adjust the height of the work surface (or footrests). The adjustable parameters are determined by nomogram, and when this is impossible - according to the requirements set out in **Table 4.2.4.**

Table 4.2.4: Recommended height of the work surface for put on shoes operator (heel = 3 cm.), posture standing

Category work	Height work surface of workplace - in mm		
	women	men	women&men
Light	990	1060	1025
Average	930	980	955
Hard	870	920	895

C. It must provide space for entry of soles with dimensions not less than 150 mm deep and 550 mm in width. **D.** Whenever it is possible to use the chair-seats for support the seat. In terms of physiology and psychology of labour optimate is free seated-standing posture. Example of unfavorable with significant static tension is forced to knees (squatting) posture in operation welding - **Fig. 4.2.4.**

Fig. 4.2.4. Muscles of the body (static and dynamic loads) in working position of welder



Legend

Dynamic load

Significant static load

Negligible static load

Relaxed state

1. chest - clavicious-mastoid muscle
2. trapezius muscle
3. small round muscle
4. muscle below the crest of the scapula
5. the large circular muscle
6. broad back muscles
7. front toothed muscle
8. abdominal external oblique muscle
9. average gluteus
10. gluteus muscle
11. stretching the general fascia
12. sunny muscle
13. the three-headed calf muscle
14. inner broad muscle of the thigh
15. right thigh muscle
16. the semi tendinous muscle
17. adduction, large muscle
18. thin muscle
19. long adduction muscle
20. sewing muscle
21. inner broad muscle of the thigh
22. triceps shoulder
23. rectus abdominis muscle
24. elbow extensors wrist
25. wrist extensors
26. extensors fingers
27. the short thumb extensors
28. the long thumb extensors
29. the short radial wrist extensors
30. the long radial wrist extensors
31. the brachial-elbow muscle
32. biceps
33. deltoid muscle

4.3. WORKING FURNITURE AND TOOLS

Working furniture is extremely essential for ergonomic optimization of work (especially in terms of anthropometric characteristics and working posture). Her includes chairs, seat devices, tables, desks, countertops and others.

A. Chairs and Seat devices. Commonly for productions, the main requirement is the chairs have adjustable dimensions of height and position of the seat and backrest and not to restrict the mobility of the body (though it depends on the specific requirements of the activities - eg. fixing body when pilots). For proper distribution of buttocks seat can be upholstered soft fabric or slightly concave, and avoid stagnation of blood in the pelvis should have a gentle slope in front-rear position of 3 to 5 degrees.

The backrest should be adjustable incline. It is right to secure and support legs by pedestal. Recommended sizes of chairs and armchairs are listed in **Table 4.3.1.**

The design of office chairs, seats and more it is best to select the specific work.

The main requirement for ergonomic **tools** is their size, shape and weight to create optimal conditions for the implementation of the work moves at the rational posture. Below are given some basic requirements for the most commonly used hand tools:

Hammers used to work with one hand must not weigh more than 3 - 3.5 kg (without handle) length of the handle is between 250-350 mm.

Shovels may be of different lengths (900-1300 mm), while their selection is better to comply with the requirement standing to body shovel to reach the lower limit of the chest.

Places to catch tools is to be constructed after accurate mechanical analysis of catch - **Fig. 4.3.1.**

4.4. WORKING MOVEMENTS

Optimum locomotor activity of man - operator is achieved with correct organization of the working movements based on the following principles:

1. Saving movement. The aim is to achieve the effect using the most appropriate class movements for activity. There are 5 classes movements - see **Table 4.4.1.**

2. Achieving optimal force working movements for a given job and the class of movements. Those with smaller class have less power. In general

Table 4.3.1: Basic dimensions of the working chairs

Parameter	Dimensions in mm				
	Chair		Armchair		
	firm	semi soft	firm	semi soft	soft
Height seating	440	450	440	430	380-420
Height of seat from the floor	850-870	850-870	850-870	850-870	800-1000
Seat width front	450	470	540	570	600
Width of the seat back	410	-	400	-	-
Full depth	520	520	400	600	750-880
Height of the armrests of the seat	-	-	130	-	-

B. Desks, Countertops others. The height of the working surface is the most significant parameter. In general rule, it is at the height of the elbow, both in straight and in a sitting posture. It is best that height is adjustable. The depth of the work surface should be at least 700, and the width - at least 1000 mm. When on a surface (table, countertop) work more persons, the minimum distance between seats should be 300 mm, and the space for each of the workers (in depth and width) - minimum 675 mm. The shelves are convenient when easily reach and can be seen from the workplace. Ergonomic requirements are: a) top shelf at a height of up to 1900 mm, and the bottom - at least to 1000 mm from the floor; b) the depth and dimensions are determined by the size of the stored material. It is recommended that the depth of the above is smaller than that of the bottom shelf, as the minimum distance between two shelves is 250 mm for the upper and lower to 375 mm.

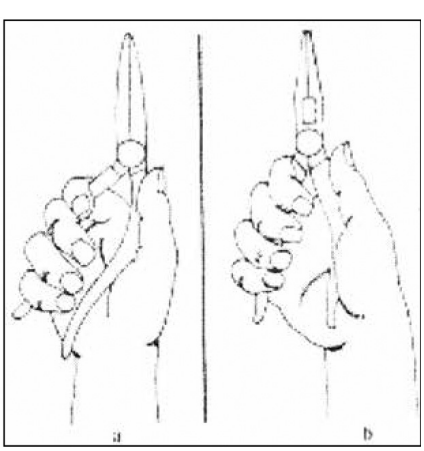


Fig. 4.3.1. Design) of pliers to avoid abduction of the wrist and a better positioning of the fingers

Table 4.4.1. Characteristic of work movements *

Class	Axis motion	Moving parts
I	Knuckles	Fingers
II	Joints of the wrist	Fingers + wrist
III	Elbow joint	Fingers + wrist + forearm
IV	Shoulder joint	Fingers + wrist + arm + forearm
V	Trunk	Fingers + wrist + forearm + arm + trunk

* Optimal movements are to III class.

strength should not exceed 33% of the maximum strength of working muscles, and must be used biomechanical working conditions. For example, optimum strength of the hand is achieved at an angle between arm and forearm 120 - 150 degrees. Foot this angle is about 120 degrees (sole, lower leg and thigh).

3. The speed of movement must be between 120 and 190 cm/s at a load up to 15 kg, the load at 45 kg - less than 25 cm/s. Moreover, movements to himself in the horizontal plane, forward - backward are faster than those of himself, in the vertical plane and away. The speed of the rotating movements is 1.5 times faster in comparison with the progressive (a straight line).

4. The rate of movement depends on the amplitude and class, and accuracy requirements. The optimal rate in production is about 2 moves per second of I-II class movements. Fewer tiring is a little faster than slower rate.

5. Both hands should begin and end their movements at the same time, but in the opposite direction, symmetrically and simultaneously. This leads to a reduction in eye movements as they are fixed in one area or move slightly. Very often the work is organized so that when one hand takes a piece, while the other leaves the treated article or taking tool. Thus the time to perform certain operations may be reduced to 40%.

6. Circular and continuous movements are more economical for the body than those in a straight line and interrupted movements. Ballistic movements are faster, more accurate and easier to implement, because usually involving only one group of muscles (flexors or extensors). For this they should be preferred.

4.5. MEANS OF PRESENTING THE INFORMATION (MPI) AND CONTROLS (MC)

To manage the majority of modern machines human operator is necessary information is obtained without transformation from sensory organs to the operator. The nature and complexity of the means of obtaining information is divided into:

A) Direct emitted by the machine information - such as noise engine, the smell of substance, hardness of a surface, color of material or process (of molten metal, flame, electric arc), etc. The reception and interpretation of these signals requires almost always considerable experience and excellent psychophysiological state - high perceptive capacity of the sensory organs of the working man.

B. Coding information, provided by appropriate means (MPI).

The most commonly used visual and auditory MPI. Their choice depends on a number of characteristics of the transmitted information and work process - **Table 4.5.1.**

Visual MPI must meet the following basic requirements:

1. To be clear, legible inscribe and properly labeled, in compliance with distance monitoring. Usually the size of the scales, digits etc. provides monitoring of about 694-700 mm (length of the hand of a woman P 50 or man P 50). For other distances these sizes must increase or decrease.

2. The information that give to read quickly and without hesitation.

3. Give only necessary to process information (no excess).

4. The information to be given to them in the most direct form.

5. To be clearly distinguishable from each other and from the background of the machine.

6. Layout, grouping and their subordination to be in the comfort zone - the best in the center of the visual field, as required for priority, frequency and consecutivity of their use.

7. Scales "open window" and the round is read by at least mistakes and should be preferred in accuracy requirements.

8. With the best readability are scales that use natural sequential order of the numbers - 0, 1, 2, 3 ...; 0, 10, 20, 30.

9. To meet the stereotypes of human behaviour. For example, to mark the intensification of a process to use the principle of movement of the clock-clockwise, or from left to right.

10. When is possible to use MPI with pictorial images. They are perceived very easy (especially if combined with inscriptions designation) without

Table 4.5.1.

Hearing MPI	Visual MPI
1. Simple message	1.Complicated message
2. Short message	2. Long message
3. The message is not related to subsequent messages	3. The message will be associated with subsequent messages
4. The communication talk about events taking place in time	4. The communication about position in space
5. The message requires immediate action	5. Communication does not require immediate action
6. Visual system of operator is overloaded	6. Auditory system operator is overloaded
7. The point of obtaining the information is too brightly lit or require full dark adaptation	7. The point of receiving the message is very noisy
8. The work of the operator impose constantly moving	8. The work allows the operator to remain in fixed position

hesitation and do not require large production experience - **Fig. 4.5.1.**

Of course there are different degrees of pictoriality - such as photography or television images are at one end of pictoriality, and maps, radar indicators, mimic, especially compasses that show some of the spatial relationships of presenting information that they, are on the other end of pictoriality .

11. MPI are compatible with the corresponding controls (MC). Ideally, MPI and MC must be designed and located so that the operator no/or small training can choose corresponding MC and work with him.

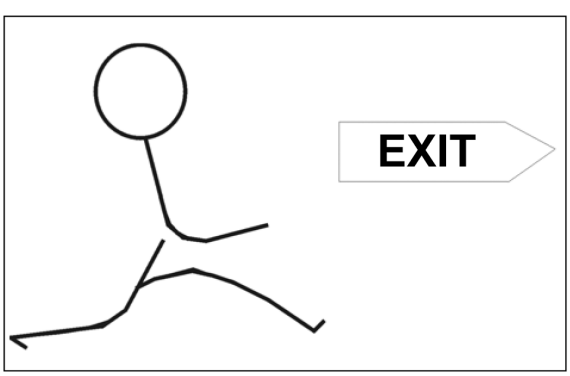


Fig. 4.5.1. Combined pictograms and inscriptions designation for emergency exit

Hearing MPI must meet the following basic requirements:

1. To hear well in the work area and work place - i.e. be selected with appropriate intensity and frequency response of the acoustic signal.

2. Use to transmit signals mostly acoustic origin - especially for human speech.

3. To always prefer when:

a) the visibility is limited or impossible

b) work in conditions of hypo- and anoxia, as man continues to hear good signals until the vision was seriously exacerbated by hypoxia (as in pilots and others.).

c) is necessary to guarantee secrecy of the message. (The speech can be understood easily by side listener, while encoded sound or noise signals can be formed so, that they are not clear to unwanted listener.)

d) is necessary flexibility of connection and/or rapid bilateral exchange of information (especially if you use the MPI base on human speech).

e) it is necessary to identify those who submitted information (the source of the message).

MC, allowing the operator to change control of processes and machines, can also be sources of information - most often visual, tactile, kinesthetic and auditory. Thus they may allow the operator to exercise retroactive control of employment.

In the modus operating differ two major classes MC: those pressures (mainly buttons - hand and foot) and such a move (handles, levers, cranks, wheels, pedals, etc.). - **Fig. 4.5.2.**

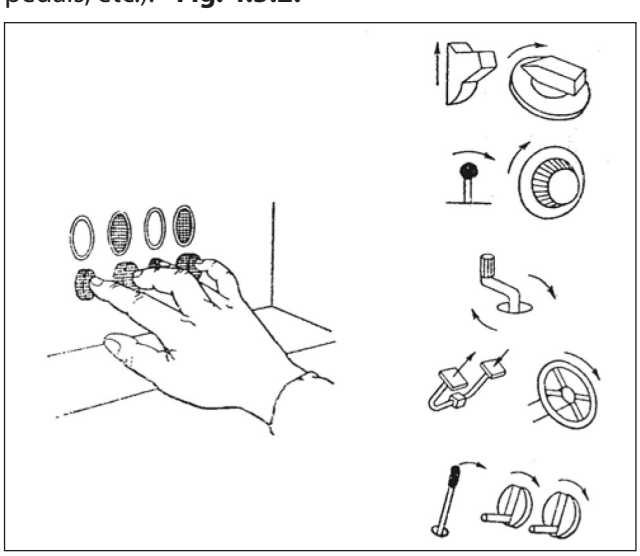


Fig. 4.5.2. The most common MC of pressure or by moving

When choosing a MC usually consider: the purpose, function, as well as some ergonomic and design features - **Table 4.5.2.**

Table 4.5.2: Principles for selection of controls

Functions	Designation	Type of control	Construction considerations	Ergonomic considerations
choosing between alternatives	start and stop equipment; sequence start and stop; temporary submission of the signal	tumbler; switch lever; foot switch	"Hammering" when switching; visually well differing various positions of MC	natural direction of movement; rational estimate of efforts; correct position, form comfortable to hold
choice of three of more options	choice of communication channel; range selection; set the operating modes of the equipment	switch with indicator fixing position; tumbler, switch lever	fixation positions	same as above
accurate adjustment	continuous adjustment of parameters; fine tuning and calibration	knob	fluency in the execution of operations; friction	same as above plus compatibility movements MC whereabouts of the MPI
crude tuning	continuously controlled (choke); change the characteristics of the channel; faucet	knob; joystick; pedal; helm	same as above	same as above
quickly adjusting the values of the parameters	for example, adjusting the electronic directory of the course on a plane	tumbler or switch lever, enforcement electrical translation	fluency in the execution of operations; small or medium resistance (friction) depending on the size of the MC	same as above plus optimal speed of MPI device
applying great effort	steering control; clutch; brake pedal (without booster)	joystick; wheelhouse; pedal foot control	optimal size	same as above

MC must meet the following basic requirements:

1. To have the reach areas.

2. To be well distinguished from the background of the machine and each other by color, tactile, form, inscriptions and combined coding.

3. Their dimensions conform to the anthropometric characteristics of the operator to meet its physiological capabilities (especially strength and biomechanical characteristics).

4. The drive can be carried out as follows: a) forward - inclusion; move forward - enhancing b) to the left - off; move left - reduction) to the right - inclusion; move right - strengthening

5. Emergency levers are in the direction of movement "to myself."

6. When requirements for greater accuracy, speed and low power use manual, while demands for greater strength and inaccurate movements - foot MC. Foot MC only be used when possible sitting posture.

7. Their distribution in work areas is even and compatible with the requirements for priority, sequence and frequency of their use, the direction of their movement is co-ordinated with muscle coordination.

8. To elect MC, which can easily be recognized mainly through standardization of their coding and position. The main and emergency MC need to identify well as visual and as tactile.

9. When dealing with MC in any case to seek a reduction in the part of optic retroactive control at the advantage of increasing touch and proprioceptive one.

4.6. LIGHTING AND VISION

In 80% of cases human-operator perceived by sight, and in some types of work eye left only its entrance. That is why one of the most important scientific and practical problems of hygiene and ergonomics is the continuous adaptation of the physical nature and hygienic qualities of lighting, physiological characteristics of vision and the specific requirements of the type of visual work.

4.6.1. ELEMENTS OF PHYSICS

1. Definition and properties. For visible light is usually accepted that part of the spectrum of electromagnetic radiation having a wavelength between 400 and 800 nm. According to some authors this limit varies from 380 to violet - to 1050 nm - a

red - or rather on short infrared rays. It should not be overlooked, however, its dependence as irritant central nervous system and synchronizer of circadian biological rhythms.

2. Intensity or power of light of a light source is measured by unit candela - cd. A candela is 1/60 the power of light emitted in the direction perpendicular to the surface of the absolutely blackbody area of 1 cm^2 at the temperature of solidification of platinum (1760° C).

3. The luminous flux emitted by a light source is measured by unit lumen - lm. A lumen is the luminous flux emitted in a solid angle of 1 steradian (sr) by source of light intensity 1 cd (1 lm = 1 cd. 1sr).

4. Lighting is the ratio of the magnitude of the luminous flux fall on a surface to its area. Measured in lux (lx). It is one lx, when on a surface of 1 m^2 fall 1 lm. The lighting increases with increasing luminous flux.

5. The brightness is the brilliance of a surface. Measured in cd/m^2: 1 cd/m^2 = 1 nit (nt - unit not included in SI). For a given lighting it depends on the reflectance **(see part 4.6.4)** of the illumination surface expressed in the following equation:

brightness = 0.32 x lighting x reflectance

fatigue, etc.

Besides the static meaning in ergonomics and occupational medicine there, so-called kinetic or dynamic visual acuity. It is particularly important for professional activities related to monitoring or detection of moving objects (working with video display, oscilloscopes, radar screens, in conveyor, transportation, military, etc.).

2. Adaptation of vision according to the lighting and brightness is one of the most important features of the visual system as a self-regulating system in the labour process. On the base of phenomenon of adaptation lie physiological, biochemical and photochemical processes that lead to changes in the threshold of sensitivity to light in a huge range - 1 to 10^5.

The adaptation takes place in a complex of visual system, but can differentiate such levels of pupil and the retina. Pupila adaptation is extremely fast. A few tenths of a second pupil shrinks harshly reduces the light beam to the retina and thus protects it from overlighting.

Adaptation of the retina level include (depending on the amount of light falling on it) double process of regulation: more nervous - relatively quick

Table 4.6.1. Dependens of the visual acuity by brightness

Lighting in lx	Visual acuity	Lighting in lx	Visual acuity
0.01	0.14	4.4	1.35
0.055	0.22	11	1.62
0.11	0.27	22	1.93
0.22	0.90	55	2.00
1.1	1.20	110	2.10

4.6.2. PHYSIOLOGY AND FUNCTIONAL POSSIBILITIES OF VISION IN LABOUR

Visible objects emit light rays that pass through the pupil and the optical system of eye and fall on the retina. Its cells transform light energy into nerve impulses that pass through the optic nerve and brain structures reach the cerebral cortex. There occurs the phenomenon of awareness of visible images.

1. Visual acuity is one of the most important visual parameters for the pursuit of the occupation. The static visual acuity is determined by the minimum distance between two points can still be seen separately. It improves for the same size monitored by raising the level of brightness within certain limits - **Table. 4.6.1.**, also by increases the differences in the brightness of the subject and the background. Visual acuity (excluding age) influence though to a lesser extent, other factors such as the color of the object, in real time - day or night - for the performance of work tasks, general condition of the body - especially the supply him with vitamins A and B_1,

and photochemical road - slow. The time for adaptation to darkness is long: in the first 5 minutes, it is fast enough, then slowed down and after about 35-40 minutes sight almost completely adapted to the darkness (recent studies have shown that this process is completed after about 60 minutes).

Adaptation to light is much faster, but to be fully effective it also requires time from 30 to 60 minutes. They distinguish two phases: rapid - about 0.05 s, which reduces the sensitivity of the retina to 1/5 of its initial value, and slower, which is based on the reactions of degradation - regeneration of photosensitive substances. In practice, for better adaptation to light are needed 8-10 minutes, as the higher speed observe the first 2-3 minutes. Closely related to the adaptation is process of glare, which occurs when poorly designed conditions of visual labour. At blindness brilliance of the light source is such that it is impossible any adaptation. The relative blindness may be due to too large contrasts in the visual field.

3. Accommodation to distance plays a significant role in the implementation of labour activities.

Accommodation is realized by changing the projection lens of the eye under the influence of the ciliary muscles. Punctum proximum (PP) is the nearest to the eye point that is clearly visible. For children, it is 6 cm, and for adults after the age of 40 - 15 cm. Punctum remotum (PR) is the most distant point that is clearly visible in the absence of any accommodation (ie completely relaxed ciliary muscles).

In unfavorable from an ergonomic standpoint conditions for visual work - especially in poor lighting and little distance to the observed object - suffers speed and reducing distance of accommodation (the distance that separates punctum remotum and punctum proximum). Therefore, when designing the system man - machine - working environment, depending on the distance to the visual object 3 different categories of visual work: **A.** Work at distances of the observed object over 183 cm. Practical in these cases there is almost no intension of accommodation and convergence. **B.** The distance to the visual object is between 30 and 182 cm (most often about 56 cm). Accommodation and convergence are loaded, especially if the distance to the subject is less than 50 cm. From the ergonomic point of view can be located, on the indicator devices at least 33 cm, but most preferably they are at least 50 cm from the eye. **C.** The distance of observation below 30 cm. Very high load convergence and accommodation whose intension can reach 8. **D.** By increasing the contrast between object and background accommodation efficiency grow.

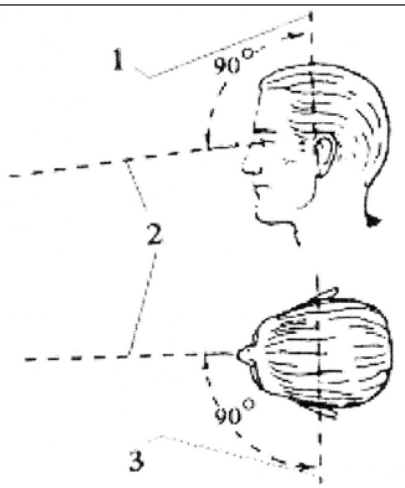
4. In binocular vision accommodation runs jointly with proportional reflex convergence of the eyes. Surely monocular vision is sufficient in some cases to assess the objects of observation in space, but only stereoscopic vision allows real assessment of depth. Both eyes stand each other about 5,6 cm and therefore see objects not quite the same. The brain combines the images of the two eyes in one which has a depth of image. This process is performed by convergence, while nearby objects, lines of sight in both eyes converge until they meet on him. Viewing far apart the lines of sight, while too distant targets the lines of sight of both eyes are almost parallel. For the needs of ergonomics and occupational medicine is important so-called threshold of stereoscopic vision, which decreases with increasing lighting (i.e. stereoscopic vision is improving).

It should be noted that about 5% of adults do not have quality depth vision, which limits their ability to exercise many of professions from transport, warfare and more.

5. Peripheral vision and visual field. This is the part of space that can spread fixed eye. Due to the movements of the head and binocular vision field of view from an ergonomic point of view is considerably larger. Here are these important concepts:

a). Standard line of observation. Starting from the base of the nose and is perpendicular to the transverse (to the line of the ears) and the vertical axis of the head of the worker, irrespective of the movements of the eyes. Since the vertical axis of the head of the seated or standing operator is slightly tilted forward (about 5 °), the standard line of observation is 5 ° below the horizontal **(see Fig. 4.6.1.)**.

Fig. 4.6.1. Standard line of observation



1. The vertical axis of the head
2. Standard line of observation
3. Transverse axis of the head

b). Normal line of observation. It is a line of observation in fixed eyes, passing 10 ° lower than the standard line of observation, since as is usually the eyes look down (seated man - 15 ° below horizontal).

c). Distance of observation is the distance from the base of the nose to the visual object.

d). Optimum angle of vision. The angle horizontal and vertical line of standard line of observation within which the operator can see the observed target only with movement of the eyes.

e). Maximum angle of vision. In its borders person can see objects with eye movement and head. The data for the optimum and maximum angles of vision are given in the **Table. 4.6.2.**

The visibility of an object in the visual field also depends on: its size (category visual work), lighting, brightness, contrast with the background and the lighting of the background, exposure time. If insufficient lighting eg., should increase the size of the object, and back if necessary to distinguish the object in a very short time (0,1 s) lighting should be very high.

The boundaries of the visual field narrows with age, wearing glasses, fatigue vision, and in some special cases (eg. when driving increases the speed of the vehicle).

Tab. 4.6.2. Angles of vision (visual perimeter) for sitting, standing, lying on their backs operator

Movement	Direction	Horizontal angle *	Horizontal angle *	Direction	Vertical angle *	Vertical angle *
		Optimum	Maximum		Optimum	Maximum
Only head	Left	0	60	Up	0	50
	Right	0	60	Down	0	50
Only eyes	Left	15	35	Up	0	25
	Right	15	35	Down	30	35
Head and eyes together	Left	15	95	Up	0	75
	Right	15	95	Down	30	85

* In degrees from the line of observation, lying in a horizontal plane

Many of jobs in transportation, construction, military, with the task of monitoring and control require a very good peripheral vision. This should be considered in ergonomic workplace design, as well as professional guidance and selection.

The brightness contrast is essential for good visibility under certain lighting. It show how the brightness of the object is distinguished from the background. It is expressed by the following equation:

Contrast = (B1 - B2) / B2, where B1 is the brightest, and B2 darker part of the two contrasting areas.

At low contrast visual acuity is lower - eg. black object on a gray background will be visible harder than on white. The dependence of the perceived brightness contrast of lighting is presented in **Table. 4.6.3.**

Example: The reflectance of white paper 0.8 (white paper reflects about 80% - i.e. white retain 20% of the light), the dark green paper - 0.1-0.2 and black letters - with 0.01. The visual task is reading black letters on a white background with lighting 50 lx. The brightness of the white paper is 50.0,8 = 40 lx, and the letters 50.0,01 = 0,5 lx. The difference between the two luminance is 40 – 0,5 = 39,5 lx. If the paper is dark green, difference is 50. 0,1-0,5 = 4,5 lx. It is clear that for the text to be read as well, as white background lighting level should be increased to 400 lx (400. 0,1 - 0,5 = 39,5 lx). This example shows that one sees objects because of differences in brightness and/or in colour.

Color vision is primarily a function of the macula lutea, as it is in this part of the eye are concentrated heel ends that perceive the basic three colours - red, green and violet. The eye is not equally sensitive to different colours **(Fig. 4.6.2)**. During the day most -well visible yellow-green part of the spectrum with a wavelength of 555 nm. In times of dusk or night, the curve shifts to the left (almost 40-50 nm) and best seen is green-blue radiation, the relative visibility of red radiation is reduced to virtually zero (all subjects looks colorless - gray) .

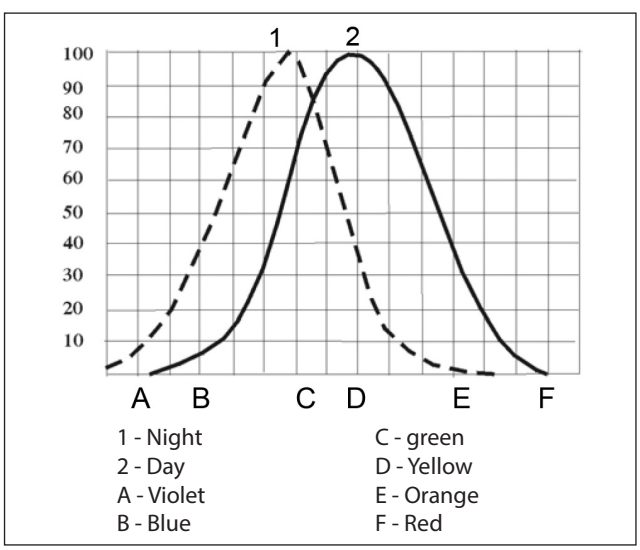


Fig. 4.6.2 Curves of relative visibility depending on the color

Tab. 4.6.3. Depending minimum distinguishable contrast of lighting

Lighting *lx*	500	100	50	20	10	5	2	1	0,5
Maximum distinct contrast	4,5	6,0	6,5	8,5	11	14,5	20	25	30

The main distinctions of colour from another is limited to distinctions in tone, saturation and brightness and colour contrast.

The colour tone is determined by so-called selective reflection of light from the object, i.e. its ability to selectively reflects and absorbs falling on it light beams with different wavelengths. Selective reflection of light from the objects of a different colour is measured in percentage and is expressed by coefficient of reflectance (Δ) - **Table. 4.6.4.**

Saturation is the degree of visibility of the colour tone (the degree of dash of white to a colour). Recommended use of colours with a considerable degree of saturation, thus improving the perception of the visual object. The colour contrast is the change in colour perception depending on the simultaneous secondary coloured irritants, that exacerbate in

him (the colour perception) its quality, or diminish their quality in a given sense (this is so called the phenomenon of contrast).

The lack of colour contrast or brightness in the colour of the manufacturing premises, machinery equipment causes difficulty in distinguishing the contours of them, resulting in visual apparatus intense and tired quickly. Strong contrasts too tired eyes and therefore must be avoided.

In this connection, it is recommended that the background color of the mashinery, the walls, the floor to choose depending on the colour of worked pieces.

So in processing gray details, as iron, steel, lead, aluminum and other preparations, recommended background is in yellow-cream colour. When processing workpieces with white (paper, etc.) the background is blue. Treat yellow details, such as bronze, copper, brass ware, wood, yellow plastic, recommended background to be blue sky saturated colour, in contrast, if treated with details blue, background should be yellow or beige. When processing red, brown or black details, at - suitable brick red or beige background.

The colour design of the work environment affects the psyche. For example:

a) red, yellow, orange colour and their shades reminiscent of the colour of the heated metal, sun and are associated in the minds of human warmth; this is called warm colours. These are dynamic colours that pulsed action (increasing productivity) and improve spirits. Red increases in respiratory rate, muscle tension and blood pressure and pulse quickens. Yellow stimulates the eye and brain, causing nice and cheerful spirits, calms the nervous system. Orange improves digestion, creates a warm feeling and a stimulating effect. In most cases, irritating and signalling.

Tab. 4.6.4. Approximately reflection on the surfaces of a different color

Colours and surfaces	Δ	Colours and surfaces	Δ
Purple-light	0,65	Beige (oil-based)	0,78
Ultramarine	0,08	White (clay)	0,76
Sky blue	0,66	Light Grey	0,44
Blue	0,55	Gray	0,30
Dark blue	0,11	Silver gray	0,35
Light green	0,48	Stone grey	0,48
Green	0,28	Zinc gray	0,12
Dark green	0,13	Zink	0,55
Yellow green	0,39	Dark gray	0,14
Light yellow green	0,68	Gray black	0,12
Light yellow	0,65	Black	0,10
Dark yellow	0,44	Black (oil-based)	0,06
Light orange	0,48	Black (matte)	0,04
Dark orange	0,35	Ivory	0,70
Orange	0,23	Cream	0,70
Brown	0,43	Light oak	0,33
Dark brown	0,32	Dark oak	0,18
Light pink	0,60	Grey dry concrete	0,32
Pink	0,47	Yellow brick	0,32
Light red	0,32	Red brick	0,25
Red	0,25	Asphalt	0,20
Tight red	0,13	Stucco	0,50
Dark red	0,09	Gypsum plaster	0,77
Chartreuse green	0,54	Lime plaster	0,51
White (zinc weis)	0,83	Pine boards	0,39
Paneling (natural plywood)	0,42	Human skin-average	0,63

b) green, blue, blue-green colour and its shades reminiscent expanse of sea, the colour of the sky and are called cool colours. Calming effect and promote a lasting increase its productivity of labour. They increase the spatial idea. The green colour is soothing, fresh and a sense of humidity. Act relaxed. The blue colour is light, reduces muscle tension, slows heart rate and respiration, lowers blood pressure.

c) light colours create a sense of lightness and joy. Increased lighting, incite to maintain cleanliness;

d) dark colours work hard, depressing, reduce the reflection of light;

e) saturated and bright colours pulsed sensitivity, spirits and enliven the workplace;

f) low saturated colours calm.

Some colours can create and taste sensations. For example, the green colour creates a sense of salty flavor, of sour yellow, blue for bitter.

Colours influence to create work habits when used with the same purpose - to begin and complete the work, danger, etc.

Colour vision is an essential condition for the exercise of many professions in transport, military, photography, television, chemical, textile, leather, printing industry.

4.6.3. OPTIMIZATION OF VISUAL ACTIVITY AT WORK.

It is implemented using ergonomic and labour - medical measures.

Increasing the level of lighting. The importance of good lighting is evident from the following examples:

A. Copy of the same typewritten text is performed depending on the level of lighting (other things being equal - experience typist, workplace organization, etc.), as follows:

Level of lighting (lx)	Time for retype on 1 line, (s)
40	16
100	14
300	13

B. Turning a cover for petrol pump in mechanical engineering:

Lighting level (lx) - average	Time processing 1 piece (min)	Increasing productivity in %
52	108	–
136	95	14
351	90	20

Level the least lighting is determined by several parameters, normalized in BS 1786-84 - **Table. 4.6.5.**

1. Contrast (k) is high at over 0.5; average - in k 0,5 to 0,2; low for k to 0.2.

2. The background is bright in diffuse reflection factor of the surface (Δ) over 0.4; average - 0.4 to 0.2 and dark in 0.2.

3. Assumed the realized value of lighting differ from normal plus 15% and minus 5%.

When necessary lighting to over 500 lx, is recommended to organize local artificial lighting. In these cases it is necessary to keep a balance between local (El.) and total (Ec.) lighting - the latter to provide lighting of not less than 10% of the combined.

Lighting is typically measured in horizontal (E x.) plan, but in a number of occupations is necessary to take into account the vertical (E v.) lighting. It is believed that the ratio E x.: Ev = 4: 1 ensures the best hygienic conditions for visual comfort. The lighting of the workplace already comply with the age factor. A number of authors believe that reading printed text by 60 year old individual it needs 15 times more lighting compared to 10-20 years and about 10 times more lighting compared to the 20-30 year old individual.

Visual comfort. It includes anti-brightness, anti-sharp contrasts, creating a uniform lighting in the visual field and others.

1. Remove the brightness and stroboscopic effect. When relatively bright light source or its reflection appeared in the visual field, creating brilliance decreases sharply visibility. Direct luster due to the presence of direct light source and reflected (mirror) shine - the presence of highly reflective surfaces in the field of vision **(Fig. 4.6.3)**.

Direct shine may be reduced as follows:

- By not dispose bright light sources within 60 ° from the centre of the visual field (or above 30 ° from the horizontal plan) - **Fig. 4.6.4.**;

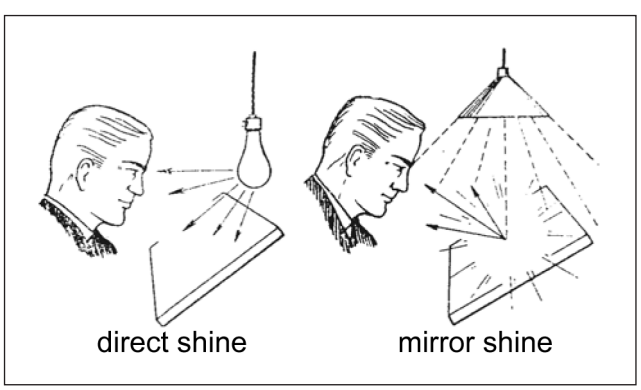


Fig. 4.6.3. Direct and reflected shine

- Using light-protetive appliance, suitable for specific workplace;

- Using the reflected light;

- By using several light sources with less power instead a more powerful.

The reflected shine may be reduced as follows:

- Using diffused light;

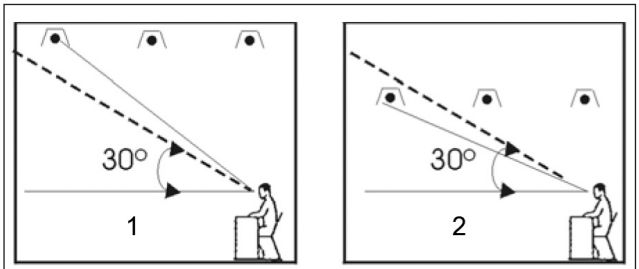
- By applying a matte, instead polished surfaces of avoiding the mirror effect - **Fig. 4.6.5.**;

- Through the deployment of the light sources so that the viewing angle of the working surface does not coincide with the angle of incidence on it of light rays - **Fig. 4.6.6.**

Stroboscopic effects due to the pulsation of luminous flux (these are for discharge lamps) is removed through proper networking of luminaires.

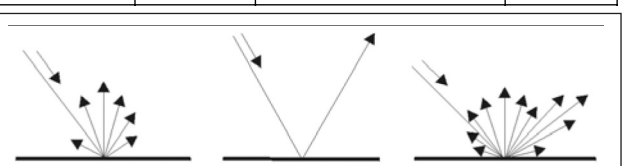
Tab. 4.6.5. Norms for the least light (lx)

Character of visual work	Size object to distinguish, mm	Category visual work	Contrast (k) object/ background 1	Characte-ristics of the backgr. 2	Lighting *(lx)* Combined lighting 3	 General lighting 4
Extremely high accuracy	To 0,15	I a I b I c I d	small small average small average great average great great	dark average dark bright average dark bright bright average	5000 4000 2500 1500	1500 1250 750 400
Very high accuracy	from 0,16 to 0,30	II a II b II c II d	As I a As I b As I c		4000 3000 2000 1000	1250 750 500 300
High accuracy	from 0,31 to 0,50	III a III b III c III d	As I a As I b As I c As I d		2000 1000 750 400	500 300 300 200
Average accuracy	from 0,51 to 1,00	IV a IV b IV c IV d	As I a As I b As I c As I d		750 500 400 300	300 200 200 150
Small accuracy	from 1,01 to 5,00	V a V b V c V d	As I a As I b As I c As I d		300 200 – –	200 150 150 100
Rough working	Over 5.00	VI	Regardless of contrast and background		–	150
Working with self-luminous materials and products in hot workshops	Over 5.00	VII	Also		–	200
General observation of the process - permanently		VIII a	Also			75
Periodically, at a constant presence of people in the room	–	VIII b	Also			50
Periodically during a temporary stay of people in the room		VIII c	Also			30



1. - The eye is not affected by light sources located above 30 ° from the horizontal plan
2. - The eye is not influenced by the light source located at an angle of less than 30 °, but provided with a light-protective appliance

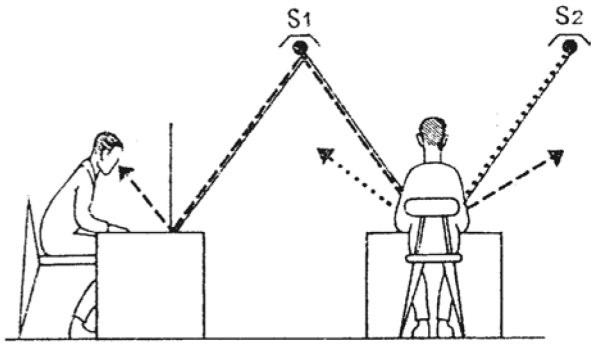
Fig. 4.6.4. Reduced shine through appropriate positioning of the light source to the horizontal plan



1. Matte surface - diffuse reflection
2. Polished surface - mirror reflection
3. Semi-gloss surface - mixed reflection

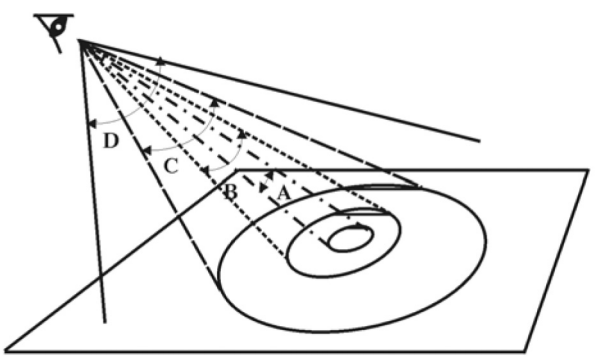
Fig. 4.6.5. Reflection from various surfaces

2. Providing uniform illumination in the field. To achieve such a desirable ratio of brightness between the four areas of the visual field is as follows: A: B = 3; A: C = 10; D = 100 - **Fig. 4.6.7.** Light, bright surfaces is desirable to have in the center and dark at the periphery of the visual field. Avoid sequential appearance of bright and dark surfaces, mobile and



1. Reflected by light source S_1, beam coincide at view direction (bad performance)
2. Reflected beams of light sources S_1 and S_2 no reach eye (good performance)

Fig. 4.6.6. Observed object and reflected light



A - object; B - central zone;
C - peripheral field ≈ 30°; D - total visual field

Fig. 4.6.7. Lighting - areas of the visual field (by H. Desoile)

with different shine objectives in the field, and light from a light source with a pronounced pulse flux. Breaking evenly lighting causes relatively dazzle the eye, causing discomfort, rapid visual fatigue and should be avoided.

3. Impact of surrounding objects. It also affects brightness and depends on the coefficient of diffuse reflection - Δ **(Table. 4.6.4)**, respectively the correct coloring of various surfaces in operating rooms. It is recommended to be as follows: for floors of 0.1 to 0.5; for work surfaces - 0.2-0.6; walling - 0.3-0.8; ceiling - 0.6-0.9.

4. The viewing comfort improves if you take into account the **types of artificial lighting** and its impact on the perception of color.

4.6.4. LIGHTING IN THE WORKPLACE

Generally modern architectural trends for more extensive use of natural lighting, in combination with the various sources of artificial light (ie setting up of so-called mixed lighting), comply with the guidelines in hygiene standards to increase the level of lighting and creating visual comfort. This leads also to increased levels of productivity and reflected in the reduction of overall morbidity, accidents and absenteeism.

Natural lighting based on external lighting at least 5000 lx and depends on: the time of day, season, method of implementation and others. It is estimated by the coefficient of natural lighting (CNL) percentage formula:

$$CNL\% = Evi / Ee \times 100$$

where

E i. = Brightness of the interior lighting, lx;

E e. = Brightness of external light, lx.

Hygiene requirements of CNL depending the professional activity are different **(Table. 4.6.6)**

Side lighting is more efficient at higher than at wide windows (with the same glazed area).

Overhead lighting has three varieties - **Fig. 4.6.8.**

Shedovoto lighting is oriented north to avoid the massive flow of infrared radiation and fonari - east-west. To ensure visual comfort (mainly in terms

Tab. 4.6.6. Hygiene requirements to CNL

Category of visual work	Coefficient of natural lighting	
	In rooflights CNL average, %	In side lighting CNL min, %
I	10	3,5
II	7	2,5
III	5	2,0
IV	4	1,5
V	3	1,0
VI	2	0,5
VII	3	1,0
VIII		
a	1	0,3
b	0,7	0,2
c	0,5	0,1

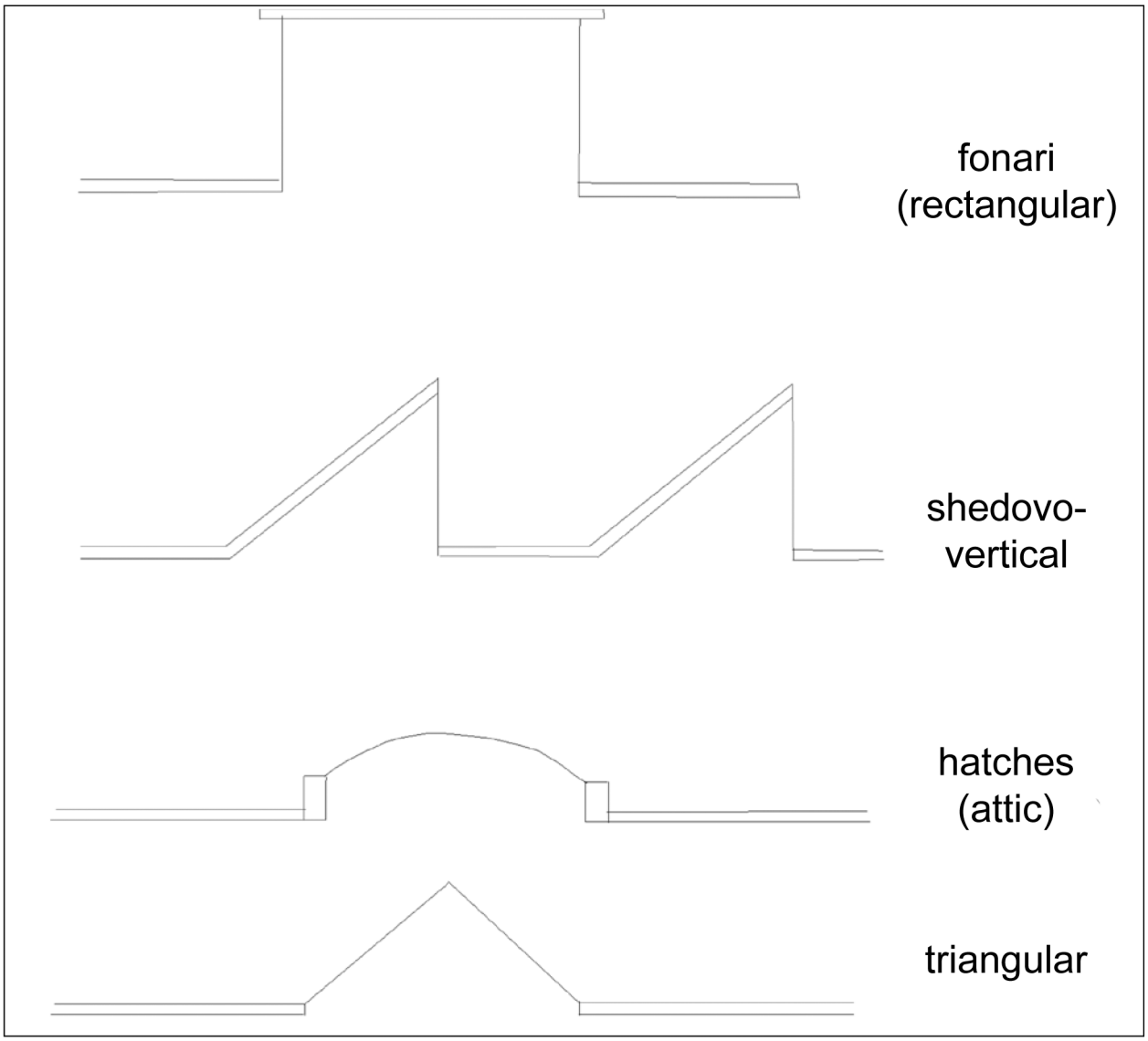


Fig. 4.6.8. Type rooflights

of brightness) visible part of the sky should not be located in the visual field. Anti-glow of infrared radiation is appropriate to use blinds - best vertical lift. Index glazing (with a view to reducing solar heat radiation and improving the microclimate) is limited to 20-30%, which requires setting up of artificial lighting to achieve the necessary illumination (especially during transitional seasons, in dim days and at different times per day).

Artificial lighting. The light sources are divided into three categories depending on the method of lighting work zone - direct, reflected and diffused light.

In direct lighting 90-100% of the light is directed downward. Easy to concentrate on the field. A major disadvantage: high brightness contrasts, shadows, reflections. Lighting reflected light (when 90-100% of the light is directed toward the ceiling and upper parts of the walls) provides uniform lighting without shadows and glare. It creates the best visual comfort and is suitable for low rooms with bright walls. Disadvantages: sometimes distorted perception of depth (relief) of the image; it is not economical.

Diffused light requires less energy consumption compared with the reflected, but sometimes creates shadows and glare (for lighting by discharge lamps - if fit with light-protectors, almost completely liquidated glare).

Artificial lighting in production use several basic types of lighting. Those with heated lamp (light bulb) are the oldest tool used now, although according to BS 1786-84 is recommended discharge lamps - fluorescent lights, mercury and sodium lamps (mercury lamps can be combined with such a heated wire, or be a luminescent layer which corrected spectrum thereof). Heated filament lamps with a continuous

spectrum and contains all colors, but they dominate the yellow and especially red. Heated filament lamps are suitable for colored in warm tones rooms. Since they are still used most widely, it should be added that in light of heated lamps light levels given in the **Table. 4.6.5** should be reduced as follows: a) one degree for a system of combined lighting in production and auxiliary premises of industrial enterprises when normalized lighting is equal to or more than 750 lx; lighting in public and residential buildings, when normalized lighting it is equal to or more than 300 lx; b) one degree on the scale of the lighting for system for general lighting categories I to V and VII (but not more than 300 lx); c) two degrees on the scale of lighting for system for general lighting category VI and VIII and in public and residential buildings, with the exception of the cases described in a).

Fluorescent lamps are several types according to the spectrum of light emitted from them. Lights "daylight" are best suited for artificial lighting, because at least change available color (as spectrum closest to them - close to that of daylight).

When lighting using fluorescent lamps type "white hot" to color the rooms are recommended warm colors, and illuminated with fluorescent lamps, illuminated with blue light - cool colors.

Mercury lamps when they are equipped with luminescent layer for color adjustment (emitted light beams) are very suitable for the production aesthetics. In their use should take into account certain hygiene requirements, the most important of which is that it can only be used in large rooms with high ceilings of 6-7 m from the floor. With sodium vapor lamps have very good performance, large luminous flux and suitable for vision no fatigue yellow color.

The new Bulgarian standard for lighting work places - BS EN 12464-1: 2006 (identical with the European EN 12464-1: 2002) indicates values of **lighting, discomfort from glare and color rendering**, guaranteeing good visual performance and visual comfort, associated with safety and health of workers.

The values for the **lighting** of the work surface in different types of activities may increase or decrease by one step (respectively eg. high accuracy and productivity, many small details, low contrast, very long period of time, or vice versa - a very large size of details, high contrast, very short period of time) in the recommended scale of lighting - 20-30-50-75-100-150-200-300-500-750-1000-2000-3000-5000 lx. Maintained lighting on surfaces with continuous work should not be less than 200 lx. In certain specific activities required lighting level can be very high - eg. in health facilities of the operating field in operating rooms - 10 000 to 100 000 lx; dental offices - illumination of the oral cavity - over 5000 lx, tables autopsy and dissection - over 5,000 lx. The lighting of the immediate surrounding areas shall not be less than one step (on a scale of lighting) than the work surface (range 200-750 lx on the working surface). The extent of light on the work surface must be ≥ 0.7, and the immediate surrounding areas ≥ 0.5.

The degree of **brightness discomfort** determine by a logarithmic index UGR (*Unifed Glade Rating* - aggregated level of glare). Its value depends on the brightness of the background in *cd x* m^{-2}; the brightness of each luminaire in the direction of the eye of the worker - *cd x* m^{-2}; solid angle (in steradians - *sr*), under which are visible luminous parts of the luminaire; positional index Guth, associated with the location of illuminant bodies to line of sight. The values of UGR_L - usually in the range of 19-25, should not be exceeded. Designated UGR_L presented in project documentation.

Color or so-called "Light color" of the light source (lamp) is reproduced color of the light emitted. Lamps with correlated color temperature (T_m) below 3300 K (Kelvin) is a warm light color; from 3300 to 5300 K - neutral, and over 5,300 K - cold. Light sources (lamps) are evaluated with the so-called **color rendering index *Ra*** of up to 100. In workplaces used lamps index Ra ≥ 80. Lower values are possible for example in passable tunnels, high halls, etc., but there is a safety color must be well recognized. Required values (minimum) for lighting (E_m), UGR_L and Ra are presented for a variety of areas (surfaces), tasks and activities, grouped into eight major groups (tables) - for example. **Table 5.7. - Health institutions**.

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Z. Ivanov

5 BIOLOGICAL RHYTHM AND REGIMEN OF WORK AND REST

The rhythm (from the Latin transferred from the Greek "rhytmus") is a universal biological phenomenon that can be expressed with quantitative and qualitative indicators and covers all forms of living matter - of subcellular structures and the cell to complex behaviours of individuals and even populations and ecosystems. The basis of biological rhythms lies rotation of fluctuate changes in matter and its functions that are inherent in every rhythm three major components: frequency fluctuations, size (amplitude) and phase. **Biorhythmology** is a relatively new science, often identified with **hronobiology**, but it is assumed that the general problem of biorhythmology include: **hronobiology, hronopathology, hronomedicine, chronotherapy, hronopharmacology etc**. The complexity of the shape of the curve biological rhythms define them as simple (sinusoidal or cosinusoidal) and complex. In humans, by virtue of biological requirement to keep the existence adapting to environmental conditions, rhythms of a number of physiological functions have been "copied" from those of the environment itself. In this respect especially wide popularity has rhythm "day - night" (light - dark) or so-called circadian rhythms (from the Latin. "circa" - and about "dies" - day). There are also weekly, seasonal, annual and multiannual (with periods of 11 and 22 years) rhythms, but they have much less impact on human performance.

Circadian rhythm has a strong impact on employment and its rhythm, which in turn can be expressed with the two-phase process: action (labour) - break (pause). The operation determines the occurrence of a number of changes in the whole organism (mostly in the locomotorium, the cardiovascular, respiratory and nervous system and brain). The pause is preparing for the next work phase and serves to restore biochemical balance and lost neuro-psychological and muscle power in the labour process. It should be explicit that rhythm "work - rest " is almost perfect physiological mechanism built into human evolution, whose main task is counteracting fatigue and wear on the body. In modern labour organization that rhythm underpins the scientific modes of work and rest, whose deeper meaning is to achieve adaptive balance between state of action and break, thereby not only counteract fatigue, but also supports high sustainable performance.

Rhythm "work - rest" has the following general characteristics: a) it is an obligate condition for any kind of labour; b) arises and is determined by the course of the work from the beginning to its end; c) has specific characteristics depending on the type of work intension and stress of labour conditions and labour organization.

At the beginning of the worker productivity is relatively low. In the course of the work process efficiency gradually grow. That its ascendant curve associated with the gradual inclusion of the overall physiological functions and depending on the specific type of work is a process vrabotvane. Reaching a certain level in work performance occurs stabilization reflected in uniform or with small fluctuations productivity. In the rhythm of work this is known as "permanent plateau of sustainable performance." After a time starts drop in performance and appear signs of fatigue. If it is substantial, it may necessitate termination of the work. The insertion of a longer break (usually lunch - at day shift) caused repeated increase in productivity, which again belongs to closing work time.

Thus, the rhythm of working day consists of at least two waves, and depending on the type of work (including the loadness and the intension of labour) and more pauses may occur several successive waves of varying amplitude. This is the typical work rhythm for day shift, which usually coincides with the circadian rhythm of performance, driven determine by its lower level in the afternoon - indicate that performance between 14 and 16 hours is about 20-30% more lower compared to the morning hours.

Rhythm of work achievenments (respectively performance) has as weekly, as seasonal aspect - the lower the productivity on Monday and during the hot summer months, and more in Tuesday, Wednes-

day and Thursday, as well as in winter, although that mental performance not seen such a distinct rhythm.

Interestingly, although many debatable biorhythmological problem is the **so-called macrobiorhythms - physical, emotional and intellectual cycle**s and their importance for employment. All these three cycles provides a common rhythm with maxima and minima every 4-5 months, which continues throughout the life of man.

The physical cycle - also called male, lasts 23 days. It determines the energy, the strength, endurance, coordination of motor manifestations of the individual. His first half (the uplift) lasts 11 days and is characterized by increased activity, especially between the 2nd and 9th day. In the second half thereof, the decrement of those functions.

The emotional cycle, also called female, lasts about 28 days. In his first half - to 13-14 th his day, a man is cheerful, optimistic, aggressive mood. In the second half made a critical reassessment of the results, there is increased irritability.

The intellectual cycle lasts 33 days. In his first 17 days mental functions in humans are on the rise, memory is very accurate, clear logic; after 17 days was observed impaired memory and noticeably decrease the intensity of the brain function.

It was found that the combination of the three macrobiorhythms defines so-called critical days and periods in the life of every person (even in 1780 on 27 May Goethe wrote in his diary: "Watching cycle of good and bad days that are repeated in a specific order ... "). In those days the resistance of human and there performance decreased, attention and memory are weakened, the thought is rigid. Then possibly arise accidents, to do our work worse than usual. So for people engaged in certain activities (especially in military affairs, transportation, aviation, etc.) has recently made calculations to establish these individual cycles in order to "critical days" to work with more attention, with more caution . Described the most common features of labour rhythm on the impact of the biological clock gain in any particular work its specifics. When conveyor forms of work organization, for example, there is the so-called imposed work rhythm with great repeatability of partial and small operations. This requires adjustment of personal biorhythm of workers to the course of the conveyor. Coincidence of both rhythm flow of work processes can be satisfactory and productive, but significant deviations occur negative phenomena of the nervous system throughout the body - monotony, manifested most commonly as an early onset of fatigue. Significant disruption of normal physiological rhythms is very characteristic at shift work and night work. Night work is reversing the course of circadian physiological rhythms in which the phase of rest and sleep are replaced by active intension of work phase. Because of the rough violation of encoded in human evolution habitual rhythm (especially that of the nervous system, autonomic functions) night work is more tiring and lower productivity. Even long - weeks, months, night work is not able to completely alter the course of circadian physiological rhythms. All this requires the creation of specific science-based regimens for work and rest in these types of work. **Intershift modes of work and rest**, which include rest within a regulated working hours, i.e. these rests are regulated, become part of the real (for manufacturing activities) time (unfortunately neither our nor in the European labour laws rests are not included in real work time, except for meal breaks in continuous production - European legislation, however, identifies the need statutory rests be subject to negotiation between employers and employees).

First question is when to introduce statutory rests. It is believed that they are effective in angagement of the worker over 75-80% of their working time in direct production activities, ie when operational (real) time is over 75-80%. In this case, it is considered generally that rests should be not less than 15% of operating time, while at many work activities they have to be bigger - 20-30%, and very hard work, especially in conditions of excessive heat production microclimate they can reach more than 50% of the effective time.

The determination of the total time for vacations is specifically for different types of work. It is realize by assessing the type and extent of factors influenced on labour and/or by evaluation of changes in labour efficiency in production, physiological and psychophysical indices. The minimum total time for statutory breaks during the work shift is 10 minutes for men and 15 minutes for women.

The number and length of breaks is determined by the nature and severity of occupational activities and changes in performance over the course of the day, they should be given just before the onset of symptoms of fatigue.

The minimum duration of each break is five minutes, the maximum - 20 minutes. Longer breaks distort the rhythm of work and require re period of the inworking to enter the economy (sustainable) operation regimen. It is therefore recommended first break to give 120-150 minutes after the start of the working day and break in the afternoon, 60-90 minutes after lunch (for light work in day shift).

It is desirable that 30-40% of the rest period is in the first half, and 60-70% - in the second half of the

work day. At an mean rate dynamic exercise break is given after 60-90 min, in heavy physical work - after 30-60 min, and at very heavy physical work - after 30-45 min job. In activities with loading of small muscle groups and expressed monotony are effective the short and frequent breaks.

In evening and night shifts introduce additional rests. Morning shift is not more than 6 h, and the night starts at 22 h. Not allowed more than 4-5 consecutive night shifts and rest between two successive shifts is not less than 12 h.

Extended shifts - 12 h, they are allowed only when working with a medium level of physical and mental intension and admissible levels of work environment factors and employment. In the extended shifts are introduced at least two breaks with a duration of 30 min each.

Statutory rests by type are: active, semi-passive and passive.

Active rests include physical exercise, kalanetics, aerobics and more. They apply to professional activities with hypokinesi, mild to moderate exercise, work in forced sitting posture and / or moderate neuro-psychological intension.

Semi-passive rests include walking, self-massage, light sports games, etc. Apply when operating at an average rate, sitting posture, increased visual load, average level of physical and mental intension, in workers over the '35 and under unfavorable sanitary conditions.

Passive rests are carried out in a sitting, lying or semi- lying postures and may include reading newspapers, listening to music, sessions of psychological unloading, etc. They are suitable for activities with: high physical exertion; significant mental stress and mental fatigue; high pace of work; significantly routing load; forced and standing working postures; unfavorable sanitary conditions.

It is best regulated rests take place outside the production environment, in specially designed for this purpose recreation recesses and recreational centres.

On **Table 5.1.** are given schemes - examples for allocation of breaks depending on the nature of work.

In practical development of inter-change regimens of work and rest are evaluated all the factors of the working environment and workflow in accordance with the requirements of the country Ordinance on a comprehensive assessment of working conditions - 1991 Additional evaluate ergonomic compliance of control means, means for presenting information, production equipment, and also working posture and working areas. On this basis it is estimated whole load of workers in labour and calculate the time needed to restore capacity. Then develops a specific regimen of work and rest, and determine the duration and distribution of breaks within the working shift - **Fig. 5.1.** It is represented graphically scientific and reasonable inter-shift mode of work and rest for the profession of ironing presses in the garment industry - first shift. The impact of all factors of the working environment and working process, whose parameters are outside of existing regulations - overheating microclimate unfavorable working position (standing), physical load, ergonomic inadequacy of equipment, control devices and display devices to human - operator opportunities.

Tab. 5.1. Example schemes to determine the duration and distribution of breaks according to the nature of work

1	2	3
1. Light exercise and minor mental intension (eg. work in offices, semi-automatic lines in machine-building).	Short and rare breaks	2 breaks 5-10 min: Ist - 130 min after the start of the working day; II nd - 90 min after the pause for meal
2. Moderate physical and mild mental intension (eg. work turners, fitters, miller, painters, etc.).	Average duration and rare breaks	2 breaks 10 min: Ist - 110 min, after the beginning of the working day; II nd - 80 min after the break for a meal.
3. Heavy physical loading (eg. Core activities in heavy engineering, metallurgy, construction, etc.).	Frequent and long holidays	20-25 minutes every hour.
4. Minor physical loading and pronounced psychological intension (work of accountants, cashiers, operators TPS and NPS, etc.).	Medium-length and frequency breaks	2 rests: Ist 120-130 min after the start of the working day of 10 min; II- nd 100 min after the timeout for feeding, of 15 min.
5. Work with great monotony, visual and motor intension (operation pipeline, in cigarette production, installation of small articles, in engineering, etc.).	Frequent and short breaks	5 min each hour or 10 min for two hours.

Characteristic of the regimen of work and rest:

1. Total duration shift time - 510 min.
2. Total duration of the statutory rests - 54 min.
3. Distribution of breaks during shifts:

I-st break from 7.50 to 8.10 - for rest and meal
II-nd break from 9.20 to 9.25 - for recreation
III-th break from 10.35 to 10.40 - for recreation
IV-th break from 11.50 to 12.08 - for rest and meal
V-th break from 13.18 to 13.24 - for recreation

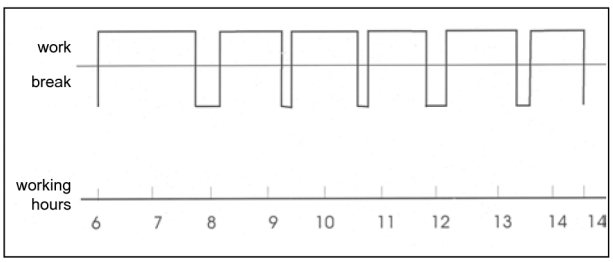

Fig. 5.1. Inter-shift regimen of work and rest for ironing presses - I-st shift

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6 PHYSICAL FACTORS

Ft. Kalburova, D. Tsvetkov

6.1 THERMAL CONDITIONS OF ENVIRONMENT (MICROCLIMATE)

In its daily household and work activity the human body is exposed of different weather environment outdoors or indoors - residential, public, industrial, school and others. Productive human activities occur or outdoors - agriculture, construction, forestry, outdoor ore and non-metallic minerals, etc., or more often indoors - engineering, metallurgy, chemistry and petrochemistry, pharmaceutical industry, food and others (in some of the industries operating procedures can run, both indoors and outdoors).

The thermal conditions (microclimate) include a set of physical environmental factors that have an impact mainly on the thermal condition of the body. These are - air temperature, humidity and speed of air movement, and heat, by infrared radiation, participating in heat exchange of human body, and determine the thermal environment of a space outdoors or indoors.

The production microclimate has a number of features: considerable intensity of its individual components, combined with other adverse factors in the workplace - the physico-chemical, chemical, biological and by labour process - the intension and stress of work, regimen of labour and so on. That can aggravate its impact on self-esteem, employability and health of influenced.

The thermal state of man is a function of factors with thermal and non thermal nature **(Fig. 1)**. Thermal factors include air temperature, humidity, speed of air movement, radiation heat, non thermal include by one side the characteristics of the human body (surface, size, physical activity, skin colour), on the other - clothing (thermoresistance, air and vapour permeability, colour).

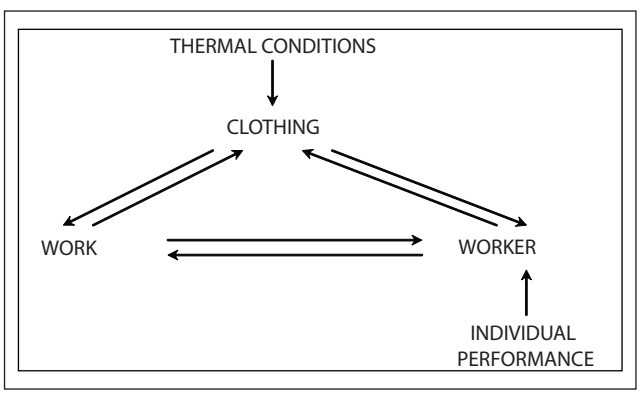

Fig. 1

6.1.1. THERMAL FACTORS

1. Air temperature

Air temperature is a result of heating the air from various sources in the environment: hot manufactoring materials, metallurgical furnaces, molten metals and glass: the work of a number of mechanisms of technological equipment: workers carrying out intense physical labour and others. The air temperature in these conditions can exceed 35 ° C, in which the heat in the environment for an hour to surpass 84 kJ/m^{3}h are so-called "hot" production conditions. "Hot" productions are also chemical, production of rubber, deep mines, ceramic production.

For industries where temperature and humidity are a prerequisite for quality of production is formed so-called technological microclimate - example textile industry, microelectronics.

When outdoor air temperature and other thermal factors resulting from climate of the region and can range from very low (below zero or subnormal values) to excess, overheating. Such nature air temperature has on jobs in the food industry, restaurants (kitchens), fishing, agriculture, bakeries, laundries, at sporting events, military training and others.

2. Humidity

Humidity is the amount of water vapor contained in the air or pressure of water vapor in the air. It is in a very direct correlation with the air temperature. In terms of the combination of the high temperature and high humidity, when the heat through evaporation of sweat is the only effective mechanism for keeping the heat balance is established states of "overheat" the body. A more accurate indicator of

humidity in this regard is so-called relative humidity or deficit of saturation.

High levels of relative humidity over 65-70% than in the open, where it can reach and 100%, are typical for a number of industries indoors - mines, paint shops, laundries, textile industry, fur industry and others.

3. Air movement

Movement of the air depends on the air flows from production equipment (operation of electric machines, mechanisms, etc.) and the influence of external air currents. The increased speed and air movement raises the possibility of the human organism to release heat through convection and evaporation. When so-called "Hot" industries air is considered "almost stationary", which trouble this type of heat exchange. When working outdoors are essential characteristics of prevailing air currents on the geographical area and who may be at high speeds.

4. Heat (infrared) radiation.

Heat radiation is electromagnetic radiation of a wavelength of 760 nm to 1 mm, divided into three ranges: A - short-rays with a wavelength of 760 to 1400 nm: C - average between 1400 nm and 3 μm and C - long between 3 μm and 1 mm. A source of infrared radiation is any heated body. Thermal radiation is governed by physical laws; depends only on the state of the emitting body, not by the environment (the law of Kirchhoff) and is proportional to the fourth degree of the absolute (K) its temperature ($E = T^4 W/m^2$ - law of Stefan-Boltzmann): ratio between the maximum wavelength of emission and absolute temperature is constant

$$\text{(law of Vin-C} = \frac{m\lambda}{T})$$

It is obvious that even with a small increase in temperature of the body, heat-transfer through radiation increases significantly. By the temperature of the source can determine the wavelength of maximum emission - eg. for bodies heated to a temperature of 400-500 ° C are typical longwave radiation, in molten metal - temperature 1600 ° C - 22% of energy is at the expense of shortwave, at 2700 ° C -43% are short-wave rays.

Outdoors major source of infrared radiation is the sun and human exposure is significant. In conditions of indoor production sources are numerous - from ordinary light bulbs and torches to electric furnaces, molten metal and glass. Typical industrial processes with infrared radiation are bread and production of pasta (ovens), ceramic, porcelain industry, installing protective coatings, adhesives and paints, drying textiles, paper, leather, meat, vegetables, welding, machine building (foundry), metallurgy.

To what extent thermal radiation will change the thermal condition of the bodies from the environment depends on the capabilities of intrusion or takeover of the emitted energy. It is known that most oxidized and dark colored surfaces absorb the emission from 750 to 9000 nm. Short-wave (1400 nm) penetrates the tissue to a few cm, and that of a wavelength of 1 to 8 μm is absorbed by the surface skin layer.

Upon passage of infrared radiation through the air she did not warm. Radiation heat exchange takes place between bodies at different temperatures, and depending on the temperature gradient is determined and the direction of flow of heat - from the environment to the human body or vice versa (i.e., heating or cooling of the body).

6.1.2. THERMAL CONDITION OF THE BODY

1. Heat balance - determinants

Thermal balance of the human body is a function of a number of both thermal and no thermal characteristics of the environment and the organism itself. Thermal environmental conditions are the components of the microclimate (air temperature, humidity, speed of air movement, thermal radiation), and no thermal - hardness and intension of labour, characteristics of the clothing, regime of work and rest, food and drinking regime, physical , physiological and physical-physiological mechanisms of heat transfer (conduction, convection, radiation, hemodynamics, sweating, metabolism).

The heat balance can generally be expressed in the equation:

$$\pm S\,(Q) \pm W = E \pm R \pm C \pm K + M,$$

where S or Q is accumulated in the body heat: W - the energy required to overcome the external physical resistance (useful work) M-level of heat production (metabolic heat), E-heat loss through evaporation of diffuse moisture (perspiration) from the skin and through more intense sweating, R-radiation heat transfer, C-convection heat transfer, K-heat transfer by conduction (all indicators expressed in W or W/m^2 body surface area). The equation is not included heating losses through evaporation of moisture from the upper respiratory tract and heating the inhaled air, but it is of little value, as well as the heat by conduction (under certain conditions and for useful work)*, whereby the equation takes the form of a more practical matter:

$$\pm S \text{ or } Q = E \pm R \pm C + M$$

* In some conditions however, eg. work outdoors in conditions of cold and polar climates or underwater work they have very high values.

2. Physiological response of the body to thermal effects.

Thermal homeostasis is maintained by thermoregulatory system of the body, which consists of a number of elements with interrelated functions and their work is similar to the system of automatic control with negative feedback **(Fig. 2)**.

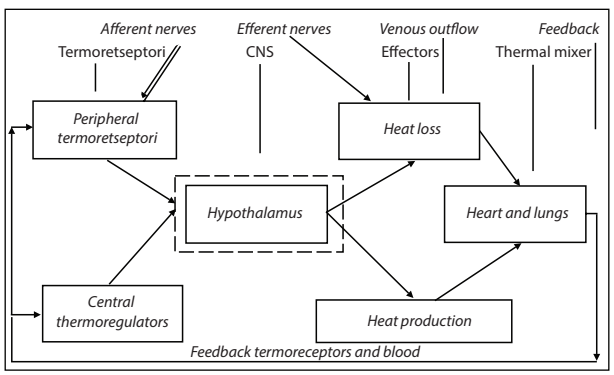

Fig. 2

Thermal information is from the surface or deep temperature receptors by afferent nerves to the center of thermoregulation within the hypothalamus then activate various mechanisms to ensure the heat production or heat. The mechanism of feedback involving the nervous system and blood flow alter the sensitivity of temperature receptors and thus the system is closed.

Temperature receptors are distributed over the entire surface of the body and in the internal organs, and the temperature sensitive nerve cells in the cerebral cortex and Auerbach plexus, hypothalamus, reticular formation, preoptic area of the brain trunk, the spinal cord.

Heat production and heat loss vary under the influence of many nerve centres.

The heat loss is regulated by the motor centres of the anterior hypothalamus, and heat production by centres in the hypothalamus back.

The centres of the anterior hypothalamus regulate the vasodilatation, sweat and respiration in response to temperature irritation of flowing blood.

There is data that the functioning of the hypothalamus important is the flow of sodium and potassium ions, possibly setting the presset temperature level.

Removal of heat from the interior of the human body (so-called "core") to the periphery or body surface area (so-called "shell") is effected by changes in vasomotor tone, which is regulated by adrenergic sympathetic nerve fibres, varying flow of blood into the periphery.

3. Thermoregulatory mechanisms of thermal equilibrium

Physical mechanisms

Heat transfer by conduction. It consists in carrying out the heat from the warmer to colder surfaces depending on the temperature gradient:

$K = hk (T_1-T_2)$, where hk is the coefficient of conduction in $Wm^{-2}. ° C^{-1}$, and T_1 and T_2 - the temperature of the contacting bodies.

Conduction is less important for heat transfer except when taken very hot or very cold foods and drinks and in proceedings requiring body contact with refrigerated or heated surfaces.

Heat through convection. When the air of the environment has a temperature lower or higher than that of the surface of the skin warmth is given or taken from the body by convection. The rate of heat transfer depends on the temperature difference and the coefficient of convection:

$C = hc (T_1-T_2)$, wherein C is the heat transfer by convection Wm^{-2}, hc - convection coefficient $Wm^{-2}. ° C^{-1}$, T_1 and T_2 respectively air temperature and the surface of the body. The main factors that affect the rate of the convection is air movement and the temperature gradient between the skin and the ambient air, for most activities it is $8.3. \sqrt{V}. W.m^{-2} C^{-1}$, wherein V is the velocity of the air in $m.s^{-1}$.

The degree of heating losses by convection depends on the difference between the temperature of the body surface and the air, and the greater is, the greater the transfer of heat. The normal skin temperature is around 31-32 ° C and if it equals the ambient air temperature, respectively equalizing the temperature of clothing to that of the ambient air by convection heat is not transferred.

Heat by radiation. Heat transfer through radiation (R) depends on numerous factors - coefficient of radiation hr, the skin temperature Tsk, radiation temperature Tr or R = hr (Tsk-Tr) wherein the variables are as above, but Tsk can refer both to skin temperature, and the surface temperature of clothing.

When the radiation energy from the longer wavelength range is absorbed by the relevant body then it is transformed into heat. Heat radiation has the same type of feed energy as visible and is independent of the presence of air and its condition - temperature, humidity, speed. The thermal radiation fall on the respective surface can be absorbed, to penetrate deeply, depending on the wavelength or to be reflect. Dark bodies (leather, clothing) absorbed radiation energy, bright and polished reflecting it to 90-95%.

Heat transfer by convection and radiation at comfortable thermal conditions or cooling is 70-88% of the total heating losses (respectively 20-30% and 40-60%).

Physiological mechanisms

Heat production (metabolism). It is necessary to maintain a constant temperature of the body can be assumed as heat tempering of the body to the environmental conditions. The maximum metabolic heat includes basal metabolisme an additional heat produced as a result of thermal environmental effects. The basal exchange correspond at so-called area of prevalence of chemical thermoregulation and forms lower and upper critical body temperature. Subject to overheating observed also two zones: a zone of secondary chemical thermoregulation and area of prevalence of physical thermoregulation. Outside these areas develop or hyperthermia and death from overheating or hypothermia and death from overcooling.

When the ambient temperature reaches a maximum value (upper critical limit), i.e. level, wherein the compensatory mechanisms for increasing the heat loss is insufficient, the body temperature began to rise. If this process continues long enough, it will die from heatstroke. The range between the upper and lower critical limits of ambient temperature is called metabolic indifferent zone.

The peculiarities of chemical thermoregulation is a taxonomic sign characterized as the systematic position of the species and its ecology. The peculiarities of chemical thermoregulation are conditioned by long evolutionary adaptation of way of life.

Cardiovascular mechanisms of heat transfer

Output of the heat from the interior (the "core") of the body to the peripheral tissues ("coat") is the first adaptive mechanism at work in conditions of overheating, wherein an increase in blood flow through the skin and mucous membranes increases 10 to 30 times. For proper functioning (adaptation) of the body during exercise and in conditions of high air temperature primary condition is good functional state of the cardiovascular system.

In terms of overheating increases blood flow to the skin and the excess heat is dissipated to the periphery. The maximum increase in blood flow can provide multiple increase of heat loss - 0.34 at 25,5 ° C of 2,58 $l.m^{-2}$. min^{-1} at 50,5 ° C. Peripheral vasodilation accompanied by vasoconstriction in other tissues and increased of the pulse to 180-200 for min., and minute volume of blood circulation increased by 50-70%.

In the event of failure of hemodynamics, physiological compensation occurs in the following sequence - an initial increase of peripheral blood flow, causing a new failure of the circulation, followed by an increased pulse rate and increased cardiac output of blood circulation, as a result of which occurs again failure of the circulation, which is offset by redistribution of the blood and release of blood depots for skin blood flow. This mechanism of compensatory response, however, has high biological value.

The initial response of the thermal impact is the release of vasoconstrictive tone synchronous by increasing blood flow to the extremities. Lower limbs supported higher vasoconstrictor tone in comparison with the above. Upon reaching a maximum inhibition of vasoconstrictor occurs secondary dilatation accompanied by sweating. The secondary dilatation is the result of the action of the sympathetic cholinergic nerve fibres, together with a vasodilating substance bradykinin and potassium.

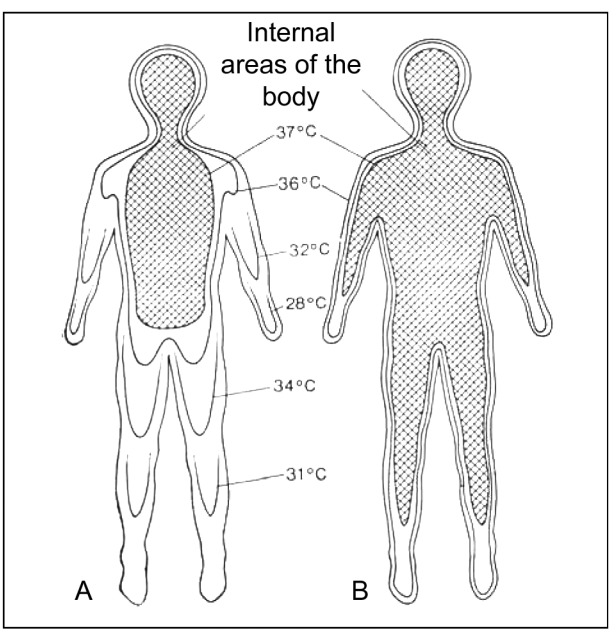

Fig. 3. The temperature of the different areas of the body at an air temperature of 20 ° C / A / C and 35 ° C / B /.

At air temperature 20 ° C, there are sharp falls in temperature between the inside area of the body and limbs **(Fig. 3A)**. At 35 ° C the temperature of the inner area and is distributed to the extremities **(Fig. 3B)**.

In response to a strong cooling the common vasomotor response reduces blood flow to the periphery by the effector vasospastic therein. The cooled venous blood is returned into the interior of the body by vessel, located near the arteries and takes most of the heat transmitted from the arterial blood.

When air temperature close to zero this system for reduced heat is not always effective, because of the intense heat exchange between arterial and venous blood, skin temperature may drop significantly - up to freeze.

Heat content changes in the body associated and with body mass. So imbalance in the heat to the environment body temperature of people with higher levels of body mass will vary less.

Physical and physiological mechanisms

Heat through evaporation of sweat. The physical nature of this mechanism is expressed by the equation E = he / Psk - Pa), where E is the heat of evaporation of sweat, W. m^{-2}, he coefficient of evaporation $Wm^{-2}.Pa^{-1}$, which depends exclusively on air movement, Psk water vapour pressure at the surface of the skin in kPa, Pa - water vapour pressure in the ambient air in kPa, ie heating losses through evaporation is determined by relative humidity and speed of air movement.

Upon evaporation of sweat 1g (surface of the body, clothing, breath) released 0.59 kcal of heat. This is a powerful mechanism for heat loss from the body under conditions of high air temperatures of the environment and normal or low relative humidity.

Evaporation of sweat become from all the body surface. In human skin has more than 2 million. sweat glands, the function of which is regulated by the sympathetic nervous system. Mediators of sweat are sympathetic cholinergic nerve fibres. Postganglion axons have the terminal branches of axon every 6 to 10 sweat glands. Sweat gland in response to pulses from the anterior hypothalamus and their number increases with the increase in body temperature in response to the rise in skin temperature above 40 ° C. At high air temperatures and physical work the amount of sweat can reach 1,7-2 l/h.

Skin cooling inhibits sweat loss even when body temperature is high, which translates into a noticeable subjective sensation to reduce fatigue and recovery of thermal comfort at work.

Water losses from the surface of the body has in the absence of a visible sense of sweating due to passive diffusion of moisture through the skin. This "invisible" sweating is about 20 to 50 $g.m^{-2}.h$ depending on the pressure of the water vapours in the environment and the values of skin temperature. In physical labour of sweat excreted in terms of thermal comfort - if not separated, the environment is perceived as "cool" **(Table. 1)**.

In comfortable thermal conditions heat loss by evaporation is about 23-27% of the total heating losses (besides about 1/3 is heating losses from evaporation from the upper respiratory tract and 2/3 by skin, clothing). With increasing values - 40% or more, the environment is perceived as "warm" and "hot".

4. Role of clothing for thermal condition of the body.

One adapts to environmental conditions through clothing and, if possible, by adjusting the temperature, humidity and velocity of the ambient air, wherein skin temperature is maintained around 33 ° C without tension on the thermoregulatory mechanisms. The role of the clothing to heat transfer from the body surface to the environment is determined by three determinants: index of air permeability, coefficient of "ventilation" in the layers of the clothing and the impedance of the clothing for sweating. Sweat between the skin and clothing depends on the nature of the clothing, water vapor pressure on the skin surface and aerial environments, air temperature and metabolic heat. Heat loss (E- in Wm^{-2}. ° C^{-1}) through sweating is defined:

$$E = \frac{P_{sk} - P_a}{(R_e, T),}$$

where P_{sk} P_a is the difference in water vapour pressure between the skin and ambient air (in kP_a), R_e, T - heat secreted thanks to clothing in arbitrary units (clo) - from 0 to 1.0.

The participation of the clothing in the heat exchange connected to the "ventilation" of the air between the layers of clothing. In different combinations of clothes and velocity of ambient air creating conditions clothing to act as a "pump" to direct air to or from clothing.

At calculation is determined the relationship between the degree of need sweating (SW req - 120 W/m^2 or 180 g/h, standard ISO-7243) as a function of the permeability of clothing for water vapor at different ambient temperatures in order to protect by condensation.

The role of the garment in the heat transfer by convection is related to its resistance. Any clothing still in the manufacturing has definitely thermoresistance - TR. TR unit is "clo" - is determined to 0,155° $C.m^{-2}.W^{-1}$ that provides thermal comfort at rest and air temperature 21 °C, 50% relative humidity and speed of air movement 0,1 m/s.

Tab. 1. Moisture separation by the human organism, g / h, at a relative rest

Warm feeling						
Very hot	**Hot**	**Warm**	**Comfortable**	**Coolness**	**Coldly**	**Very coldly**
500-2000 a significant portion of the sweat runs down	250-500 a significant portion of the sweat runs down	60-250 sweat does not flow	50±10 sweat is not released	40 sweat is not released	Moisture separation is not typical as an indicator for warm feeling	

The total thermal resistance between the skin and the air environment is the sum of that of the clothing and the ambient air and is dependent on the speed of air movement. The air permeability of clothing markedly reduces thermoresistance - so for example at fast walking can reduce by half thermoresistance to moderately thick clothing. This effect is partly due to the mechanical effect of "pump" of the moving body, which ventilates microspace between clothing and skin surface, i.e. due to the increased speed of air movement.

Clothing can absorb or reflect heat radiation. Radiation heat transfer for dressed man is between the skin and the inner surface of the clothing and between the outer surface of the clothing and the environment, but here he has a small role in heat balance than convection and evaporation of sweat.

6.1.3. INFLUENCE OF THERMAL CONDITIONS – PATHOLOGICAL DISORDERS AND DISEASES.

The thermal conditions can be described as comfortable and uncomfortable (overheating, cooling, static, etc.).

Overheat conditions caused severe (acute hyperthermia, heat stroke, cramps disease, "sunny" stroke) and chronic (heat exhaustion, heat fatigue, myocardio- and vasopathics, poor performance and poor function of the gastrointestinal tract and the urinary system, decreased resistance to other adverse environmental factors), disorders and diseases.

They cause skin damage and local - miliaria, intertrigo, heat erythema and dermatitis, burns (infrared radiation). Also ocular lesions - heat cataract (infrared radiation) and occupational diseases - early atherosclerosis (up to 40).

The cooling conditions also cause acute general (acute hypothermia - "white" death, colds and upper respiratory tract, myositis, neuralgia), acute local (frostbite) disorders and diseases and chronic (decreased performance, lowered immunity, allergic reactions, decreased resistance to other unfavorable environmental factors - ex. very often and more quickly develop vibration disease by activity of local vibrations.

A special place holds so on **static or monotonous microclimate**, characterized by minor modifications to its parameters in time. On the one hand - has a stimulating effect on termoretseptorniya apparatus resulting in no trained organism is more sensitive to changes in weather conditions outside manufacturing - more easily vulnerable to cause infectious diseases, colds, the other - being the monotonous stimulus (ex. in radio and microelectronics) leads to a reduction in capacity.

The health effects of working in a condition of overheating or cooling resulting from the influence of multiple factors - duration of exposure, type of clothing, heaviness of the work, mode of work and rest, age, gender and health status of workers.

6.1.4. HYGIENE REGULATION OF THERMAL CONDITIONS

Evaluation of thermal conditions should include all elements that are relevant to their formation: microclimate components, rated complex as act in real life, burden and intension of labour, regime of work and rest, features clothing and others. The physiological response of the organism is evaluated respectively by the state of metabolism, of the hemodynamic system (heat transfer by convection and radiation) and of sweating.

Those considerations in hygienic regulation of thermal environmental conditions are included in the international standards ISO - 7243, ISO - 7730 and ISO - 7933, in which the main indicators that summarize most parameters of thermal protection-effective temperature, WBGT index, index forecasting subjective heat sensation (PMV), an index for predicting thermal discomfort (PPD) index of heat stress (HSI), which indicators allow assessments such as thermal comfort, level of discomfort, warning of the danger, zone of thermal tolerance, regime of work and rest and others.

Comprehensive evaluation of thermal conditions by a WBGT index (BS EN 27243; ISO - 7243) allows to determine its reference values at different intensity of work (metabolic heat in W or W/m^2) and at rest, in acclimatized or no acclimatized persons to overheating conditions, at a speed of air movement above or below 0,1 m/s, standard clothing-0,6 clo.

Depending on the values of WBGTi and metabolic heat can determine the mode of work / rest for every hour of work, in acclimatized individuals, speeds of air movement over 0,1 m/s (as the rest is the same or close to the microclimate conditions as in the workplace - the same values WBGTi).

By the method of Fanger (ISO-7730) based on data of exercise intensity (in units met - 1 met = 58 W/m^2), thermoresistance of clothing (clo), tension of water vapor (kPa) and air temperature and on surfaces (average radiation temperature), are determined subjectively predicted heat sensation (PMV) and predicted percentage of subjects with discomfort under the given conditions (PPD). The thermal comfort is at PMV ± 0,5 and PPD to 10%.

In terms of overheating more informative and more important for the essence of preventive measures is heat stress index (HSI) ISO - 7933.

In the index of heat stress can be defined and its degree (mild to moderate, strong, very strong, maximum), its impact on affect people, the necessary preventive measures and contraindications for work **(Table. 2)**.

The method of heat stress index is used to determine the permissible exposure and work with relevant thermal conditions.

Used and analytical (separately) regulation of microclimate parameters (BS 14776-87), in depending on the intensity of physical labour, seasons (hot and cold) and the release of convection heat in the working space (above and below 23,26 J. $m^{-3}.s^{-1}$). Standards are optimal and acceptable (with some intension of thermoregulatory mechanisms and decrease in efficiency).

In recent years, it is recommended to create a strategy for evaluating the thermal conditions, under which studies and assessments made in stages (preliminary, working, in-depth analyzes), indicate the most informative indicators and the most appropriate solutions for the protection of influenced.

6.1.5. PREVENTION OF ADVERSE EFFECTS OF UNCOMFORTABLE MICROCLIMATE

Main directions of prevention activities are:

- Controlling microclimate parameters and other related factors that are relevant to the thermal state of the body.

- Control of the state of the acting organism - an assessment of physiological mechanisms of thermal equilibrium and medical control on health (preliminary medical examination, professional selection, periodic medical examinations)

- Rational regime of work and rest

- Rational feed - and drinking regime

- Thermal adaptation (heat acclimatization and training)

- Technical means - shielding, remote control, ventilation and others.

- Personal protective equipment - protective clothing, head protection, eyes, hands.

1. Medical control

The preliminary medical examinations in appropriate volume of research and testing, providing information on the health status of job applicants with adverse climate, it is particularly important to ensure the reliability of the physical and psychological realization without overintension of thermoregulatory mechanisms, i.e. biosocial low price.

Discovering or presence of acute or chronic diseases (current or past history), of cardiovascular system, kidneys, liver, endocrine system, lungs and skin are contraindications to work after prolonged heat exposure.

Attention should be paid and to clarify the used drugs for the cardiovascular system - diuretics, antidepressants, sedatives, tranquilizers, and also narcotics, alcohol, reducing opportunities for adaptation to thermal environmental conditions.

The scope of research must include glucose tolerant test, renal clearance, X-ray of the lungs, abdominal ultrasound scan, pulmonary function tests, ECG, complete blood count and urine.

All persons subject to heat stress are subject to periodic medical examinations - for persons 45 years of age every two years and over 45 - every year (NIOSH). The scope of such reviews shall include a set of preliminary examinations.

Persons over 45 are also subject to extensive studies of the functional state of the cardiovascular,

Tab. 2. Index of heat stress (HSI) at 8 h exposure

Index	Effects, protection
0 10 20 30	Comfort Mild to moderate heat stress, exercise capacity is reduced, difficulties are intellectual activity, dexterity and vigilance
40 50 60	Strong heat stress, reduced working capacity; application of rational regime of work and rest; medical control on the status of workers; contraindications - mental activity, people with diseases of the cardio- and respiratory system and skin
70 80 90	Very strong heat stress in which even minor ailments in workers can affect the physical capacity to inability to perform the job; application of technical means of protection; rational regime of work, rest and feed- drinking regime (filling of water-salt losses); medical examinations (preliminary and periodical); conducting heat adaptation (in real terms or artificial heat-training)
100	Maximum stress, that can be borne only by young men acclimatized

respiratory, urinary systems, basal metabolism and skin state.

For individuals who are loaded heat only sporadic (seasonal workers in the summer heat), reviews conducted before the campaign starts. And when the thermal stress is constant (year-round) examinations are carried out by seasons.

2. Thermal adaptation (acclimatization, heat trainings)

The purpose of conducting acclimatization (adaptation to real environmental conditions) is to improve the general condition, the easy tolerance to heat or cold stress, shortening the recovery of functional status and performance, and depends of individual characteristics of the organism.

Acclimatization to heat is a process that involves complex physiological (and pathological may temporarily) mechanisms that occur during the first weeks of real work in state of overheating or artificial conditions (heat chambers).

Initial effects from exposure to overheating environmental conditions are fatigue and discomfort, irritability, difficulties in the implementation of physical and mental activity, susceptibility to muscle cramps.

The process of acclimatization is carried out in a certain consecutivity. First increases skin temperature as a result of the maximum dilatation of the cutaneous vessels. This was loaded cardiovascular system, as demonstrated by a decrease in pulse rate, an increase in stroke volume, reduction in body temperature.

The first adaptive response to thermal effects is to increase the degree of sweating to two times compared with the usual values of unadapted persons. Exercise and workouts lead to the amplification function of the sweat glands. Reduced sweating on thermal effect shows a deficit of physical capacity and behavioural adaptation to heat, i.e. these are persons with reduced tolerance.

Potential risks of significant sweating are dehydration and salt loss (also and separation of water-soluble vitamins - C, B_1, B_2). Reducing water capacity does not increase tolerance to thirst or dehydration, but otherwise deteriorated thermoregulation and reduced work capacity and immunological reactivity.

The loss of salts, however, is considerably less after acclimating - whereas previously the salt concentration is 3 g/l, is then reduced by more than half in sweat. The excretion of salts and reduces in the kidneys.

The adaptation of the cardiovascular system to heat impact is related to the degree of sweating.

Changes in metabolism by thermal effects towards the reduction was not observed or is it slightly - to about 10%.

By hormones in the process of thermal adaptation, mainly to water balance, prevailed aldosterone and antidiuretic hormone. Aldosterone promotes the reabsorption of sodium chloride from the sweat glands, and kidneys. Conversely, aldosterone secretion is stimulated by the negative salt balance.

Antidiuretic hormone (ADH) leads to water retention by the kidneys. In thermal impact and adoption of water reduces the secretion of ADH. In acclimatization increases the rate of production of ADH and aldosterone.

After 4-6 weeks in an overheating conditions there are signs of good adaptation, characterized by less strain on thermoregulation and cardiovascular system, increasing efficiency. For full stable acclimatization to overheating conditions take years.

Adaptation speak on condition that the fluctuations of the parameters of thermal environmental factors trigger response does not go beyond compensatory possibilities of the organism. Emergency actions - high intensity for a long time, lead to the collapse of adaptation. The latter is associated with lowered immune reactivity, and the appearance of a variety of disease disorders, respiratory diseases, peripheral nervous and cardiovascular system (myocardiopathy, miocardyodistona, vazopathy, atherosclerosis, hypertension, coronary heart disease), gastrointestinal tract (chronic gastritis , colitis, peptic ulcer disease), of ENT diseases (pharyngitis, laryngitis, chronic tonsillitis, rhinitis), allergic diseases. Acclimatization is a constant process. Only after a week of absence from overheating conditions needed four days to re-acclimatization, and after 3-4 weeks of cooling acclimatization is completely lost.

Artificial acclimatization (heat workouts). Physiologically natural and artificial conditions for acclimatization are similar, but in natural there are mitigating factors - choice of dress, work stoppage in the hottest time of the day, the adoption of water-salt solutions, relaxation in state of thermal comfort.

In artificial conditions, in special heat chambers create microclimate parameters imitating a production with a certain intensity of work and using garments with different TR. It is believed that it is better when worn clothing preventing the release of sweat. Skin temperature increases significantly (due to difficult sweating) and the application of light exercise for 1h, leads to increase in body temperature above 38 ° C. After a period of 2-3 weeks the adaptive response is characterized by acclimatization to thermal impact and increased physical condition.

Acclimation to cooling. It will not matter so much as overheating as much more easily retained skin and body temperature by appropriate clothing,

mode of operation, nutrition and drinking regime. Increasing heat production in cold-effect can be increased by proper diet to include more protein, fat and vitamins.

In the adaptation of the worker to cooling increases metabolism, increases in heat production, faster restores skin temperature, less is expressed spasm of dermal vessels, increased's blood supply, is increased volume of circulating blood, activates thyroid gland, increasing the excretion of corticosteroids and catecholamines.

3. Drinking mode in the conditions of thermal load

Intense sweating under thermal stress causes a loss of sweat of a significant amount of water and mineral salts, mainly sodium chloride, but the same and potassium, calcium, trace elements - copper, zinc, iodine, water soluble vitamins - C B_1, B_2. The reduced inventory of the salts in the body cause the acceptance of larger quantities of liquids, whereby sweat increases, but also the salt losses. Despite the adoption of large amounts of fluids exist constant feeling of thirst, dry mouth and nasopharynx.

If the loss of sweat is up to 5% of body weight no noticeable correlation between sweat and water intake.

The question of the necessary intake water (fluid) has not yet been resolved. Unlimited fluid intake in conditions of intense sweating no overloading the cardiovascular system to 8-9 l. Usually it recommends the adoption of 2-3 l cold water. Thirst can be removed with the adoption of drinking water or weakly mineralized, not necessarily salted, improved taste with ascorbic or citric acid. Use and specially prepared drinks for working in hot shops.

4. The role of workers in overheating conditions

Acclimatization to heat impact requires above all a positive motivation of workers who, through appropriate behaviours can reduce adverse effects and accelerate the process of adaptation. To fulfill this role, working under conditions of overheating must maintain a good level of physical and mental health, to conduct endurance treatments, gymnastics and implement appropriate preventive measures. For this purpose, workers must be informed of the thermal conditions in production, the impact on the body and preventive measures.

5. Rational regime of work and rest

6. Technical and technological protective means

7. Protective clothing - features to meet the thermal conditions and technological process to reflect the thermal radiation, to facilitate the effect of "pump" and the heat loss from the surface through evaporation of sweat, for retaining body heat, etc. Personal protective equipment (goggles, gloves, shoes) are also in line with the thermal conditions and major adverse component. In the prevalence of the risk of damage from infrared radiation - metalling with a highly reflective clothing, spectacle lenses, gloves.

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6.2 NON-IONIZING RADIATION

Electromagnetic radiation includes oscillating (wave) electric and magnetic field. The electric waves are distinguished by their frequency (Hz), the wavelength (m) and energy (eV). Non-ionizing electromagnetic radiation is one that under normal conditions can not cause ionization in biological objects (in the tissues of humans and animals) due to low power - less than 12,4 eV. These radiations are of a longer wavelength from ionizing (X-rays, gamma rays) and with less energy. Depending on their physical characteristics (wavelength, frequency, method of radiation) and their biological effects, they are divided into ultraviolet radiation (UV), visible light, infrared radiation (IR), "microwaves" and radio waves, low frequency waves (transmission of electricity) lasers. These radiations cause a variety of changes to the substance - changes in vibrational and rotational energy of molecules (thermal effects); excitation of the ions and molecules (electrons passing from one orbit to another); photochemical, phototoxic and photosensitizing effects. In air and in gaseous environment they can cause and ionization (short wave UV radiation). A general rule is that the smaller the wavelength, the higher the frequency and energy of the radiation **(Table. 1)**.

Tab. 1. Non-ionizeing rays - frequency, wavelenght, energy

Type of radiation	Frequency (Hz)	Wavelength (m)	Energy (eV)
Ionizing	>3000 THz	<100 nm	>12.40 eV
Ultraviolet (non-ionizing)	3000-750 THz	100-400 nm	12.40-3.10 eV
UV-vacuum	$3x10^5$ to 30000-1580	1 to 10-190	1240 to 124-6.53
Far	1580-1000	190-300	6.53-4.13
Near	1000-750	300-400	4.13-3.10
UV-C	3000-1070	100-280	12.40-4.43
UV-B	1070-952	250-315	4.43-3.94
UV-A (black light)	952-750	315-400	3.94-3.10
visible light*	750-385 THz	400-780 nm	3.10-1.59 eV
Infrared (IR)	385-0.3 THz	0.78-1000 μm	1590-1.24 meV
IR-A	385-214	0.78-1.4	1590-886
IR-B	214-100	1.4-3	886-413
IR-C	100-0.3	3-1000	413-1.24
Near	385-100	0.78-3	1590-413
Middle	100-10	3-30	413-41.3
Far	10-0.3	30-1000	41.3-1.24
Laser	1500-15	0.2-20	6200-62
Class 1 - no risk			
Class 2 - low risk, low power			
Class 3a - low risk, medium power			
Class 3b - medium risk, medium power			
Class 4 - high risk, high power			
Radio frequency (RF)	300GHz-0.1MHz	1mm-3000m	1240μeV-0.41neV
Extremely high frequency (EHF) } "microwaves" (MW)	300-30GHz	1-10 mm	1240-124μeV
Super high frequency (SHF) } "microwaves" (MW)	30-3	10-100	124-12.4
Ultra high frequency (UHF) } "microwaves" (MW)	3-0.3	100-1000	12.4-1.24
Very high frequency (VHF)	300-30 MHz	1-10 m	1240-124 neV
High frequency (HF)	30-3	10-100	124-12.4
Medium frequency (MF)	3.03	100-1000	12.4-1.24
Low frequency (LF)	300-30 kHz	1-10 km	1240-124 peV
Very low frequency (VLF)	30-3	10-100	124-12.4
–	3-03	100-1000	12.4-1.24
Extremely low frequency (ELF)	<0.3 kHz	>1000 km	<1.24 peV

* The limits of the human eye varies individually from 380-400 nm to 750-780 nm

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6.2.1 INFRARED RADIATION

Infrared rays were discovered by English astronomer William Herschel in 1800 when studying the spectrum of solar radiation and received its name from Becquerel in 1869.

Infrared region of the spectrum lies between the red limit of visible light (750-780 nm) and range of radio waves ("microwaves" - 1-10 mm).

The biological properties of infrared rays are closely related to their wavelength. Depending on three set parts of the IR spectrum: (A) - near the region with a wavelength of 0.76 to 2.5 μm; (B) - middle - from 2.5 to 25 μm and (C) - far - from 25 to 1000μm (division is arbitrary and different authors indicate various levels of the three ranges).

Another characteristic of infrared waves is their density - the amount of radiation passing through a 1 cm^2 area (W/cm^2).

Infrared radiation (IR) as all electromagnetic radiation has characteristics - radiation, absorption, reflection, diffraction, transmission. Like other broadcasts it can be absorbed by the body over which falls and is converted into heat (hence the name of their "heat rays"). Heat radiation is of all bodies at a temperature above absolute zero, and character (spectrum) depends on the physical state of the substances. While passing through the air (unpolluted), IR radiation, it no heats.

Radiation heat transfer between the bodies depends on the so-called temperature gradient - radiating - absorbing body. Different objects absorb infrared rays in varying degrees. The lens is transparent to wavelengths from 2.5 to 27 μm, water - about 1.5 or more μm. There are different opinions on the permeability of skin to infrared rays with different wavelength. Indicate - Gersten, Mellete, Strow et al., the skin is transparent to infrared rays having a wavelength of 0.7 to 1.5 μm and relatively opaque in the range of 1.5 to 5 μm. Conversely, Taylor, Rucket et al., Indicate strong absorption by the skin of IR-rays having a wavelength of 3-6 μm - close to the transparency of the water.

Thermal radiation is subject to the physical laws that are important hygienic matter - depends only on the condition of the emitting body, not from the environment (Law Virchow) and is proportional to the temperature of the radiating surface. When increasing the temperature of the source, the spectrum of radiation moves towards shorter rays and the intensity of the emitted power increases. For bodies heated to a temperature 400 500 ° C, is characterized by long-wave radiation; in molten metal (temperature 1600 ° C) - 22% of energy is at the expense of shortwave and at 2370 ° C - 43% shortwave broadcasts. Even with no large increase in the temperature of the body, letting the heat by radiation significantly increased. At the temperature of the source may tentatively to determine the wavelength of maximum radiation and assess the biological effect (Vin's law

$$C = \frac{\max. \lambda}{T}$$

where C - constant Vin is 2880, T - degrees K, λmax - μm). By raising the temperature of the emitting body power of radiation is directly proportional to the fourth degree of absolute temperature - $E = \delta T^4$ (W/m^2) - law Stefan-Boltzmann, where $\delta = 5.775.10^{-9}$, T - degrees K.

There are numerous sources of infrared radiation. The most intense natural source of IR is the Sun and the number of exposed persons and the duration of exposure is significant. The radiation energy of the sun falling on the Earth's surface is approximately 1.355 kWh/m^2, half of this energy is due to infrared radiation. The radiation from the sun comparable to that of a black body heated to about 6000 ° C.

Artificial sources are also numerous in recent years greatly increased use of IR in different industries - bread, ceramic industry, heat treatment of various materials, drying of various products and the like, rose and exposed contingents and duration of exposure.

The intensity of the heat radiation of work places and people depend on: the nature of the process, the temperature of the radiation source, the distance from the source, the degree of thermoinsulation, collective and individual preventive means.

To hot workshops relate those that are heat (convection heat) over 23 J/m^3. In the main workshops of the steel industry (blast, converter, martin furnace, blooming, electrical steel etc.), the intensity of IR is 348 to 13 920 W/m^2; engineering (foundry, blacksmith workshops and workshops for processing details) intensity of radiation from 1392 to 3480 W/

m^2. With the intense heat radiation are characterized and working conditions outdoors in summer heat - construction and agricultural activities. Endangered contingents are involved in the production of glass - intensity 2100-2800 W/m^2, electric welders - intensity about 3 W/m^2. In the latter, however, it is a small area of the radiating surface and health problems are related primarily to UV-radiation.

6.2.1.1. Biological effects

Thermal (infrared) radiation is an important component of the microclimate (see Section 6.1).

Biological activity of IR has a number of features that are directly dependent on the wavelength exposure dose, duration of exposure, repetition rate of impact, peculiarities of influence tissues.

Effects on eye structures

Ocular structures are particularly sensitive to the action of IR. Under natural conditions of human (non-production exposure) there are safeguards to reduce the adverse effects - blink, pupillary light reflex. In production conditions damage to the ocular structures are mostly made of shortwave range:

Lentils. - The exceptional exposure in this range leads to the development of so-called. heat cataracts (first described in workers in the glass industry as a "cataract of the glass blowers"). It is expressed in turbidity of the posterior surface of the lens, such as at a wavelength of 1.2 to 1.4 μm is more pronounced at the periphery. The temperature of the lens can be increased directly (rays having a wavelength of 0.8 -1.2 μm) depending on its spectrosorption characteristic or indirectly by heat transfer of the iris. It is believed that at energy levels of about 1.4 kW/m^2 for many years the development of cataract is not certain. In modern technologies sources IR risk of developing cataracts is less or cataract is less pronounced. The development of cataracts important are the energy levels of impact, exposure, age. Heredity, race, use of drugs, diseases, immune and climatic factors may predispose to cataractogenesis.

For now adopted two theories about the development of cataracts by IR. One that cataract due to direct absorption of thermal energy or by transfer of heat from other ocular structures (iris, chamber fluid) and the other that the initials are photochemical damage of the lens, which are depending on the energy dose (energy level and exposure).

Retina. - It absorbs short infrared rays, which do not differ in penetrating from visible and have a predominantly thermal effect. Sudden exposure to high energy levels of IR causes temporary blindness and chorioretinal burns as a result of which occur scotoma - eg. when using electric arc and unprotected eyes.

Several factors are important in determining the exposure of the retina: pupil diameter, the spectral transmission in the middle segment of the eye, the spectral absorption of the retina and choroid, and the size and quality of the image. Adapting to darkness pupil has a 7 mm diameter compared with normal - 2-3 mm in normal sunlight. This allows the entry of a greater amount of energy into the eye. Some short-wave radiation to 1.4 μm reach the retina through absorption of the middle segment (cornea, vitreous, lens). The optical transmission is greater in length from 0.5 to 0.9 μm, reduced by 50% at 1 μm and reached the lowest levels in the 1.2-1.4 μm. With the increase of incoming energy and its absorption by the retina and choroid increases the risk of their defeat.

Iris. - It is very sensitive to the IR due to the high absorption of dark pigment. Moderate doses cause constriction of the pupil (hyperemic miosis) and formation water secretion - two protective mechanism against IR.

Cornea. - Upon exposure to high intensity long infrared rays cornea reacts painfully and includes safeguards - reflex closing of eyelids and turning his head in the opposite direction of the source. It is assumed that the primary is the response of the thermal irritation (it is for a parts of a second and is obtained and at low temperatures - approximately 45 ° C, corresponding to the power level absorbed by the cornea about 100 kW/m^2), because of the high sensitivity of sensory corneal nerve, after which arises and pain reflexes. Under normal production exposures of IR does not observed burns. At high intensities burns occur in corneal epithelium, and sometimes may even develop opacities.

Conjunctiva. - Cases of conjunctivitis with reduced lacrimation were observed in exposure to near IR - effective wavelength of 0.98 μm and intensity of the radiation 650 W/m^2. Continued exposure to IR is associated with increased evaporation, leading to tear deficiency and development of so-called "dry" eyes.

Eyelids. - Flashing protects the eyes from the effects of IR and move liquid to the front surface of the eye - double protection from the heat. Anatomical structures of the eyelids are similar to those of the skin and thus to damage on them will also be similar - erythema, edema, vesicles.

Effects on the skin.

Damage the skin depends on its thermal and optical characteristics, of its ability to absorb radiation energy. On reflection and absorption characteristics can be determined and the effects - in IR below 2 μm reflective properties depend on skin pigmentation and blood flow, and the maximum reflection is

between 0.7 and 1.2 μm, which corresponds to the maximum intensity of infrared effect. The lesions of the skin depend on the degree of difraction which is determined by the microstructure of the skin and of penetrating ability of IR (i.e. a transmissible spectrum, dependent on the water content, which is about 60-70%). Typically, the skin is impermeable to lengths of less than 2 μm, but the shorter IR radiation can cause burns. Medium and long-rays are important factors as thermal load in the manufacture exposed persons. Manifest signs of skin lesions by short infrared rays at chronic effects are erythematic, skin pigmentation and burns. Short-wave radiation penetrates to a few cm into the tissue and causes skin burns and persistent skin pigmentation.

Other effects.

Besides lesions to so-called 0rgans targets for IR (eye structures, skin) were observed also in humans and experiment: vascular congestion of the spleen and kidneys; changes in the immune reactivity (phagocytic index, bactericidal properties of the skin); damage to the upper respiratory tract (chronic rhinitis, laryngitis and sinusitis - 10 times more common in persons exposed to IR); temporary reduction in sperm production; genetic effects - chromosomal aberrations by short IR rays; mutagenic effects in experimental animals; probable carcinogenic effect.

Typical heat effect of IR on the brain in intensive direct irradiation of unprotected head is so-called "Sunstroke" (see labour microclimate).

6.2.1.2. Hygiene normalization

Safe exposure to IR on the eyes is offered by a number of authors. According to some acute ocular changes (on the cornea) were observed at exposure to the energy levels of 40-80 kW/m^2 (however, ignores the duration of effect in acute cases) and are recommended as safe levels in chronic effects - 4 to 8 kW/m^2. Other authors indicate as safe levels for chronic exposure in attitude of ocular 0.1 kW/m^2, and a few minutes of exposure is allowed up to 10 times more.

To protect the eye safe levels for sources of short-wave IR and visible light - 385-3000 nm, recommended by the WHO, ILO, the United States Conference of Labour Hygienists (ACGIH) and the International Commission on Non Ionizing Radiation Protection (ICNIRP), in manufacturing exposure for 8 h working day, at known radiation spectrum (Lλ) and total radiation from the source (E), measured at the position of the eyes of the worker:

1. To protect against retinal thermal damage, spectral radiation from sources of visible light, weighted against function R (λ), showing the degree of risk of burns **(Table. 2)** should not exceed:

$$\sum_{385}^{1400} L(\lambda).\ R(\lambda) \le 5/\alpha.t^{1/4}$$

Where Lλ is the spectral radiation in W (cm^2. sr.nm); R (λ) - measured risk of burns associated with the range of the wavelength Δλ source; t - time (exposure) in s, but a period of 10 μs to 10 s; α (in rad) = l/r, wherein l is the distance to the source, r - the visible average size (diameter) of the source (and the two dimensions in cm or m).

2. To protect the cornea and lens (cataract-genesis) of thermal injury from IR - 770 nm-3 μm, exposure to hot proceedings should be limited to 1000 s, and up to 10 mW/cm^2 or:

$$\sum_{770}^{3000} E\lambda.\ \Delta\lambda \le 1.8^{-3/4}\ W/cm^2 \text{ (for } t < 1000 \text{ s)}$$

3. To protect the retina from a near IR (IR-A-770-1400 nm), the absence of a strong light stimulus of source of - brightness under 10 $^{-2}$ cd/cm^2, the effect is limited to

$$\sum_{770}^{1400} L\lambda.\ \Delta\lambda \le 0.6/\alpha$$

a period over of 10 s. For periods of less than 10 s is used first equation (but for Δλ - 770-1400 nm). This limit is based on pupil diameter of 7 mm. Both equations are empirical and for accuracy introduces spatial correction factors to their right part - respectively K_1 = 1W. rad. s$^{1/4}$/(cm^2. sr) and K_2 = 1W. rad/(cm^2. sr).

Normalization of IR as thermal environmental factor is performed as a complex acting with other thermal factors (temperature, humidity and air velocity) by standarts ISO 7730; 7243; 7933. For existing BS 14770-87 value of the IR in the workplace are standardized separately - up to 350 W/m^2.

6.2.1.3. Prevention of adverse biological effects

Medical control. - Preliminary medical examinations are conducted with particular attention on the state of the eye and skin structures and contraindications for work in conditions of intense IR. Periodic medical examinations to be aimed at establishing early, subclinical changes in view to implement timely protection of vulnerable groups - removal from the working environment, differentiating the age of the professional changes.

To refine the biological effects is neccessary analysis of specific subclinical abnormalities, study of late effects - mutagenic, carcinogenic and clarify the basic mechanisms of the injured on biological systems.

Technical control. - Regularly performing control measurements of the sources of IR; specifying the distance from the source; shaping separated cabins and work places; shielding of the sources (metallic curtains, which reduce the intensity of the radiation by 80%); installation of glass or plastic intermediate doors; application of special signaling systems for locking; ensure effective ventilation and water circulation.

Personal protective equipment. - Protective clothing with a reflective metallic coating - mostly aluminum foil; IR filtering goggles and shields, gloves, and at very high intensities and reflective costumes. Glasses unless protective qualities must have a good aesthetic design, are comfortable to wear and do not become a secondary source of heat effects on the cornea. The choice of metal coatings for glasses depends on the type of radiation - length, intensity and are different for different industries - eg. in foundries advised cobalt blue glass, to control the temperature of the metal combined with glass absorbing heat.

Tab. 2. Spectral weighting functions for determining the risk of photochemical and thermal damage to the retina of radiation in the range 305-1400 nm

Wavelength (nm)	Lensectonomy Risk Function A(λ)	"Blue light" Risk Function B(λ)	Thermal damage Risk Function R(λ)
305-335	6.00	0.01	–
340	5.88	0.01	–
345	5.71	0.01	–
350	5.46	0.01	–
355	5.22	0.01	–
360	4.62	0.01	–
365	4.29	0.01	–
370	3.75	0.01	–
375	3.56	0.01	–
380	3.19	0.01	–
385	2.31	0.0125	0.125
390	1.88	0.025	0.25
395	1.58	0.050	0.5
400	1.43	0.100	1.0
405	1.30	0.200	2.0
410	1.25	0.400	4.0
415	1.20	0.800	8.0
420	1.15	0.900	9.0
425	1.11	0.950	9.5
430	1.07	0.980	9.8
435	1.03	1.000	10.0
440	1.000	1.000	10.0
445	0.970	0.970	9.7
450	0.940	0.940	9.4
455	0.900	0.900	9.0
460	0.800	0.800	8.0
465	0.700	0.700	7.0
470	0.620	0.620	6.2
475	0.550	0.550	5.5
480	0.450	0.450	4.5
485	0.400	0.400	4.0
490	0.220	0.220	2.2
495	0.160	0.160	1.6
500	0.100	0.100	1.0
505	0.079	0.079	1.0
510	0.063	0.063	1.0
515	0.050	0.050	1.0
520	0.040	0.040	1.0
525	0.032	0.032	1.0
530	0.025	0.025	1.0
535	0.020	0.020	1.0
540	0.016	0.016	1.0
545	0.013	0.013	1.0
550	0.010	0.010	1.0
555	0.008	0.008	1.0
560	0.006	0.006	1.0
565	0.005	0.005	1.0
570	0.004	0.004	1.0
575	0.003	0.003	1.0
580	0.002	0.002	1.0
585	0.002	0.002	1.0
590	0.001	0.001	1.0
595	0.001	0.001	1.0
600-700	0.001	0.001	1.0
700-1050	–	–	$10^{[(700-\lambda)/500]}$
1050-1400	–	–	0.2

Note: *To protect the retina from photochemical damage in the so-called sources "Blue Light" (305-700 nm) - fluorescent lamps, halogen lamps, film projectors, arc lamps, etc., using other equations depending on the exposure time (over or under 10^4s), spectral radiation - L blue, over 10 mW / (cm2.sr), viewing angle up to 0.011 rad, working with the lensectonomy.*

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D. Tsvetkov

6.2.2 ULTRAVIOLET RADIATION

Ultraviolet radiation includes the spectrum of non-ionizing radiation between visible light and ionizing radiation - a wavelength of 100-400 nm (indicate and lower values - up to 1-4 nm). Due to the relatively high photon energy when it has a wide variety of biological effects on the one side and on the other - its lowest penetration limited its direct biological activity to surface tissues and organs of the body (skin, eyes).

By their physical characteristics and biological effects UV-radiation is divided into near range - 300-400 nm range and far - 180 (200) - 300 nm. Ray shorter than 180/200/nm are absorbed by the air and can not practically exhibit a biological effect (they exist in a vacuum or in a non-swelling them gas). Since this division is associated primarily with the penetration of the UV-rays into the skin and of air absorb, by biological point of view used their division: range A -320(315)-400 nm (predominantly pigmentforming action); range - 280-320 (315)nm (predominantly erythema and vitamin D forming action) and a range - below 280 nm (bactericidal action). The biological effect is determined not only by the wavelength of the UV-rays, but also by the radiation power (W/cm^2), the exposure time and their penetration into the tissues, so that using both classifications. For example, a low bactericidal effect is visible light, but under irradiation with the same intensity of test cultures Esh. coli they perish for 2-30 s in exposure to radiation 210-280 nm and above 1900 s - with rays 300-330 nm, ie varying the degree of expression (or practical presence) of these biological effects.

6.2.2.1. Sources to UV-radiation

Sources to UV-radiation are all bodies with a temperature of over 2500 K.

The main source of UV-radiation is the **sun**. As a absolutely blackbody at temperature of 5600 K, it emits UV-rays by wavelength substantially below 290 nm, but on earth ground reached rays only over this length, making and all life on earth in its present state (short-wave UV-radiation it is lethal to most organisms on the surface of the earth). Short UV-rays are absorbed by Earth's atmosphere, at an altitude of about 200 km and an lower absorbed rays with a wavelength below 220 nm (240 nm) (from atmospheric oxygen, nitrogen and hydrogen) and lower - from molecular oxygen resulting in the formation of ozone - O_3). Producing photochemical ozone, with a maximum concentration at a height of about 50 km (35-70 km) absorbs radiation with a wavelength of 220-290 nm **(Fig. 1)**. The formation and disintegration of ozone is a dynamic process dependent on natural conditions - latitude, annual and diurnal variations and human activities. Separating the hydrochlorofluorocarbons (CFCs) in the atmosphere (gas refrigerant in refrigerators, sprays, etc.), as well as nitrogen oxide, catalytic reduces ozone. The possibilities for reducing ozone can predict the expected increase in the incidence of skin cancer. Indicate that expected reductions in the thickness of the ozone layer by 1% will increase by 3% cases of basal cell skin cancer and by 5% - of planocellular skin cancer. It should be pointed out, however, that the pollution of the Earth's atmosphere with dust, soot, gases - ammonia, sulfur gases, also nitrogen oxides, leading to strong absorption of UV-radiation, so now air pollution is associated primarily with absorption of UV-radiation, not by its increasing resulting of effect on the ozone layer. Absorbtion to UV-rays depends generally on the thickness and "transparency" of the atmosphere (air layer) through which they pass - so that altitude, season, far geographic latitude, time of the day strongly influence the intensity and spectrum. The contaminated air, low standing in the sun above the horizon (latitudes, winter season, early and late hours during the day) not only greatly re-

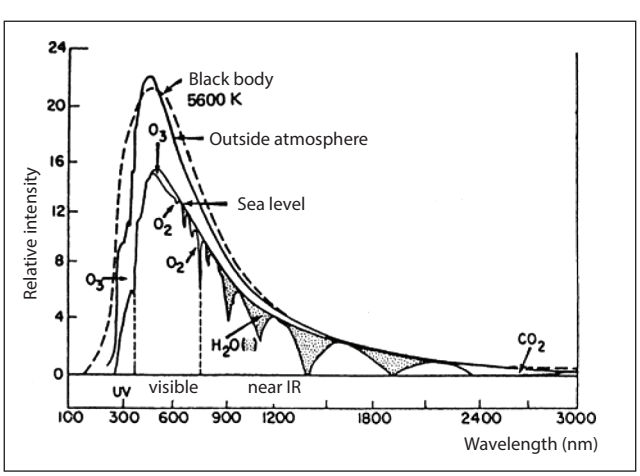

Fig. 1. The solar spectrum in the upper atmosphere and sea levels (by Giese A.C., 1976)

duce the total intensity of UV-radiation, but absorb much stronger its shortwave ingredient (increases the lower limit of reach the earth's surface UV-radiation). For example, in the summer in 12 h lower limit to UV-radiation in Sofia is 302 nm, and the Black Sea coast - 297 nm.

In rooms UV-radiation is almost entirely absorbed by ordinary clear glass (containing traces of iron and titanium oxides).

Artificial sources of UV-radiation are numerous. Discharge lamps - mercury at low-, medium- and high pressure, metalhalogen lamps, xenon lamps, lamp "lightnings" have different lumination power and spectral composition. These by low pressure emit interrupted UV-spectrum (with determinate wavelengths). Arc welding (metal electrodes with protective coatings) emits a continuous spectrum UV-rays, depending on the metal surface, its cover and the gas protection during welding (nitrogen, carbon dioxide). Carbon electrodes (arc) are also a powerful source to UV-radiation. Fluorescent lamps used for lighting have retentive filter for the UV-rays (usually glass), while those emitting UV-rays (UV-B and UV-A range) are made of quartz glass. The spectrum of the UV-rays depends also on the kind of the fluorescent substance. They are also used lasers emitting UV-rays - in nitrogen lasers (337 nm), heliumcadmium lasers (325 nm) and others. The wavelength here depends on the active base and the operating mode of the laser.

6.2.2.2. Absorbtion and general biological action

Penetration of UV-radiation in biological tissues is limited **(Fig. 2)**. In the skin near UV-radiation (300-400 nm) penetrate to a depth of 0,1-1 mm - in str. corneum and str. Malpigii the epidermis. Far UV-radiation (200-300 nm) penetrates only to 0,01-0,1 mm (greater part is absorbed by str corneum, and low fraction penetrates into str. Malpigii). A similar is penetration to UV-rays in the eye. They are absorbed primarily in the cornea. To reach the vitreous rays with a wavelength exceeding 295 nm, and the retina - up 330 nm.

Fig. 2. Penetration to UV and visible radiation in lightly pigmented skin in Europeans - in% compared to perpendicular radiation (in Bruls W.A. - 1984).

0		100	100	100	100	100	100	100	100	100
		42	30	28	30	50	67	80	85	89
20	Str. corneum	18	9.1	7.9	14	25	44	64	72	80
30		5.0	2.4	2.5	8.5	15	33	50	62	72
40	Str. Malpigii	1.4	0.65	0.78	3.0	9.6	24	39	54	65
50		0.39	0.17	0.25	1.3	6.0	18	31	46	59
60		0.11	0.047	0.077	0.60	3.8	13	24	40	53
70		0.030	0.012	0.024	0.27	2.4	9.5	19	34	48
		254	270	280	290	297	313	365	436	546

Heereditary changes due to genetic, chromosomal and gene mutations are possible in lower organisms not in humans and animals due to the low penetration of UV-radiation.

Specific photochemical effect of the UV-radiation is the formation of vitamin D by irradiation of sterols. In skin cells of humans and animals under the influence of UV-rays (primarily from range B) of 7-dehydrocholesterol formed cholecalciferol (Vit D_3) - an important factor for calcium-phosphorus metabolism and prevention of rickets and senile osteomalacia.

Upon irradiation of ergosterol (in vegetable oils - in vitro) is obtained Vit D_2 (ergocalciferol) - no matter the prevention of rickets in humans (not absorbed by the digestive tract). Another characteristic effect of UV-radiation is the formation of melanin in cells of the str. basale of the epidermis. The UV-rays catalyzed oxidation of tryptophan to dihydroxyphenyl-L-alanine (DOPA) which forms melanin under the influence of DOPA-oxidase. The formation of melanin pigment is a protective reaction of the organism against UV-rays, associated with the condition and reaction of sympathicoadrenal system. Cutting the sympathetic nerve fibers in the experiment causes depigmentation of the skin. Conversely, the skin irri-

tation (mechanical, thermal, and primarily to UV-radiation) leads to enhanced production of adrenaline (vasoconstrictive action). Melanin is also such (like adrenaline) action, i.e. they have antagonistic activity on histamine and histamine like substances, related to the generation of UV-erythema. In addition, melanin (pigmented skin) has a higher reflectance (albedo) of the incident on the skin UV-radiation. Also in persons with more highly pigmented skin at the same other conditions is less risk of skin cancer upon irradiation with UV-rays.

Hypersensitive (photosensitive) effects are of two types - phototoxic (non-allergic) and photo-allergenic. In the first type UV-radiation is absorbed by the photosensitive substances to an exogenous or endogenous origin. Such as: medication - griseofulvin, tetracycline, chlorpromazine, sulfonamides, acridine, methoxsalen, furocoumarines, tar (containing anthracene and other photosensitive substances) bergamot oil (in many cosmetics) as well as chromophores - lactic acid, hematoporphyrin. The occurrence of hypersensitivity phototoxic reactions need simultaneous action to UV-radiation and photosensitizing agents. In photoallergic reactions need not be present (except the initial) of chemicals - only irradiation with UV-radiation may cause them. We need a little exposure and the effect usually occurs in individuals with acquired previously altered reactivity of type antigen-antibody or cellular immune response to a particular substance.

Photochemical reactions can be suppressed by substances such as vit E, β-carotene, vit C, which either resulted, excited by the UV-rays molecule to baseline or neutralize formed photochemical products.

6.2.2.3. Health effects (risk) under irradiation with UV-rays

These can be divided as follows: early and late non-stochastic effects; stochastic effects; indirectly affecting health.

Early non-stochastic effects. They occur on the skin and eyes (in rare cases describe effects on oral mucosa using faulty older models of apparatus for UV-photopolymerisation in the dental practice).

Skin describes four types of reactions:

Fast skin pigmentation, stronger occurring in people with pigmented skin. This effect manifests itself in the maximum irradiation with long UV-rays - maximum at 360 nm and is reported for radiation with a wavelength of 400 nm and above (visible light) - **Fig. 3.**

The ultraviolet erythema is characterized by: sharply defined borders (only where the skin is irradiated with UV-rays); latency period - 1-8 h, its

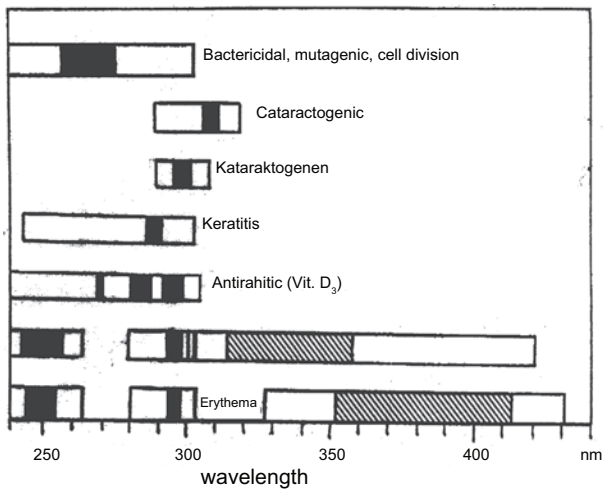

Fig. 3. Ultraviolet radiation - wavelength and biological effect: as in black and bar have a maximum effect (by Curtis R. and Nichols M. - 1983)

penetration; passing in pigmentation (tanning). Higher doses of UV-radiation reduces latency and increases the duration and degree of erythema. As most effective UV-rays and identifying those in the range 300-307 nm in sunlight and 254 nm and 297 nm - in artificial sources. UV erythema has a character of an inflammatory response - increased capillary permeability, edema, lymphocytic infiltration, which reaches a maximum on the second day after irradiation. At high doses of radiation, non-pigmented skin, young children, are possible and general reactions such as fever, chills, headache, vomiting, rare vascular collapse. Erythema followed by desquamation rarely leads to ulceration due to microorganisms (secondary infections in impaired skin protection). Erythema minimum dose (MED) is individual, very different and also depends on the exposure area of skin. In lightly pigmented skin MED is about 200 J/m^2 on body (chest, abdomen) in UV-rays in 250-300 nm, rises sharply in lengths exceeding 310 nm, and is 2.105 J/m^2 at radiation in the range 330-400 nm.

Third skin reactions **pigmentation (tanning)**. It is related both to the migration of pigment granules (melanin) of the adjacent deeper lying cells (from str. Malpigii to str. corneum) - initial phase and also by increased formation of melanin. To the extent that the pigmentation is mainly due to UV-erythema, the most effective UV-rays here are the same as those of the UV-erythema. But as mentioned in the long UV-rays - over 315/320/nm induce pigmentation in a much weaker erythemal reaction.

UV-radiation affects the **division of the skin cells**.

Immediately after irradiation (up to 24-36 h, depending on dose) there is inhibition of cell division followed by amplification - up to 72 h and continuing (at single exposure) 5-6 days. This hyperplasia of the epidermis combined with dropping the highly damaged cells (desquamation).

On eye the acute (once) action to UV-radiation occurs on the superficial tissue - conjunctiva, cornea and deep lying - vitreous and retina.

The most frequently observed effect is photokeratitis and fotoconjuctivities. It is caused by UV-rays with a wavelength below 315 nm, and is most pronounced at 270 nm (similar to the ultraviolet erythema). Minimum degree of manifestation to UV-keratitis (conjunctivitis) is required irradiation of 50 J/m^2 at 270 nm, 550 J/m^2 at 310 nm and 22500 J/m^2 at 315 nm. The effect is manifested 2-24 h after irradiation and lasts 1-5 days. The symptoms are photophobia, feeling of a foreign body ("sand") in the eye, blepharospasm, pain, lacrimation. Observed erosions of the cornea. The events resolved without sequelae. This effect can be manifested in the range of UV-A, but at very high doses of radiation. The effect is also called fotooftalmiya (ophthalmia electrica in arc welding).

Another acute effect is a ultraviolet cataracts caused by UV-rays in the range 295-400 nm, which pass through the cornea and about 80% is absorbed by the lens (lens crystalina). Especially relevant problem became acute UV-cataract after introduction to UV-lasers (powerful sources of UV-rays with specific wavelengths). UV-cataract is of two types. The first relates to thermal effects, to greater lengths UV-rays - maximum at 365 nm. In the second type are expressed photochemical reactions associated with the formation of pigment (melanin) of tryptophan (the so-called "pigment" cataract). Maximum this effect occurs in the range of 295-320 nm UV-rays. Acute UV-cataract occurs at high radiation doses at which pass both types of reactions (heat and pigment) to varying degrees.

Damage to the retina by intense UV-ray at A range established in an experiment on animals. In humans, it is possible the removal of lens crystalina (aphakia) and replacing it with a synthetic one (permeable to UV radiation below 300 nm). It is possible that such an injury when working with powerful lasers in the UV-long-wave range.

Another early non-stochastic effects unrelated to the exact dose of irradiation with UV-rays is hypersensitivity - **phototoxic** and **photoallergic effect** to UV-radiation.

Non-stochastic early effect associated with the dose of UV-irradiation is the **formation of vit D_3 in the skin**. With the high efficiency of this effect is erythema range (280-320 nm). The formation of vit D_3 is very important biological activities to UV-radiation of phosphor-calcium metabolism, prevention of rickets and osteomalacia senile, but the doses required for this are very low. Indication of necessary daily doses of about 5% of the MED of the skin surface of 200 cm^2 or about 60 MED irradiation annually in the head and arms (hands and fingers) for the prevention of rickets in children.

Late non-stochastic effects to UV-radiation also occur on the skin and eyes.

On the **skin** continuous UV-radiation causes the so-called skin aging - reduced elasticity, degeneration of collagen fibers, hyperpigmentation, telangiectasia. No dose for occurrence this condition (actinic skin), but it often goes into precancerous, such as solar keratosis, keratoacanthoma and Hatchinson melanosis.

On the **eyes** repeated UV-radiation in the range 295-400 nm causes photochemical cataracts. Epidemiological studies indicate an increased incidence of so-called senile cataracts in living near the equator (intensive UV-radiation) or working outdoors.

Stochastic effects are non-threshold, they are not related to the severity of the lesions, but with a probability of development and an increased incidence of disability - at irradiation with UV-radiation that is a skin cancer. Three types of skin cancer have been associated with UV-radiation - basal cell carcinoma, squamous cell carcinoma and melanoma. All ranges to UV-radiation can induce skin cancer, but believes that a more pronounced effect was erythema range B. Long and higher doses of radiation increases the incidence of skin cancer. It is rare in people with highly pigmented skin and more often in albinos, in people with precancerous skin. Indicate in US, much of the approximately 400 thousand cases of skin cancer annually due to exposure to UV-radiation from the sun (in production is not exactly proven this effect). Upon irradiation open-air demonstrated increased incidence of basal cell and squamous cell carcinomas of the skin, while melanoma malignum in humans is considered to be demonstrated increased its frequency of exposure to UV-radiation only in individuals suffering from xeroderma pigmentosum.

Some authors indicate that strong exposure to UV-radiation in children (with frequent sunburns) is associated with an increased risk of melanoma. On the other hand, melanoma develops more often in the trunk rather than on exposed parts - hands, face, neck exposed much more to UV-rays.

Stochastic effects in the eye are also associated with the development of precancerous (pterigium) of the conjunctiva, but also with melanoma malignum on anterior eye - chamber and iris (more common in people with blue eyes). These and other malignancies have been observed in experiment animals. In humans it is found at a higher frequency in people in tropical regions.

UV-radiation indirectly affects on health through its photochemical effects into the air and

foremost on air pollutants. The most typical example is the formation of Los Angeles smog. Under the influence UV-rays with a wavelength below 380 nm was carried out the most important primary reaction to the formation of this photochemical smog - decay of the liberated with exhaust gases from motor vehicles nitrogen dioxide to nitric oxide and atomic oxygen, and the last form ozone. In this nitrogen oxide with atmospheric oxygen again returns to nitrogen dioxide and the reaction is repeated throughout, while there is direct sunlight **(Fig. 4)**.

Second in importance primary reaction is the photo-dissociation of aldehydes; ketones; alkilaum and peroxyacyl nitrates; hydrogen peroxide and organic peroxides; nitric and nitrous acid to form free radicals - OH, NO, RCO, RO. These primary photooxidants - ozone, nitric oxide and free radicals, enter into complex chain reactions with formation of secondary photooxidants or other substances - olefinozonides, nitroolefines (nitropentan, nitrohexane), peroxyacyl nitrate (PAN), alcohols, ethers, acids. All these substances are highly irritating damaging effect on the skin, the mucosa of the airways and lung and eye, damage plants, degrade plastic and rubber products. Ultraviolet and less and the visible radiation, involved and other photochemical reactions in the atmosphere, such as excitation of the molecules of the oxygen and of other primary pollutants - halogens, condensation metal aerosols, are also included in the other chain of chemical reactions with the formation of a skin irritant , mucous membranes and lungs substances.

UV-rays have an indirect health effects, and the formation in the air with negatively charged light ions and ozone (from atmospheric oxygen), but this effect have radiation with a wavelength below 280 nm, i.e. these are the effects of artificial sources of UV-radiation (outdoor influence to UV-radiation of air ionization is very small).

A large number of experimental studies have shown that UV-radiation to change **the response of the immune system** alone or in combination with other factors impact. Indicate suppression of skin sensitivity and changes in the distribution of sub-populations of lymphocytes in humans and experimental animals irradiated with UV-rays, and also suppression of defense against tumors implanted in experimental animals. The popular belief that UV-radiation and obtained through her leather tanning increases the immune system, increase the resistivity against infectious agents and are an obligatory moment in the annealing procedure is not necessary scientific confirmation. Studies in humans and experimental animals no showed significant positive effect of irradiation with UV-rays for insensibility to acute respiratory infections.

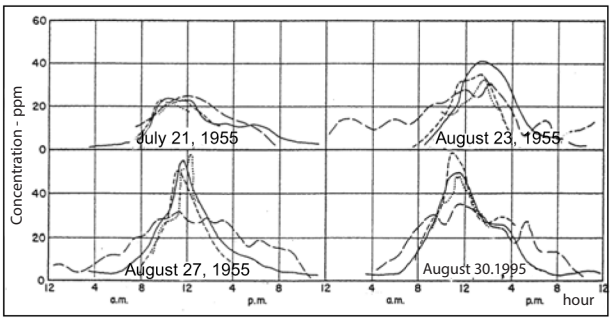

Fig. 4. - daily concentrations of ozone and oxidants in Los Angeles on various methods - at Renzetti and Romanovski (1956)

Prophylactic irradiation (in the so-called Photariums Fotarii) in miners and workers in windowless workshops during the winter season; in children and other contingents in situations with increased epidemiological risk to respiratory infections showed no significant differences with irradiated with UV-rays contingents in the incidence of diseases or resistance to cold. As far received some differences in incidence is explained primarily by reduced air microflora in rooms - bactericidal effect of UV-rays. Not recommended the use of artificial sources of UV-rays ("erythema" UV-lamps) at home for this purpose. Increased general immune defense of the body during the warm seasons (summer) is associated with a complex of factors - recreation, physical activity, nutrition, physical and chemical factors of the air environment, etc. where needed quite moderate (even lower) UV-radiation doses.

6.2.2.4. Endangered professions

Endangered professions at risk of damage by UV-rays are first all working and staying long outdoors, affecting intensive solar radiation - working in agriculture, construction, forestry and logging, open pit mining, water transport, professional sports, working in tropical regions and many others. Here it is important that not only the profession, but also prolonged exposure to the sun during the holiday periods.

Intensive UV-radiation are subjected electric welders and metallurgists in electric steel production. Also affect individuals have used UV-lasers; working in rooms with UV-bactericidal lamps (operating units, microbiological boxes); UV-sterilization of food and water; in physiotherapy departments using UV-A radiation in the treatment of certain skin diseases - herpes, psoriasis; in some laboratory equipment (spectrophotometers) and apparatus for graphic reproduction using UV-lamps.

6.2.2.5. Hygiene standards

Standards for permissible exposure to UV-radiation have been proposed by the American Confer-

Wavelength (nm)	Norm (mJ/cm²)	Relative spectral efficiency (Sλ/
200	100.0	0.03
210	40.0	0.075
220	25.0	0.12
230	16.0	0.19
240	10.0	0.30
250	7.0	0.43
254	6.0	0.5
260	4.6	0.65
270	3.0	1.0
280	3.4	0.88
290	4.7	0.64
300	10.0	0.30
305	50.0	0.06
310	200.0	0.015
315	1000.0	0.003

Tab. 2. UV-radiation - norms

ence of Industrial Hygienists (ACGIH). They do not apply to exposure outdoors or in people with photosensitive reactions. This includes persons exposed in production with artificial sources of UV-rays (without UV-lasers) and standards are based on the effects on the eye (photokeratitis) and skin (erythema) in people with normal pigmented (white) skin and repeated exposure.

According to this standard allows irradiation with UV-A-rays (320-400 nm) is not more than 1 mW/cm² for a period of 10^3 s (about 17 min), and for the shorter periods - up to 1 J/cm². At irradiation with UV-rays in the range 200-315 nm values for a period of 8 h are shown in **Table 2** - these values are used directly only by irradiation with UV-rays with a precisely defined wavelengths (eg. only 290 nm). When sources with a wide spectral range (also in this area - 200-315 nm) is determined so-called effective irradiation (Eeff), relative to the peak spectral efficiency (1.0) at a wavelength of 270 nm according to the formula:

$$Eeff = \Sigma E\lambda.\Delta\lambda.S\lambda$$

wherein: Eλ is the measured intensity in W/cm² for the corresponding wavelength or more often narrow spectral band (5-10 nm width); Sλ - relative spectral efficiency (whichever takes the highest value in spectral band with a width of 5-10 nm); Δλ - the spectral width in nm (5-10 nm). The permissible exposure (s) is obtained by dividing 0,003 J/cm² of Eeff (W/cm²). These rules do not give quite precise boundary between safe and dangerous levels of irradiation with UV-rays primarily in terms of stochastic effects (they occur in exposure to solar radiation). It is believed that the permissible irradiation with UV-radiation from the sun should be limited to the dosages needed for the production of vit D_3 (about 60 MED per year). These doses are considered sufficient for maintaining the "tolerance" of the skin to increased UV-radiation during the summer months (**Table. 3**)

Tab. 3. Protection against exposure to ultraviolet rays

The months of the year that need protection from sunlight depend on latitude	
Latitude	**Months in which sunscreen is necessary**
60⁰N до 70⁰N	From May to August
50⁰N до 60⁰N	From April to September
40⁰N до 50⁰N	From March to October
30⁰N до 40⁰N	From February to November
30⁰N до 30⁰S	All year
30⁰S до 40⁰S	From August to May
40⁰S до 50⁰S	From September to April
50⁰S до 60⁰S	From October to March

At 2010 in accordance with the European Directive 2006/25/EC on the protection of optical radiation in Bulgaria introduced regulation of UV radiation for workers in risks related to exposure to artificial optical radiation - limits for the energy dose (H) in Jm^{-2} and the density power (L) in Wm^{-2} sr^{-1} at different wavelengths (nm), and permissible of exposure (s).

6.2.2.6. Prevention of adverse effects.

Prophylaxis against UV-radiation from the **sun** include: limiting direct exposure; wearing appropriate clothing - light but maximum covering all parts of the body; use of protective (filtering UV-radiation) creams and lotions. Use of appropriate glasses. It should know, however, that reflected from the sky UV-radiation can reach 50% of total UV-radiation reaching the Earth's surface in the presence of high, feathery clouds and reflected UV-radiation from the snow reaches 85% of falling on it up to 20% of the sand, to 6% of water and only 2% of the grass (in this short UV-rays reflected better in a smaller angle of

incidence, but generally more strongly reflect more long UV-rays), i.e. by certain conditions only protection from direct UV-radiation is not always enough; also very thin textiles (cotton) and synthetic are particularly permeable to UV-rays; protective creams and lotions should not only moisturize the skin and grease, and contain UV-rays absorbing substances - ρ-aminobenzoic acid and β-carotene; glasses except dark (reduce visible light) must be made of glass (not synthetic).

In **artificial sources** defense includes engineering tools and personal protection. Sources to UV-radiation must be shielded and maximum production process automated. Use goggles, shields (protection of face and eyes), clothing and gloves. In arc welding required and ventilation (removal of ozone, nitrogen oxides and metallic aerosols), painting the walls with paint containing ZnO absorbing the reflected UV-radiation, away of other workers from the source (**Fig. 5**).

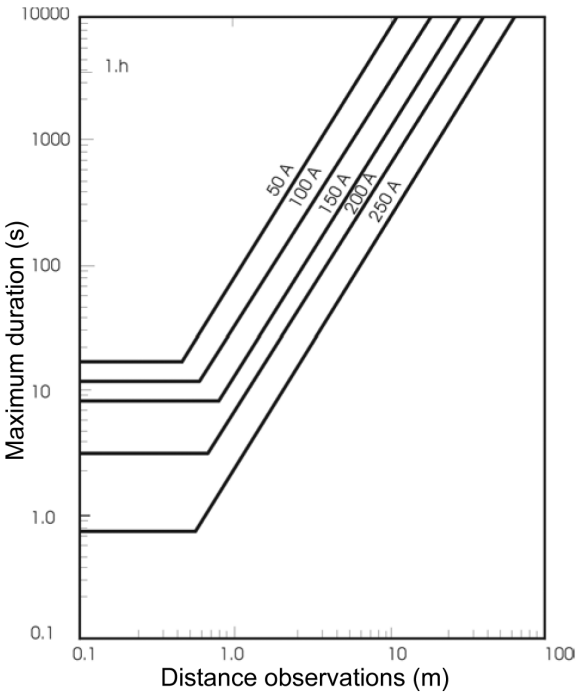

Fig. 5. - Recommended maximum exposure based on distance from arc welding (ICNIRP - International commission on Non-Ionizing Radiation Protection).

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B.Stefanov

6.2.3 RADIO-FREQUENCY ELECTROMAGNETIC FIELDS (RADIO WAVES)

6.2.3.1. General Characteristics

Radio waves are electromagnetic oscillations with a frequency of 1 kHz to 300 GHz and occupy the long wave part of the electromagnetic spectrum to the border area with infrared rays. The configuration of the radio field (RF) in the space depends on the distance from its source. Are distinguished three zones:

- **near** (area of induction) - with a radius from the source up to a distance much less than 1/6 of the wavelength;

- intermediate (interference zone) - between the near and distant;

- a distant (wave zone) - with a radius of at least 10 times greater than 1/6 of the wavelength.

In the area of induction EMF is not yet formed, the two components of the field do not match in phases and not in a specific quantity ratio. This requires in assessing the intensity of the field to determine the values of the electric and magnetic components separately. In the zone of interference is as noforming field and also formed electromagnetic waves. In the wave zone field now is formed and spread as a "running" wave.

Practical when working with sources of radio waves employees are always to near or intermediate zone. In service of sources SHF fields (microwaves) jobs are in distant (wave) zone.

The main features of radiofrequency EMFs are:

Frequency - express the number of complete oscillations per unit of time; measured in Hz, respectively kHz, MHz and GHz.

Wavelength - the distance between the two closest points of the wave with the same phase of oscillation; measured in m, cm and mm.

Intensity - at relatively low frequencies are determined separately tension of the electric field in volt/meter (V/m) and the magnetic field in ampere/meter (A/m).

Energy - Ultra-High Frequency waves (UHF, SHF) measured density of energy flow (DEF), i.e. falling energy per unit time per unit area (in W/m^2, mW/m^2 or $\mu W/m^2$).

For the purposes of medicine and biology using the classification is presented in the **Table. 1**.

6.2.3.2. Main sources of radio waves

Radio waves most widely used in various industries, transport, communications, science and medicine. The ability of EMF quickly heated material is used for heat treatment of metals (precision smelting, volumetric heating, surface treatment) of semiconductors and dielectrics. The thermal effect of EMF on the metals is determined by the eddy currents that are induced in a given environment of the magnetic component of the EMF.

High-frequency induction heating units are used in metallurgy, metal processing, mechanical engineering, in the manufacture of electronic components and others. Radiating elements of such plants are oscillating circuits, transformers, feeder lines (in ineffective shielding) and working inductors.

Heating dielectrics used the energy of the electrical component of EMF. RF generators are applicable to the drying of wood, yarn and other materials, to adhere to plastic, compression of the synthetic powders, vulcanization of rubber, and the like. In these generators the greatest radiation of the working capacitor.

Table 1: Classification of radio waves used in medicine

Range	Frequency	Wave	Wavelength
ultra-low frequencies (ULF)	under 1 кHz	Ultra-long	over 30000 km
low frequencies (LF)	1 – 100 кHz	Ultra-long	3 – 300 km
high frequencies (HF)	100 kHz-30 MHz	Long Average Short	1 – 3 km 100 – 1000 m 10 – 100 m
ultra-high frequencies (UHF)	30 – 300 MHz	Ultrashort	1 – 10 m
super-high frequencies (SHF)	300 MHz-300 GHz	decimetered centimetered millimetered	1m -10 cm 1 – 10 cm 1 – 10 mm

Radio waves are the most widely used radio, television, navigation and location, for telephone connections (mobile phones), Internet connection (radio modems) and others. Except through stationary radios, radio communications are carried out by means of transport (water, air, rail and road). Ships and aircraft mounted and radar equipment. When effectively shielding the transmitter, feeder lines and antenna switches hygiene problems do not arise. If there are defects in the shield possible radiation not only operators but also indirectly personal involved.

RF fields and used to ionize the gas to obtain plasma for flaw detection and the like. Intensive EMF emitted by the laser installations in some working areas of radio-electronic industry. Microwave energy is used in chicken, beer, dairy, meat and fish industry to heat, freesublimationze drying, defrosting, sterilization and pasteurization products. The same energy is used in microwave ovens in bits. In imperfect construction or insufficient sealing of the working chambers and waveguides possible leakage of microwave energy.

The feature EMF to heat tissue is used in physiotherapeutic practice. Apply different methods and frequencies: diathermy, darsonvalization, inductothermy, UHF therapy, microwave therapy and others.

6.2.3.3. Biological action. Clinical manifestations

The nature and severity of biological effects on exposure to radiofrequency EMF is determined by the following factors:

1) wavelength, respectively frequency of radiation;

2) Intensity of EMF;

3) Nature of field - sinusoidal, pulsed, modulated etc.;

4) Polarization of the wave;

5) Mode of radiation - continuous or by breaks;

6) Method of radiation - local, general;

7) Duration of irradiation;

8) Location man against vectors of EMF;

9) Availability of environmental factors that can modify (potentiate or weaken) the effect of radio waves.

It found that inequal conditions SHF EMF are most pronounced biological activity, followed by UHF and HF fields, ie biological effect is in direct dependence on the frequency.

In a high intensity radio waves are expressed thermal action, which is manifested in an integral increase of the temperature of the body or of its individual parts. In less impact on the field are only possible microthermal effects due to uneven heating of microstructures of heterogeneous biological tissues. The rich phenomenological material witness for expressed changes in the human and experimental animals at intensities far below the thermal effect. Namely non-thermal effect of radio waves is the starting point for its normalization.

It is believed that the main "target" on exposure to radio waves is the brain, respectively nervous system - arise reversible phase changes in the bioelectric activity, of the brain, in conditioned reflex activity, in certain biochemical reactions ensuring nerve processes.

Acute effect of radio waves is possible only in an emergency or gross violation of the rules for safe operation. It is characterized by adinamiya, feelings of anxiety, expressed vegetative and vascular disorders, diencephalic crises, episodes of paroxysmal tachycardia, nosebleeds, leukocytosis. There are reports of cataracts of the eyes and infertility.

Chronic effect of radio waves occurs mostly functional changes in the central nervous system (especially its vegetative sections) and the cardiovascular system. These may with some conditionality to unite three main syndromes: asthenic, asthenovegetative and hypothalamic (diencephalic).

Asthenic syndrome is initial. There is after work experience of 3-5 years. It is expressed by a number of subjective complaints: headache, dizziness, fatigue, irritability, reduced working capacity, general weakness, sleep and menstrual cycle disorders, lowered sexual potency and others. Vegetative changes nosharply expressed by vagotonic predominant reactions, hypotension and bradycardia. The syndrome occurs favorable, performance does not decrease significantly.

With the continuation of the work in an environment with radio waves (5-10 years of exposure) asthenia increases, join and cardiovascular disorders. It becomes astenovegetative syndrome with vegetative-vascular dysfunction (neurocirculatory dystonia). The latter may be of hypotensive or hypertensive type depending on the baseline functional status. Vascular tone is increased, observed functional angiospastic changes, are registered abnormal oscillograph, reograph, reoencephalograph and ECG curves.

An increase in the pathological phenomena (continuation work of intensive EMF) arises vegetative-vascular form of hypothalamic syndrome. It is characterized by diencephalic crises expressed agitation, tremor, sharp headache, astringent chest pain, hyperhidrosis, acrocyanosis, low-grade fever. Blood pressure is significantly increased, particularly diastolic. Electrocardiographic changes deepen, are possible conduction abnormalities, ventricular

extrasistoly and others. The changes are usually reversible. In advanced stages of the disease develop clinical picture of ischemic heart disease and hypertension. Working capacity of patients is permanently and significantly impaired.

Typical of paraclinical changes is their instability and phase nature. Predominant trend towards cytopenias - leukopenia, thrombocytopenia, relative lymphocytosis, reticulocytosis. Often defined moderate dysproteinaemias with an increased proportion of globulins, so albumin-globulin ratio decreases. Changes and quantity of mediators: histamine in the blood increases, the content of acetylcholine is changed to a lesser extent, but cholinesterase activity substantially reduced. Possible further endocrine disorders, ovarian hypofunction, impotence.

Other biological effects found primarily in experimental impacts of microwave (MW) and radio frequency waves (RF) are: death by hyperthermia (temperature of the brain 70-90°C) at very high power (described cases in humans); reduced relative weight of adrenals, pituitary and thyroid gland and secreted by these hormones; changes in the EEG, with an increase of the alpha rhythm and its amplitude; changes in the immune system-lowring of antibodies generating cells, antibody titre, disturbances in the nucleus of lymphoblasts; mutagenic activity - established chromosomal aberrations, teratogenic and embryotoxic effects in pregnant animals - increased fetal mortality and decreased fetal weight, increased postnatal mortality and altered behaviours; changes in male reproduction - decreased spermatogenesis, sperm motility and osmotic resistance and other biological effects, incl. and possible carcinogenesis. All of these effects, however, besides being observed mainly in an experiment are not enough epidemiological confirmation at people, are not always confirmed by similar studies are often used in high power - over 10 mW/cm^2, with pronounced thermal effect. In chronic exposure to the UHF radio waves is possible occurrence of cataracts. Recent studies in this direction have shown that this is possible only when the action of the radio waves at a frequency of 1.5 to 10 GHz. At frequencies around 35 GHz can be observed keratitis and damage to the stroma of the cornea.

Some authors propose to separate independent nosological unit "RF disease." This proposal is not supported by the majority of scientists mainly due to the nonspecific nature of the changes caused by radio waves. In Bulgaria professional conditioned by action of microwave radiation are considered cataracts, arterial hypertension, coronary heart disease, leukemia, cancers of the CNS.

Of course polyetiologicality of these diseases requires reporting of hereditary predisposition, constitution, age, preceding diseases, exposure and intensity of the impact and others. It is obligatory and reliable exclusion the role of others (professional and no-professional) risk factors.

6.2.3.4. Prevention of damages

To avoid damages to the health of workers applies a system of measures of a different nature: planning, organizational, technical, structural, technological, medical, biological and others.

The indispensable is **hygienic standardization**. Under Decree № 7 of 1999 in work places when operating in conditions of electromagnetic radiation of radio frequency and microwave range observed the established norms according to BS 14525-90 and BS 17137-90.

When working with low frequency electromagnetic fields requiring maximum permissible value of the intensity of the electric field does not exceed the following values (**Table. 2**):

Table 2: Hygiene standards for low frequency EMF

Frequency	Emax
from 0 to 100 Hz	25 kV/m
100 Hz to 4 kHz	2,5.10^6/f (Hz) V/m
4 kHz to 60 kHz	625 V/m

Maximum permissible value of the flux density of the magnetic field must meet the following relationship: Emax = 60/f, mT, where f is the frequency in Hz.

BS 14525-90 for high-frequency and ultra high frequency range regulates the maximum energy parameters and intensities individually to electric and magnetic field (**Table. 3**).

In the presence of VHF radio maximum permissible density of energy flow (DEF) should not exceed 1,000 μW/cm^2. This value is reduced 10 times, if the working environment is ionizing radiation, or the air temperature is above 28 °C.

The maximum permissible energy load of the body (En) for one shift is 200 μW.h/cm^2. Upon irradiation with rotating and scanning antennas is allowed up to 10 times higher value of En. The permissible exposure is determined by the formula En = DEF. T where T is the time in hours. For example, if the workplace is measured values of DEF 50 μW.h/cm^2, maximum permissible time is 200:50 = 4 h.

Prevention when working with sources of radio waves based on the principles: reducing intensity and exposure, protection by distance and screens.

Applied power source should be minimal, enough for that process. Reducing exposure seek at higher values of RF EMF to reduce the radiation dose. In norms is a possibility of working with repairs

Table 3. Standards for exposure with SHF and UHF in production

Frequency range in MHz	Maximum energy parameters (W)		Maximum intensity (E and H)	
	el. field $W_E(E^2T)$	mag. field $W_H(H^2T)$	el. field E (V/m)	mag. field H (A/m)
0,06 – 3	20000	–	500	–
3 – 30	3200	–	200	–
30 – 300	800*	–	60	–
0,06 – 10	–	200	–	50

* For rotating and scanning antennas a maximum of 10 times the value of W_E

generators and emergency to operate at higher DEF, but for a limited time and with the use of personal protective equipment. This principle of protection also includes various compensations and privileges of workers (reduced working hours, extra days off etc.).

Protection by distance is through the creation of sanitary-protection zones around the antenna equipment with application of automatic machines and robots and remote monitoring and management of processes at high intensity EMF.

The most common method of protection is shielding. It is used both to sources and to protect personnel or unprofessional exposed persons. Windows for monitoring processes must be shielded. Used tinned wire-net, spacing between the threads no more than 3 mm. Recommended command and display bodies are bring out to the screen. Particular attention requires grounding the screens. The wire should be as short and with a greater section. Otherwise, he could become a source of EMF.

Failing to reduce the intensity of the field to the standards, is obligatory use of personal protective equipment radioprotective glasses with fine metal (brass) net or from a special glass with pressed net, shields and overalls with woven metallic threads. In our country we have developed a new type of radio protective tissue on the basis of chemically metallized polyester textile with very good hygiene indicators. In local irradiation may be used gloves and aprons of metallised tissue.

Medical prophylaxis, along with hygienic normalization, includes preliminary and peridichni medical examination.

Medical contraindications for employment with sources of electromagnetic waves are:

1) Diseases of the cardiovascular system (arterial hypertension and hypotension, ischemic heart disease, angina pectoris).

2) All diseases of the blood and secondary anemia (hemoglobin less than 14 g% for men and 12 g% for females).

3) Organic diseases of the central nervous system, incl. epilepsy.

4) Expressed autonomic dysfunction.

5) Expressed asthenic condition.

6) Diseases of the endocrine glands with permanent impairment of their function.

7) Expressed neuroses (neurasthenia, hysteria, psychasthenia).

8) Cataracts.

9) Durable and expressed abnormalities in ovarian - menstrual cycle.

10) Psychiatric illness.

11) Chronic skin diseases.

Periodic reviews are conducted every 24 months. The review must take part therapist, neurologist and ophthalmologist. Make a determination of hemoglobin, red blood cells, platelets and proteinogram; in indications and ophthalmoscopy, biomicroscopy, ECG, oscillography, determination of serum electrolytes.

B.Stefanov, D. Tsvetkov

6.2.4 LOW-FREQUENCY ELECTROMAGNETIC RADIATION. MAGNETIC FIELD

6.2.4.1. General characteristic

Magnetic field (MF) is a form of existence of matter, force field, thereby realised the interaction between moving electric charges or currents. MF is a constituent of EMF. When changing value in the time always arises electric field. Self-existence is possible only if its value is constant in time. Permanent magnetic field (PMF) is created by the direct current - DC (uniformly moving charges) or by substances having properties of permanent magnet. Low-frequency electromagnetic radiation is in the lower end of the electromagnetic spectrum - frequency 0-300 Hz, mostly in alternating current (AC) - 50-60 Hz, and it forms a variable magnetic field.

The main characteristics of the magnetic field are:

1. Tension - it does not depend on the properties of the medium, and is determined by the current strength and the shape of the conductor. Its measurement unit is the SI ampere/meter (A/m). Out-of-SI unit, which occurs in the older literature is oersхted (Oe) - 1 Oe = 79,58 A/m.

2. Induction - a vector quantity that depends on the properties of the medium. The direction of the magnetic induction vector coincides with the direction of the magnetic field. It is measured in Tesla (T), endeavored unit is gauss (G) - 1 G = 10^{-4}T; 1m T = 10G.

3. Stream - physical quantity, numerically equal to the product of the magnitudes of the magnetic induction and the area perpendicular to the vector of induction. Determine units in Weber (Wb) - 1 Wb/m^2 = 1 T.

The magnetic field penetrates easily into many substances, including biological matter and is very difficult to shield. Variable magnetic fields with low frequency of great interest in view of their relationship with molecular biocurrents and health effects. The source of such fields are all appliances and systems using alternating current and the electrical lines. In contrast of the magnetic field electrical no significant penetration of the human body.[1]

[1] Man is constantly subjected to the action of the Earth's magnetic field ≈ 450 mG, changing slightly - ± 0,5 mG day due to changes in solar activity (but there is a sudden change - "Magnetic Storms"). Earth's electric field is ≈ 120-150 V/m (comparable to that of 12 kV power line), but the bad weather (thunderstorms) reaches 3-5 MV/m.

6.2.4.2. Main sources

Constant magnetic field is more prominent factor in the work environment in the manufacture of magnets, in quality control and assembly of magnetic systems of individual magnets, installation of magnetic parts (generators, DC motors, gauges and more.).

Electromagnets have the most diverse application: devices for magnetization (in preparation of permanent magnets), for fixing parts in metal machining, in cranes, magnetic separators, devices for focusing the electron beam, installations for nuclear magnetic (NMP) and electron paramagnetic resonance (EPR) and others. Magnetohydrodynamic effect upon the interaction of moving charges with MF is used to generate electricity - magnetohydrodynamic generators with superconducting cryogenic electromagnets.

Sources of the MF in production conditions are and electrolysis baths, the conductivity belts and other electrical devices, which direct current flows. Low-frequency MF are used in magnet impulsive and electro hydraulic installations.

MF is used in medical practice. Natural magnets are used relatively rarely. Artificial MF are generated by the solenoids of different shapes, which are fed with a sinusoidal current at a frequency of 50, 100 or 5000 Hz. These are eg. apparatus Biopuls (Germany) produced in our country apparatus Magnet-H-80 apparatus Pole-1 Alfa-pulsar, MAG-30 and others.

Exposure to low frequency electromagnetic fields have anywhere using electric power (AC/DC) - power lines, electric motors, electrical in household, production, education, transport and so on.. With high intensity (A/m) and induction (T) magnetic field are working with arc welding in power plants, electrical transport, maintenance of electrical lines and transformers, metallurgists in electric arc furnaces, television and radio-engineers (**Fig.1**). Working with video displays are also subjected to a low-frequency electromagnetic field, but they must be at a distance under 10 cm. to it in order to have a field strength comparable to those mentioned above.

6.2.4.3. Biological activity

Biological effects of MF are determined primarily by: a) the type of field - constant, sinusoidal, pulsed

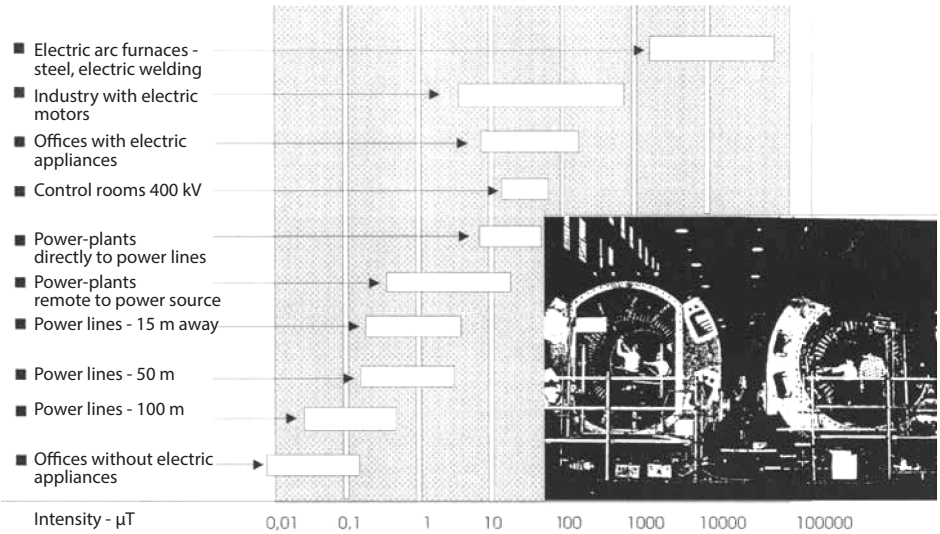

Fig. 1. Magnetic fields at the electric current (50 Hz) and steam turbine generators - 1800 and 3800 rev./min (according to H. Lundren, 1992).

or impulsed; b) the magnitude of the magnetic induction, respectively the field tension; c) the gradient of the field, respectively extent of its inhomogenous; d) the duration of the effect; e) modes of radiation - continuous or with breaks.

The first changes that precede any other reactions of the body are changes in bioelectric activity of the brain. In the initial stages of excitatory effects processes prevail. The further impact of CMF leads to a slowing of the electrical rhythms of increase in the intermediate EEG amplitudes and slow waves.

Subjective complaints in intensive impact with the MF most frequently: headache, recurrent vertigo, tinnitus, increased irritability, anxiety and worry.

Under the influence of the CMF in the body of workers observed vegetative-vascular changes, which are characterized mainly by phenomena of vasodilatation. The disorders in the various regions of the vascular system does not occur simultaneously. At first they cover arteries and arterial part of the capillary network, and then spread in the parts of venous capillaries and veins. In some of those chronic staying in an environment with MF has a tendency to arterial hypertension.

Changes in the activity of the cardiovascular system, morphological composition of blood and metabolism are usually transient, reversible and of a functional nature.

It is noted that in the body induced by the magnetic field biocurrent in persons standing in the vicinity of the power line - 5OHzAC, is much less than those induced by the electric field. In addition induced MF biocurrent significantly weaker and those caused by normal heart and brain activity - respectively 10 mA/m^2 and 1mA/m^2.

Like the biological effects of action of EMF in the radio frequency range and here reported changes do not always confirm, they are not links "dose / effect" (most messages are in action on "low-dose") does not indicate pathogenetic mechanisms explaining found changes. Indicate: the presence of persons "hypersensitivity to electricity" - established in Sweden (1993) 10 000 people with subjective symptoms and complaints, working with video display and other electrical equipment; epidemiological studies in people living near power lines (but low magnetic induction - 0.2 mT) for increased frequency of cases of leukemia in children and brain tumors; increased incidence of premature pregnancy and malformations in some working in power plants or using electric warming blankets in the night; a higher incidence of certain types of leukemia - acute myeloid leukemia and chronic lymphocytic leukemia, when operating in industries with large electric motors; have been developed and theoretical models for changes in cell membranes (transmembrane potential), explaining found reduced melatonin and permeability of the calcium ion.

Prevention. Regulations for low ER and magnetic component of EMF are listed (see Radio frequency electromagnetic fields). In our in manufacturing processes and work equipment, which are a source of constant magnetic field, magnetic induction is regulated in the workplace to 60 mT (equivalent to 600 Gs) per shift. The maximum value of the flux density of the magnetic field should not exceed 2 T regardless of exposure time

In our legislation are not regulated contraindications to work with constant magnetic fields and periodic examination of such categories of workers. (In view of the similarity in physical nature and biological activity should be regulated on the radio waves applies to the magnetic field).

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6.2.5 STATIC ELECTRICITY

6.2.5.1. General characteristics

Static electricity is a set of phenomena associated with the occurrence of electrical charges on the surface or volume of dielectrics or isolated conductive bodies. Static electricity occurs upon contact of two different materials, which becomes a redistribution of the electrons or ions them and form a double electric layer with opposite signs. Depending on how and source of formation electrostatic field can exist as their own (field of fixed charges) or stationary electric field (electric field of the DC).

Main indicators of the electrostatic field are its intensity and potential of point charges. Intensity is a ratio of the force F, with which field acts on the point charge Q, to the magnitude of this charge: E = F/Q. Unit for intensity is volt/meter (V/m), and a potential - volt (V).

6.2.5.2. Main sources

Electrostatic field on work places as a result of using any electronic-ion technology or adverse effect of a production process.

In the first case using the opportunities provided by static electricity for purification of gases (in electro filters) for electrostatic separation of ores and other materials for electrostatic application of paints, varnishes, and polymers. These technologies are widely used in chemical, grain feed, food and paper industries - for rubberise of tissues, production of plastic foils, paper, flooring textile base (linoleum), processing and production of granular materials and powders and others.

As an unintended consequence static electricity may occur in the process friction, spraying, crushing, blending, movement of liquids through pipelines and others. Generating electrostatic charges are observed in production, processing and storage of powders (bulk materials with particle size of fine powders to granules) and to electroinstallations and high voltage direct current lines.

Significant electrostatic fields arise in the textile industry in the processing of chemical fibers with high dielectric properties. The acetate and synthetic fibers are hydrophobic, have high specific resistances and load in the process of production and processing - in warp, spinning, drawing, twisting, finishing operations and others. End spinning and weaving machines is determined static field intensity of 100 kV/m.

High intensity of the electrostatic field and is registered in the processing of dried wood, which has a high dielectric constant, which makes easier its electrization occurs. This factor is typical for paper pulp industry, which are widely used materials with pronounced dielectric properties - cellulose, rosin, paraffin, wood and others. At separate technological operations are measured values of the field from 60 to 150 kV/m.

Increased level of static electricity can be established and workplaces equipped with computers. To speed the flow of electrons in the picture tube of a video monitor serves electrode, which is fed very high potential. He became a source of electrostatic field. In falling electronic beam on the luminophorous layer of the screen also create electrostatic charges. Their presence can be felt by touching the monitor screen if he has no special anti-static coating.

Source of static electricity are electrifying synthetic materials that abound modern business interior - carpets, plastic coatings, incl. and clothing (underwear and outerwear) and shoes from synthetic fabrics.

6.2.5.3. Biological action

Static electricity could have harmful effects on the human body not only in direct contact with the charges, but due to the action of the electric field that occurs around the charged surfaces.

Experimental animal studies have shown that the most sensitive to **electrostatic fields** are nervous and cardiovascular systems. There are changes in the conditional reflex activity and electrical activity of the cortex and deep structures of the brain. Upon prolonged exposure to the electric field in the animals is determined reduced reactivity of the autonomic nervous system with transient characteristic (**Table. 1**).

Working for long periods in an area with intense electrostatic fields complain of increased emotional excitability, irritability, headache, rapid fatigue, weakness, sleep disorders and appetite. It should

Tab. 1. Experiments on animals exposed to static electricity

Biological changes	Effects observed	Terms of exposure
Haematological and immunological	Inconstant changes in serum albumin and globulin fraction in rats There were no significant differences in blood cells count, serum proteins and biochemical parameters in mice	Permanent exhibition on fields between 2.8 and 19.7 kV / m from 22 to 52 days of life Exposure to 340 kV / m for 22 hours a day for a total of 5000 hours
Nervous system	Reliable EEG changes in rats, but the effect is not permanent There were no significant changes in the concentration and rate of implementation of various neurotransmitters in the brain of male rats	Exposure to electric fields with intensities up to 10 kV / m Exposure to fields with intensity 3 kV / m to 66 hours
Behavioral reactions	There were no effects on behavioral responses in rodents Occurrence of dose-dependent behavioral abnormalities in male rats, no change in the air ionization	Exposure to HVD electric fields from 55 to 80 kV / m Exposure to fields with an intensity of 12 kV / m
Reproduction and development	There were no significant differences in the total number of offspring and per cent survivors mice	Exposure to 340 kV / m for 22 hours a day before, during and after pregnancy

be noted that the static electricity presents a danger not only to health but also to life. Following the blast power of **electrostatic discharges**, there is a high probability of explosions and fires - particularly for inflammable, combustible and explosive materials in contact with metal unearthed facilities (**Table. 2**).

6.2.5.4. Prevention

Recommended that workplaces limit value of electrostatic field is 60 kV/m for 1 h. If the contact with static electricity is over 1 h, was used the formula $E = 60/\sqrt{T}$, where T - time in hours. For example, 4 h, regulates the intensity $E = 60/2 = 30$ kV/m.

The general requirements for protection from static electricity in Bulgaria are regulated in BS 15969-84 and in Regulations on labour safety and operation of electrical installations and equipment (D-01-008). Fighting electrostatic fields is carried by a number of technical, technological, structural, organizing and other events. The most important of them are:

1. Grounding electrostatic conductive parts of machines, equipment, piping, ventilation systems, tanks, etc., where generate electrostatic charges.

2. The use in some technological processes of neutralizers of static electricity, which ionize the air and thereby relax charge.

3. Use of other antistatic additives, and the coating material for reducing the specific volume and surface resistances of the materials to values corresponding to a low degree of electricity.

4. Maintain a high relative humidity in the working environment - up to 65-75%.

5. Reduce speed and turbulence of movement of liquids in pipelines.

6. Floors and floor coatings must be electroconductive;.

7. Use an alarm and (or) blocking at risk of electrostatic discharges due to high intensity electrostatic field.

8. Use personal protective equipment - anti-static shoes and more.

To prevent sparks from static electricity in hazardous areas and external systems, with belt drives must be used antistatic belts or flat belts of semiconducting material in the required coating with antistatic paste. It is prohibited flammable liquids to pour in containers by flowing stream. Rightly pouring liquid drip on the walls of the tank. For work in

Tab. 2. Typical lower limits of flammability of dusts, gases and vapors

Substances	Limit
Some powders	Some joules
Very fine sulfur and aluminum aerosols	Some joules
Vapour from hydrocarbons and other organic liquids	200 micro joules
Hydrogen and acetylene	20 micro joules
Explosives	1 micro joule

hazardous areas and environments are not allowed to use clothes made of synthetic material which can accumulate electrostatic charges.

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6.2.6 LASER

One of the most remarkable discoveries of modern times are optical quantum generators or ***laser*** - **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation.

Nikolay Basov and Alexander Prokhorov and Charles Townes (1954) first developed the physical foundations of the so called ***Maser*** (**M**icrowave **A**mplification by **S**timulated **E**mission of **R**adiation) or a generator that works in the range of microwaves. Optical maser is a laser. In 1958, Charles Townes and Arthur Schawlou offer the principle of the laser. The first ruby laser was created in 1960 by Theodore Maiman (in 1964 Nikolay Basov and Alexander Prokhorov and Charles Townes become Nobel Prize in Physics).

The lasers are quantum generators of electromagnetic waves of infrared, visible and ultraviolet range using the phenomenon of stimulated emission. In general, any laser device consists of an active medium (solid, liquid, gaseous, semiconductor, etc.), A system for excitation of the active medium with a source of energy -optical, electrical, chemical, gas dynamic, electronic and resonator device - element which makes laser of quantum amplifier in a quantum generator.

In **Table. 1** shows the main types of lasers and their characteristics.

Tab. 1.

Active medium	Wavelength	Mode of transmission	Type	Power
Ruby	694 nm	pulse	solid	100,000 W
Neodymium	1060 nm	continuous	solid	3 W
Helium-neon	632,8 nm	continuous	gas	0,2 W
Argon	488-514 nm	continuous	gas	2 W
$CO_2 + N_2 + He$	10 600 nm	continuous	gas	10 – 300 W
Gallium arsenide	840 nm	continuous	semiconducting	2 W

Pulse lasers emited energy as single or series of pulses with a duration of less than 0,25 s.

Laser radiation has several extremely important and specific properties, namely: high *monohromatism* - a very narrow range of frequencies or wavelengths, *coherence* - sinhronous in time and space emitted from the laser source light waves, high *directivity* and minimal divergence, which determines the largest concentration of energy in the section of the beam.

6.2.6.1 Use of lasers

Described characteristics of the laser beams make them widely used in industry (in the processing of high temperature stable metals and alloys, for drilling, cutting and welding under high pressure, calibration, in instrumentation, etc.), Military equipment, construction (mining, road, buildings), communication and communication systems, in various research apparatus and methods in spectrophotometry holography. Lasers are becoming more widely used in medicine - surgery, oncology, ophthalmology, dermatology, gynecology, dentistry and in bits (CD-ROM components, PC drives and CD music systems). Increasingly used and various therapeutic laser systems for conducting physiotherapy and acupuncture.

6.2.6.2. Occupational risk factors

Persons working a long time with lasers can be exposed to a complex of adverse factors in the work environment - direct, reflected or diffuse laser radiation, ultraviolet and infrared radiation, impulse noise, toxic dusts and gases separately from laser process materials. As a result of electrical discharges or ionization of the air environment around the high power laser devices, in the air of the working environment is ozone. Cooling of solid-state lasers are sometimes used liquefied nitrogen and helium that skin contact can cause burns. These refrigerant gases at their expiry in closed (unventilated) room replace oxygen and there is a risk of hypoxic damage. It is possible defeat of high voltage electricity using laser equipment. In some manufacturing operations using flammable organic solvents and materials, that can be easily ignited by the laser beam. Often lasers used in medical practice have such power characteristic, that if no observing the rules for working with lasers can cause tissue damage not only applies them medical staff but also for patients.

6.2.6.3 Classification of lasers in the degree of occupational risk.

Occupational risk assessment of laser radiation is carried out using a classification system (Decree № 9 for sanitary rules when working with lasers SG. 95 of 1986 .; American National Standards Institute - ANSI Z136.1-1986), based on the baseline characteristics of laser radiation (power features, impulse duration and frequency of repetition, duration of radiation, wavelength, etc.) and limit levels (LV) for exposure to laser radiation (Decree № 8 for hygiene standards laser radiation SG. 92 of 1986).

Since the main risk of laser radiation results from damage to the visual system and, to a lesser extent, to skin lesions, *classification* is based on the ability of a laser radiation to cause these injuries (classification of the lasers must be done by the manufacturer).

Class I belong a class of lasers and laser devices with the lowest power or some lasers emitting in fully enclosed space (fully shielded laser). They do not cause eye damage, even in direct directing the laser beam to the eye at duration maximum for type of laser (sometime unlimited).

Class II includes low power lasers and laser devices visible range. Do not cause damage to the eye by direct observation of the laser beam for a time $t < 0.25$ s.

Class III includes average power lasers and laser devices emitting in the infrared, ultraviolet or visible area. These lasers can cause ocular damage by direct entry of the beam into the eye, such as beams with certain wavelengths and can damage the skin. Lasers of this class are divided into class IIIa and IIIb class. Class IIIb lasers belong, in which the risk of causing damage to the eye is higher.

Class IV belong lasers and laser devices with high power where even diffusely reflected laser light creates a risk of eye damage. Direct laser beam causes skin lesions and create a fire hazard.

Control measurements of laser radiation are carried out mostly using laser class IIIb and IV.

6.2.6.4. Biological action

Peculiarities of the biological action of laser radiation is determined by:

- The characteristics of radiation - wavelength mode of radiation, pulse duration, energy and power of radiation;

- Properties of biological object - absorption coefficient and reflection, thermal conductivity and heat capacity, content of certain chemical compounds.

The biological action of laser radiation is associated with the following main effects: thermal, mechanical, electrical, photochemical and biostimulating.

Typical of the ***thermic effect*** is the formation of large amounts of heat (up to hundreds of degrees) in a small volume of tissue in a very short time. Increases were mainly the temperature of liquids, so suddenly increases and intracellular pressure. ***Mechanical*** or hit, the devastating effect of laser radiation is the result of kinetic and ultrasonic changes. The resulting shock wave is the result of descriptions thermal effect on cellular fluids and is most powerful when it is formed in the deep-seated tissues. ***The electric effect*** is related to the electric field that accompanies radiation and may affect influence on molecular structure and permeability of the cell membrane. ***Photochemical effect*** is the result of stimulation of photochemical reactions and the selective absorption of laser radiation by some cellular components (eg. photocoagulation of colored, light absorbent tissue). This effect is also the therapeutic use (eg. In oncology), as in the tissue prior to inject a specific photosensitizer. ***Biostimulates effect*** is typical for treatment (low energy) lasers. In these cases, laser radiation given quantum energy without pursuing destruction. The cell uses the resulting energy for the "management" of its own metabolism.

The described biological effects are the basis of the mechanisms involved in the pathogenesis of radiation-induced damage the laser. However, often the action of these radiations is not limited to these effects. To specific biological effects of laser radiation relate changes in the genetic apparatus, in enzymatic and biochemical systems, in some components of the blood (hemoglobin) and the like. The basis of specific biological action lie complex processes caused by selective absorption of electro-magnetic energy from the tissues.

Occupational exposure are possible as severe (more common), and caused by distracted, reflected rays chronic disabilities. Last expressed as functional disorders in the nervous and cardiovascular system - increased fatigue, headache, tremor of the hands and eyelids, poor self-esteem, dizziness, lability of pulse and blood pressure. Formed astheno-vegetative syndrome.

Most sensitive and susceptible to acute injury from laser radiation organ is **the eye**; relatively rare, but also are possible **skin** damage.

Damage to the cornea by laser radiation in the infrared region(1400 nm - 1 mm).

Depending on the power of laser equipment damage to the cornea can vary - from minimal, affecting only the epithelium to the massive destruction of the cornea with severe burns adjacent

conjunctiva and eyelids. The injuries are a result of absorption of energy from tears and corneal tissue fluids (thermal effect). Recovery after minimal damage can occur within 48 hours, but heavy damage causing lasting consequences.

Damage to the cornea by laser radiation in the UV region (100-400 nm)

Radiation damage by laser in the ultraviolet region is similar to that caused by other natural or artificial sources of ultraviolet radiation. Clinical symptoms include photophobia, redness and tearing. The damage caused by ultraviolet radiation was not due to thermal effects, but rather to a photochemical denaturation of proteins and other macromolecules, such as DNA and RNA.

Retinal damage from laser radiation in the visible and near infrared region (400-1400 nm)

Laser radiation within the visible region mostly pass through the cornea, the lens and the ocular media without damaging them. Almost all the energy of laser radiation reaches the retina, where he practices harmful effects. Due to the focusing power of the lens density of the energy that reaches the retina is increased, thereby it becomes possible laser radiation with a lower energy to cause localized, is often substantial damage. The clinical presentation varies - from minimal, often asymptomatic and resolved within 24 hours ophthalmoscopic disabilities, in small focal degenerative changes in the macula and paramacularic area of the retina, occurring in immediate loss of vision and retinal hemorrhages. Generally, the degree of organic and functional damages depends on the area of the lesion and its localization.

The mechanisms participated in the pathogenesis of injuries caused by laser radiation in the visible region are different, depending on the type of laser device. Discusses the role of thermal effects (by denaturation of proteins), as well as that of the mechanical and photochemical effects.

Other ocular lesions

Laser radiation in the near ultraviolet region and a near infrared region to have characteristics allowing the absorption of large amounts of energy in ocular structures, arranged between the cornea and the retina (including in the lens and the iris). Acute injuries of these structures are possible under the effect of high power laser devices. Chronic exposure to non-coherent sources of ultraviolet and infrared radiation induced cataracts. It is theoretically possible occurrence of the same pathology and by exposure to laser radiation with a similar wavelength. In real production conditions, it is unlikely chronic exposure to laser radiation without presence of personal and collective protective equipment. Chronic effects of laser radiation on the lens have not been studied.

Skin damage

Skin damage occurred at impact of laser radiation with more energy. The laser radiation is absorbed selectively by the pigment of the skin structures and therefore advantageously damaging pigmented cells. In connection with the increased absorption of laser radiation by hemoglobin possible rupture of the blood vessel with bleeding, and the development of thrombosis in small blood vessels and skin capillaries.

Laser radiation in the *ultraviolet region* in acute exposure cause photochemical damage to the skin, similar to acute sunburn. Theoretically, chronic exposure to these radiations can lead to premature aging of the skin and the increased risk of developing skin cancer.

Laser radiation in the *visible and infrared range* can cause thermal burns in acute exposure.

In production terms, particularly in respect of the rules for working with lasers, a real risk of skin damage is unlikely.

Table 2 provides a summary of eye and skin lesions caused by the laser radiation in the ultraviolet, visible and infrared regions.

Tab. 2.

Wavelength	Injuries:	
	eyes	skin
100-280 nm (UV-C)	photokeratitis	erythema, skin cancer
280-315 nm (UV-B)	photokeratitis, photo-chemical cataracts	hyperpigmentation, skin cancer, accelerated skin aging
315-400 nm (UV-A)	photokeratitis, photo-chemical cataracts	pigmentation, photosensitive reactions, skin cancer, accelerated skin aging
400-780 nm (visible)	photo chemical and thermal damage to the retina	pigmentation, photosensitive reactions, skin burns
780-1400 nm (IR-A)	cataract, retinal burns	skin burns
1400-3000 nm (IR-B)	corneal burns, cataracts	skin burns
3000 – 10^6 nm (IR-C)	corneal burns	skin burns

6.2.6.5. Prevention of laser impact

Prevention involves complex events. Developed rules and requirements for protection against laser radiation (Decree № 9 for sanitary rules when working with lasers SG. 95 of 1986).

To ensure safe operation with lasers apply safeguards based on the class of laser system - the level

of occupational risk, the specific operating conditions of the laser, environment, presence of other harmful environmental factors, path of the laser beam, constructive features of the laser device, type target and others.

Not allowed intra ray monitoring of the laser beam (laser class I) and directing the laser beam to the eye (for lasers of classes II and III). It is shielding the laser beam for laser class II, III and IV, for lasers from third class shield and the reflected rays and those from class IV is shielded completely and the path of the laser beam and remove any reflective objects and surfaces. The premises for laser devices should not be located mirrored surfaces.

Technical safety also includes the use of filters, absorbers and attenuators to the outlet of the laser to reduce power and energy, automatic locks, remote control - for lasers class IV and others. Lenses, prisms and other optical elements should have blinds and accessories for the adjustment of the radiation be fitted with protective filters with a range of absorbtion, which coincides with the length of the laser beam. On laser head and the doors of the premises must be sign for laser class, with signs warning of the danger. An important preventive measure is the formation of a controlled area marked with special signs (for lasers of classes III and IV), access to which have only specially trained persons. Staff not directly involved with the operation of the laser allow at safe distances from laser danger zone, to ensure the safety shall require the presence of light and sound signal during the radiation.

Of utmost importance is the use when working on **goggles** fitted with passport and selected based on their spectral characteristics, the optical density and the period of prevention, resistance for demolition of absorbing medium by laser beam or other environmental factors, comfort wear, ventilation and others. **Protective clothing** applies when there is danger of radiation on skin with values above limit levels for skin. Used clothing dark colored, laser radiation absorbing tissues and gloves, shields and more. Laser class IV protective materials must be fire resistant and protect from heat.

Rules and requirements for the protection of other harmful factors. Toxic substances emitted from the target, cryogenic liquids and the processing of different materials with the laser beam should not exceed the MAC by simply build appropriate local and general ventilation. Protection emitted from power radio frequency devices, electromagnetic fields, X-ray radiation or optical radiation of pulse lamps are carried out through shielding. When excessive amounts of noise is necessary to implement individual and/or collective protection.

The coloring of the premises with laser systems must be tailored to the wavelength of emitted radiation in them, to absorb them and prevents mirror reflections. The lighting in workrooms should provide the requirements of hygiene standards, except when the process having to work in a darkened room.

Medical prevention. In preliminary medical examination contraindications for work with lasers are: eye damage - reduction of visual acuity, cataracts and other diseases whose course can be aggravated when exposed to laser radiation; skin diseases, in particular malignant; organic diseases of the central nervous system; expressed neuroses and autonomic dysfunction; all blood disorders and secondary anemia; diseases of the cardiovascular system (permanent arterial hypertension, coronary heard disease, angina pectoris); endocrine diseases; pregnancy. Workers in the production and application of lasers subject to obligatory periodic medical examinations once every 12 months. They should participate therapist, neurologist and ophthalmologist. Required tests include ophthalmoscopy and determination of hemoglobin and leukocytes, and at indications - biomicroscopy, ECG, platelets and red blood cells.

Hygiene standardization. Under Decree № 8 on hygiene standards for laser radiation are established limit values (LV) of work places with the use of lasers with continuous wave and pulsed mode. Limit values apply to occupational risk assessment of the primary effects of radiation on humans and does not apply to chronic exposure of human continuous or pulsed low-intensity laser radiation for extended periods (months or years). Justify limit levels for exposure of the skin (**Table. 5**) and eyes (**Table. 3 and 4**) with continuous radiation.

Tab. 3. Limit levels in radiation monitoring - $\alpha < \alpha_o$

Wavelength, λ (μm)	Duration of radiation, t (s)	Limit levels	
0,200 – 0,302	$10^{-9} – 3.10^4$	$3 .10^{-3}$	J . cm^{-2}
0,303	$10^{-9} – 3.10^4$	$4 .10^{-3}$	J . cm^{-2}
0,304	$10^{-9} – 3.10^4$	$6 .10^{-3}$	J . cm^{-2}
0,305	$10^{-9} – 3.10^4$	$1,0.10^{-2}$	J . cm^{-2}
0,306	$10^{-9} – 3.10^4$	$1,6.10^{-2}$	J . cm^{-2}
0,307	$10^{-9} – 3.10^4$	$2,5.10^{-2}$	J . cm^{-2}
0,308	$10^{-9} – 3.10^4$	$4,0.10^{-2}$	J . cm^{-2}
0,309	$10^{-9} – 3.10^4$	$6,3.10^{-2}$	J . cm^{-2}
0,310	$10^{-9} – 3.10^4$	$1,0.10^{-1}$	J . cm^{-2}
0,311	$10^{-9} – 3.10^4$	$1,6.10^{-1}$	J . cm^{-2}
0,312	$10^{-9} – 3.10^4$	$2,5.10^{-1}$	J . cm^{-2}
0,313	$10^{-9} – 3.10^4$	$4,0.10^{-1}$	J . cm^{-2}
0,314	$10^{-9} – 3.10^4$	$6,3.10^{-1}$	J . cm^{-2}
0,315- 0,400	$10^{-9} – 10$	$0,56 t^{¼}$	J . cm^{-2}
0,315- 0,400	$10 – 10^3$	1	J . cm^{-2}
0,315- 0,400	$10^3 – 3.10^4$	10^{-3}	W . cm^{-2}
0,400- 0,700	$10^{-9} – 1,8.10^{-5}$	5.10^{-7}	J . cm^{-2}
0,400- 0,700	$1,8.10^{-5} – 10$	$1,8 t^{¾} . 10^{-3}$	J . cm^{-2}
0,400- 0,550	$10 – 10^4$	10^{-2}	J . cm^{-2}
0,550- 0,700	10 – T	$1,8 t^{¾} . 10^{-3}$	J . cm^{-2}
0,550- 0,700	$T – 10^4$	$10C_B . 10^{-3}$	J . cm^{-2}
0,400- 0,700	$10^4 – 3.10^4$	$C_B .10^{-6}$	W . cm^{-2}
0,700- 1,050	$10^{-9} – 1,8.10^{-5}$	$5C_A . 10^{-7}$	J . cm^{-2}
0,700- 1,050	$1,8.10^{-5} – 10^3$	$1,8C_A t^{¾} . 10^{-3}$	J . cm^{-2}
1,051- 1,400	$10^{-9} – 5.10^{-5}$	5.10^{-6}	J . cm^{-2}
1,051- 1,400	$5.10^{-5} – 10^3$	$9 t^{¾} . 10^{-3}$	J . cm^{-2}
0,700- 1,400	$10^3 – 3.10^4$	$320C_A .10^{-6}$	W . cm^{-2}
1,4 – 10^3	$10^{-9} – 10^{-7}$	10^{-2}	J . cm^{-2}
1,4 – 10^3	$10^{-7} – 10$	$0,56 t^{¼}$	J . cm^{-2}
1,4 – 10^3	> 10	0,1	W . cm^{-2}

Tab. 4. Limit levels eyes when irradiation with divergent laser radiation - viewing angle - α> α o

Wavelength, λ (μm)	Duration of radiation, t (s)	Limit levels	
0,200 – 0,302	$10^{-9} – 3.10^4$	$3 .10^{-3}$	J . cm^{-2}
0,303	$10^{-9} – 3.10^4$	$4 .10^{-3}$	J . cm^{-2}
0,304	$10^{-9} – 3.10^4$	$6 .10^{-3}$	J . cm^{-2}
0,305	$10^{-9} – 3.10^4$	$1,0.10^{-2}$	J . cm^{-2}
0,306	$10^{-9} – 3.10^4$	$1,6.10^{-2}$	J . cm^{-2}
0,307	$10^{-9} – 3.10^4$	$2,5.10^{-2}$	J . cm^{-2}
0,308	$10^{-9} – 3.10^4$	$4,0.10^{-2}$	J . cm^{-2}
0,309	$10^{-9} – 3.10^4$	$6,3.10^{-2}$	J . cm^{-2}
0,310	$10^{-9} – 3.10^4$	$1,0.10^{-1}$	J . cm^{-2}
0,311	$10^{-9} – 3.10^4$	$1,6.10^{-1}$	J . cm^{-2}
0,312	$10^{-9} – 3.10^4$	$2,5.10^{-1}$	J . cm^{-2}
0,313	$10^{-9} – 3.10^4$	$4,0.10^{-1}$	J . cm^{-2}
0,314	$10^{-9} – 3.10^4$	$6,3.10^{-1}$	J . cm^{-2}
0,315- 0,400	$10^{-9} – 10$	$0,56 t^{¼}$	J . cm^{-2}
0,315- 0,400	$10 – 10^3$	1	J . cm^{-2}
0,315- 0,400	$10^3 – 3.10^4$	10^{-3}	W . cm^{-2}
0,400- 0,700	$10^{-9} – 10$	$10 t^{⅓}$	J . cm^{-2} . sr^{-1}
0,400- 0,550	$10 – 10^4$	21	J . cm^{-2} . sr^{-1}
0,550- 0,700	10 – T	$3,83 t^{¾}$	J . cm^{-2} . sr^{-1}
0,550- 0,700	$T – 10^4$	$21C_B$	J . cm^{-2} . sr^{-1}
0,400- 0,700	$10^4 – 3.10^4$	$2,1C_B . 10^{-3}$	W . cm^{-2} . sr^{-1}
0,700- 1,400	$10^{-9} – 10$	$10C_A t^{⅓}$	J . cm^{-2} . sr^{-1}
0,700- 1,400	$10 – 10^3$	$3,83C_A t^{¾}$	J . cm^{-2} . sr^{-1}
0,700- 1,400	$10^3 – 3.10^4$	$0,64C_A$	W . cm^{-2} . sr^{-1}
1,4 – 10^3	$10^{-9} – 10^{-7}$	10^{-2}	J . cm^{-2}
1,4 – 10^3	$10^{-7} – 10$	$0,56 t^{¼}$	J . cm^{-2}
1,4 – 10^3	> 10	0,1	W . cm^{-2}

Tab. 5. Limit exposure levels of the skin with laser light

Wavelength, λ (μm)	Duration of radiation, t (s)	Limit levels
0,200 – 0,400	10^{-9}– 3.10^{4}	Same values as in Tables 3 and 4
0,400- 1,400	10^{-9} – 10^{-7}	$2C_A . 10^{-2}$ J . cm^{-2}
0,400- 1,400	10^{-7} – 10	$1,1C_A . t ¼$ J . cm^{-2}
0,400- 1,400	10 – 3.10^{4}	$0,2C_A$ W . cm^{-2}
1,4 – 10^{3}	10^{-9} – 3.10^{3}	Same values as in Tables 3 and 4

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6.3 AIR IONIZATION

The electrical conductivity of the air is related to the presence therein of particulate carriers of electric charges. Modern research has shown that crucial to the electric state of the air have its ions.

6.3.1. MAIN CHARACTERISTICS AND SOURCES

Under air ionization means disintegration of gases from air environment with the release of electrons from electrically neutral molecules and atoms under the influence of external factors of different nature (natural and artificial, physical and chemical), which gives electro charged particles - positive and negative.

Ionization requires the transfer of energy - energy ionization (Vi), and is expressed in electron volts (1 eV = 1,6.10^{-12} erg). The minimum energy causing ionization of a gas molecule is called the critical potential (Vil), which depends on the nature of the gas **(Table. 1)**.

Gas composition of air	Critical potential eV
Oxygen molecule	12.5
Oxygen and hydrogen atoms	13.6
Nitrogen atoms	14.5
Nitrogen molecules	15.5
Hydrogen molecules	15.6

Tab. 1. Critical potentials

The easiest process of ionization is carried out at these gas components which have the lowest energy of binding. In the atmosphere it is the molecule of oxygen.

In the ionization of air primary molecular ion the s^{-6} form stable secondary so-called light ions. Light ions carry an elementary electric charge (e_o = 1,6.10^{-10} coulomb (C)). Light ions at normal humidity represent a complex of an ionized molecule, and 4-12 polarized molecules of water. The most typical are: $H^+(H_2O)$; O^-_2 (H_2O); O^-_3 (H_2O); $OH^-(H_2O)$; H_3O^+ (H_2O); CO^-_4 (H_2O) – n ranges from 4-8. Air nitrogen does not form a stable positive ions.

The size and mobility of its ions are divided into small (light), medium (intermediate) and heavy or very slow (Langeven ions) cleave to particulate matter and so-called eitkin nuclei - **Table. 2.**

Ionizer air environment are natural and artificial origin. **Natural are:** radioactive radiation from the earth's crust; UV radiation; cosmic radiation; thunder activity; disintegration of water droplets (baloelectronic effect - i.e. hydroaero ionisation); for rapid passage of large air masses over the mountains; sand (disintegration of particulat,e matter) and air storms; some plants that produce essential oils and phytoncides. **Artificial** are highly heated metal surfaces (i.e. thermo electronic emission), with the use of ionizing radiation in various industries and in medical practice, electric motors, copiers, monitors, presses and others.

Mobility(cm/s/V/cm)	Diameter (μm)	Characteristics
3.0-0.1	0.001-0.003	**Lightweight,** great mobility, short life
0.1-0.005	0.003-0.03	**Intermediate**, slower
0.005-0.002	>0.03	**Heavy (Langevin ions)** associated with larger particles (dust)

Tab. 2. Characteristics of ions - mobility diameter

The characteristics of the ionization of air, that are important for the sanitary conditions of the air environment and its biological effects are: absolute number of ions of each sign per unit volume (cm^3); unipolar ratio (q) - the ratio of the number of positive to the number of negative ions ($q_{light} = n^+ / n^-$ and $q_{heavy} = N^+/N^-$); ion indicator of air pollution (the ratio of the number of heavy to the number of light ions); the relationship of the ionization of air with other parameters of the air environment - weather complex gaseous and particulate pollutants, antropotoxins, vegetation, ozone - ion state.

6.3.2. IONIZATION OF THE AIR ENVIRONMENT OF INDOOR AND OUTDOOR

Normally the number of ions in the ground layers of the atmosphere is about 400-500 for positive and 400 negative (according to other sources respectively 1,000 and 3,000 pairs in cm^3).

The air in the mountains is clean (free of dust and gaseous pollutants) and has improved airi-

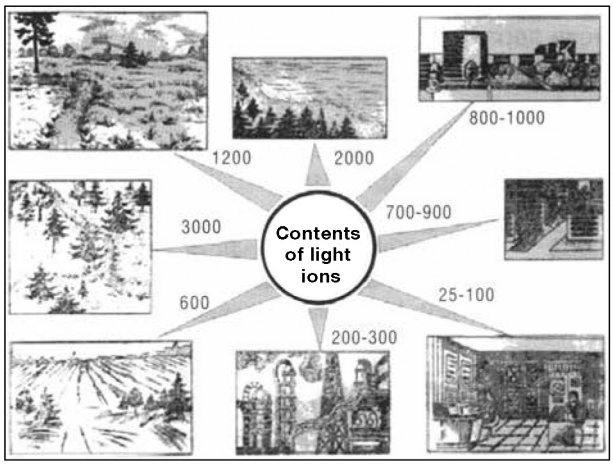

Fig. 1. Ionization of airspace

oned characteristics. In residential and especially in the industrial areas, the number of positive and heavy ions strongly increased. This is related to the availability of large amounts of water vapor and air pollutants **(Fig. 1)**. In the free atmosphere near the Earth always has an excess of positive ions and the coefficient of unipolarity is slightly above 1.0, at 1.22 to 1.5 in resort areas, but in industrial centers it may reach 4-6.

Found direct dependence on the degree of ionization of air with temperature and humidity and reverse to atmospheric pressure - reduced atmospheric pressure and increased air temperatures contribute to increase the release of radon (^{222} Rn) from Earth. Larger quantities of heavy ions in the fall, associated with increased relative humidity and lower speed of air currents (windlessness).

Light ions are more in good visibility and winds, and reduced in fog, high humidity and air currents from the mainland (in coastal regions) - except in industrial areas where the wind carries heavy ions and nuclei. Heavy ions decrease with altitude. Light ions are growing in conditions of air in high trees, particularly one that emits phytoncides (eg. siberian and white fir).

Smoking adversely affects aeroioning composition of the air. When smoked one cigarette increases the concentration of heavy ions, and decreases intermediats, with both positive and negative sign. Ion indicator of pollution increases 4.5 times.

The nature of the ionization of air in residential rooms and public buildings is significantly altered by his conditioning. Light negative ions in the rooms are twice more than in ambient air and light positive retain concentration. Filtration and humidification, however, increase the coefficient of unipolarity. The transport of the air ducts decreases the concentration of ions (ions recombination), and also increases the coefficient of unipolarity. Heavy ions in the presence of people grow 2-3 times. In windowless shops and underground sites with full recirculation of air, reduces the amount of ozone - a factor for air ionization.

In the air mainly contain light negative ions of ozone and positive - nitrogen. After the irrigation chamber of climate systems, ozone ions decreased 71 times, and nitrogen - 6.3 times. A major share of light ions in these rooms are ions of water vapor (H_2O^-, H_2O^+). In residential premises without air conditioning ozone ions also decreased - 10 to 30 times, and nitrogen (NO^+) - 3 to 5 times. Unpleasant subjective sensation at low concentrations of air ions or deionized air environment are not due only quantitative but also qualitative changes in the ionic composition (in their chemical nature) and is considered more appropriate to monitor the impact of the so-called ion-ozone mode of the premises (i.e., and the concentration of ozone).

6.3.3. BIOLOGICAL EFFECTS OF AIR IONIZATION

Biological effects under natural conditions. Epidemiological studies found that some people develop specific responses to weather conditions when ion concentration is amended sharply and significantly in the so-called "bad" winds as Sharaf (Middle East), Sirocco (North Africa), Saint An (Western USA), foehn (Europe). It was also at that time increased crime, suicides, road accidents, accidents at work - i.e. "serotonin syndrome".

Observed conditions are associated with the total increase in the concentration of ions and particularly positive 24-48 h before the advent of "bad" wind. At that time, 30% of people develop particular symptoms: nausea, migraine headaches, blood flow in the head, palpitations, anxiety, intestinal hyper-peristalsis. These symptoms are more pronounced in older people and women to form three main syndrome: serotonin excitable syndrome (increasing the concentration of serotonin); adrenaline exhausting syndrome (diminished adrenal function) and syndrome thyroid gland hyper function. In conditions of natural ecosystems, however, it is difficult to precisely determine which of environmental factors in these situations is a priority.

Biological effects in experimental studies with laboratory animals. Conducted studies have found various effects of air ionisation such as reduced mortality in mice infected with influenza virus after prolonged exposure (48-72 h) with negative light ions and back, under the action of positive ions; accelerated wound healing with negative ionization of air, also increasing the amount of total protein and alpha-globulins in blood serum; increase the

regenerative ability of the liver tissue on exposure to ionized air of experimental animals with induced carcinogenesis; decreased EEG activity during continuous action of positive ions; reduced oxygen consumption, reduced partial pressure of oxygen in the muscles and the uptake in liver tissue in the action of high concentrations bipolar air - $1,8.10^5$ cm^3 and coefficient of unipolarity 0.88; increasing concentrations of blood glucose, pyruvate and lactate in blood serum in the effect of positive ions at high concentration - 5.10^5 cm^3 and the like.

Biological effects in experiments with volunteers. Surveys were conducted in varying degrees of air ionization (ions from one sign; doses - optimal, high and ultrahigh; deionized air), created in special chambers with options for dosing and thermal comfort.

It has been found that de-ionised air leads to fluctuations in titer of lysozyme and diminished of the phagocytic activity of neutrophils in saliva, changes in hemogram.

Was established antagonism in effects of ions in physical activity - in positive increases oxygen debt, pulse rate, systolic pressure. The continued impact of positive ions is adversely affects the self confidence and performance.

When studying subjective reactions to negative ionization, back establishes that supports relaxation of the body - reduces fatigue and improves mental state (according to some authors "causes mental and emotional upsurge "). Saturation of the air with negative ions reduces complaints of shortness of breath, headache, heaviness in the heart area, improving quality of attention, visual-motor response - a total mental activity.

In real working conditions among staff to monitor complaints of headache and temperature sensitivity exacerbated by the impact of positive ions. In rooms with an air conditioning describes increased irritability, nervousness, headache, somnolence, often nasal catarrh, etc. disorders which may be associated with the shortage of negative ions in the air. Saturation of the air with negative ions, back reduces complaints of headache, shortness of breath and weight as well as reducing the number of mistakes at work.

In microelectronics high psycho-sensory tension combined with static microclimate and ionized air, creating conditions adversely affects the functional state of the nervous and cardiovascular system. In compensate aeroionic deficiency is achieved balance between the two parts of the higher nervous activity and the cardiovascular system expands the functional reserves. Increased incidence of working in radioelectronics associated with aeroionic hunger ("clean" rooms). In conducting bipolar ionization (up to $5\text{-}6.10^3$ pairs cm^3) subjective symptoms and functional changes are less pronounced and morbidity is reduced.

Effects observed in unipolar ionization can be explained by changes in natural immunity; in strengthening bactericidal (barrier) function of the skin; reduction in air microflora.

Aeroionization and air quality protection. Aeroionic state of air has a certain relation to microbial characterization of the air environment. It is known that air ions possess bacteriostatic and bactericidal effect - with the use of artificial ionization in weaving sheds the number of microorganisms is 2.5 times smaller and 2-3 times lowered the number of staphylococci, streptococci and fungal microflora.

Bipolar ionization - $5\text{-}6.10^3$ cm^3 and q-1,2-0,8, lead to changes in gas composition of indoor air electronics - ozone and nitrogen oxides exceed the MAC. Artificial ionization, however, in rooms with dust-separating and inadequate ventilation (extraction of the dust), leads to a 50-100 rate increase of the suspended substances in the air, thereby increasing significantly the dose dust entering the body. Also artificial ionization increases the toxic properties of the substances.

Mechanism of action of air ions. Roads for penetration of air ions in the human body are inhalation and dermal. In lung, besides gas and water exchange takes place and electro exchange of electric charges. This connects the primary effect of air ions - changes in the electrical properties of the blood, and the secondary action is explained by transport of electro charged colloidal systems to tissues. Along with this electrohumoral mechanism exists and neuro-reflex. Electric load bearing gas atoms irritate the nerve receptors and nerve impulses reaching the centers in the cerebral cortex form appropriate biological response. Other studies biotropic effect of ions lies in the effects of electromagnetic impulses arising from the irritation of the skin receptors and interoreceptors of lung. In general accept is a positive effect of negative ions, but it still needs further studies.

6.3.4. PREVENTION OF ADVERSE EFFECTS.

Aeroionic assessment of the state of air environment, hygiene standardization under natural atmospheric conditions or conditions of the working environment at this stage is difficult, because the results of the studies were not sufficiently substantiated yet. For norms matter: the determination of the minimum and maximum allowable limits of air ions dosage; unipolar ratio; length of sessions and the rate of application in use for treatment; type of regimen - static or dynamic.

As a biological unit for ionization of air is recommended 8.10^9 negative ions, ie the number of ions that adult takes per day. Other recommended norms (optimal and acceptable) are light positive ions respectively - $0,5.10^3/cm^3$ to $6.10^3/cm^3$ and negative light - $0,6.10^3/cm^3$ to $6.10^3/cm^3$, with a coefficient of unipolarity from 0.8 to 1.2.

Russian standards for manufacturing premises using sources ionizing air (X-ray rooms, departments for physical therapy) are: minimum - a positive 400/cm^3, the negative - 600; optimal respectively 1500 and 3000/cm^3 and maximum - to 8000 and 50000/cm^3. In cases with concentrations around and above the exposure necessary control the content of ozone and nitrogen oxides.

6.3.5. ARTIFICIAL IONIZATION OF AIR. ION GENERATORS.

Artificial ionization of air **improves the quality of air environment** - oxidation of organic matter, reducing suspended matter (dust and microorganisms). It indicates that the artificial ionization in administrative buildings reduces the dust to 3.5 times and bacterial contamination - 2-3 times. In production premises, however, high dust and other chemical factors (gases and vapors), artificial ionization of air counterproductive, adverse effect.

Artificial ionization of air is also a means to **increase efficiency** and immunological reactivity of the organism. It is believed that short-term treatment with negative ions, followed by a short positive has a more favorable effect than the continuous influence of negative ions only, i.e. it is advisable dynamic aeroionic regime, creating conditions close to natural.

Studies have shown that ionization of air and has a **therapeutic effect** - favorably influenced suffering from cardiovascular diseases, allergic and immune diseases, with neurotic conditions, dyslipidemias. Positive results were obtained in the treatment of tuberculosis, asthma, hypertension, certain types of sinusitis, diseases of endocrine glands, skin diseases of hard healing wounds and burns, silicosis, vibration disease. With regard to the number and duration of the sessions is generally recommended: duration of one session of 10 to 30 min, with a total dose (for medicinal purposes) from 75 to 180 billion negative ions and duration of the course of 15 to 25 days.

There are various **air ions generators** using different principles - sealed sources of ionizing radiation (eg. tritium - alpha rays)*; baloefekt (hidroaeroionizatsion); UV-rays; thermoelectric effect; high-voltage electric current (50/60 Hz). The latter are the most prevalent. It is necessary to them using multiple electrodes (cathodes) and a lower voltage - up to 8-10 kV, since the one electrode and the higher voltages increases much emission of ozone and nitrogen oxides. Also for even distribution of air ions in the room is needed fan to generator and proper disposal of the unit - the ceiling, walls or other open space.

* Aeroionizatori using sealed sources of ionizing radiation are banned in most countries.

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6.4 NOISE

6.4.1. GENERAL CONDITION, BASIC CHARACTERISTICS AND SOURCES

Objectively noise is a chaotic set of sounds, devoid of musical properties, but the sounds are mechanical vibrations of elastic medium, physical form of motion of the wave due to movement of particles in the elastic medium (usually air). Noise is a subjective sensory perception of the central nervous system caused by unwanted and unpleasant sounds. According to the generally accepted definition of noise is regarded as an acoustic phenomenon (sound) that causes auditory sensation evaluated as unpleasant and adverse health. Under normal conditions, the sound accompanying most human activities is a characteristic feature of life and is one of the most effective signal systems of the physical environment surrounding the man. Continued absence of sound, for example soundproofed chamber, causes disorientation in time and space and psycho-emotional disorders.

Noise is not a new risk factor for health. It is indicated from B. Ramazzini in "De morbis artificum diatriba" (1700) in copper mining, the cause of "hard of hearing" at miner reaching for continuous operation even to deafness. Before the Industrial Revolution, however, relatively few workers impacted by intense noise.

Currently, few industrial areas no less or more pronounced noise. Values apply risk of noise damage exists in the mining industry, mechanical engineering, metalworking, metallurgy, textile industry, transport, logging and wood processing, chemical industry, mechanized tillage **(Fig. 1)**. Indicate that now the US 1/3 of workers in the manufacturing and services impacted by noise levels of over 85 dB/A and more than 8 million workers suffer from occupational hearing loss, which would require more than 20 bln. dollars compensation for them.

Physiological and pathophysiological responses of the body to manufacturing noise is determined by its physical parameters (sound pressure level, character, etc.) and individual characteristics of people (age, sex, psycho-emotional status, intellectual and cultural level). Auditory perception increasing logarithmically of increasing the sound pressure, ie **stimulus intensity** (Weber-Fechner law), so as a measurement unit used relative intensity unit - decibel (dB). Decibel is a unit that reflects the logarithm (decimal logarithm) the ratio of the measured to the reference amount of sound energy - W or pressure - Pa. The scale covering 0-140 dB. Increase by 3 dB means a doubling of sound energy: the 0 dB corresponds to the threshold of normal hearing at a frequency tone 1000-2000 Hz, and 140 dB is the threshold of pain. Minimum change the intensity of the noise that the human ear perceives is about 1 dB. The slight rustle of the trees is about 20 dB, calm speech - about 40-50 dB, transport noise - from 75 to 80 dB, the metal press or riveting machines reach 100 to 115 dB, and pneumatic tools - up to 130-140 dB level sound pressure.

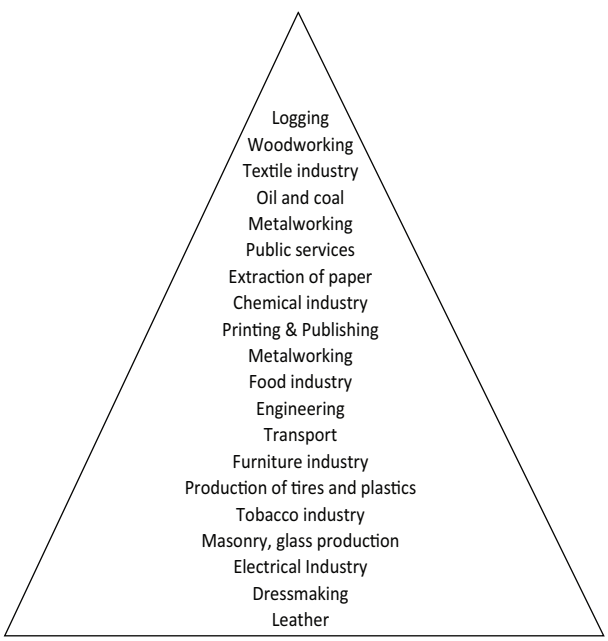
In certain dimension of sound pressure severity of the response of the auditory organ varies depending on the **frequency of sound**. According to the dominant sound pressure level of a frequency band noise conditionally divided into: **low frequency** to 400 Hz; **mid** - from 400 to 800 Hz and a **high frequency** - over 800 Hz. The sensitivity is at a maximum in the frequency range of 1000-4000 hertz (Hz). The frequency range determines the noise conditioned **irritation**. The more high-frequency noise is, the more perceived as unpleasant and faster damaging auditory sensation. The narrow band or pure tones (single frequency) are also more harmful to hearing than the broad band noise. The lowest hearing threshold at a frequency of 1000 Hz.

Depending on the change in intensity over time **noises are *permanent (stable) and no permanent (unstable)***. Permanent noises are those whose temporal change of intensity (sound pressure level) is less than 5 dB/A. In same conditions intermittent noises are more harmful than permanent. Intermitent noises in turn can be: ***impulsed*** - noise, composed of one or several sound pulses, each lasting up to 200 ms, intensity above the background - more than 6 dB, and intervals - more than 10 ms; ***discontinuous*** - noise alternating sudden drop in level to the background noise and the duration of the intervals is greater than 1 s; ***fluctuating*** - noise with continuously variable level - differences over 5 dB, and others (**"white" noise, broadband noise, tonal noise, etc.**).

Hearing-related injuries are parallel to the intensity and length of exposure of the noise. The inten-

sity of the noise, spectrum and its noise exposure varies too much during the work shift, which is why a realistic assessment of the risk of disability, applies dose method or so-called equivalent noise level, ie sound energy level relative to 8 h impact. The adequacy of dose approach is proven mainly for hearing loss. Reactions to noise depends, however, and its information content - pleasant or unpleasant, expected or unexpected, conditions and nature of work (mainly psycho-sensorial or not).

Fig. 1. Graduated in descending row industries with excessive noise



6.4.2. SPECIFIC - HEARING, EFFECTS OF NOISE

Hearing impairment due to noise exposure at work is one of the most important occupational diseases. The effects of noise impact on auditory organ may be physiological and pathological. Among the physiological *effect is masking noise* or difficult to distinguish sounds (music, speech, telephone, sound signals) in the presence of background noise. The stronger the masking effect at the same intensity noise in coincidence or near the frequency composition of noise and that of the useful acoustic signals (eg. speech - 300-800 Hz).

Auditory fatigue is a transient decrease in the activity of auditory receptor as a result of non-continuous noise impact. The degree of hearing threshold increase is more pronounced at higher intensity noise, higher frequency and more prolonged exposure. It occurs in the first 3-4 weeks of service in a noisy environment and results in hearing loss, tinnitus, whistling, feeling occupancy ears and perception of the sounds as more unpleasant (higher and stronger) . After the rest auditory fatigue disappears. The recovery of hearing becomes advantageously from 1 to 24 hours after impact (but can continue in a weaker degree and up to 1 week).

Some authors indicate the **acoustic adaptation** - decrease in hearing threshold by up to 10 dB, and recovery to 10 min after inreruption of noise impact.

Prolonged exposure to high noise levels occurs so-called professional ear hearing hard - ***professional noise damage*** to hearing function in which there are three stages. Hearing loss is sensoneural, bilateral. Concern is the sensory hair cell (cellulae pilosae), located on the basal membrane of organ of Corti. The initial stage is characterized by slight but definitive lesions of sensory cells. Localization of hearing impairment in the area of high frequencies, especially around 4-6 kHz and is not noticed by the infured (in some cases violate the possibility of differentiation of consonants, especially in the presence of background noise). In a continuation of the noise impact occurs manifest stage with deepening and broadening of hearing impairment - beginning to 2000, then at 1000 and finally to 500 Hz. If the person is not removed from noisy environments may develop late-stage practical and complete deafness (hearing loss - 60-90 dB and more).

Although acute overstimulation of acoustic energy can cause a short time irreversible hearing loss, usually in the production noise-induced deafness is related to duration of exposure. Permanent hearing deficiency occurs in the acoustic levels exceeding 85 dB, for a longer period - depending on the sound level **(Fig. 2)**.

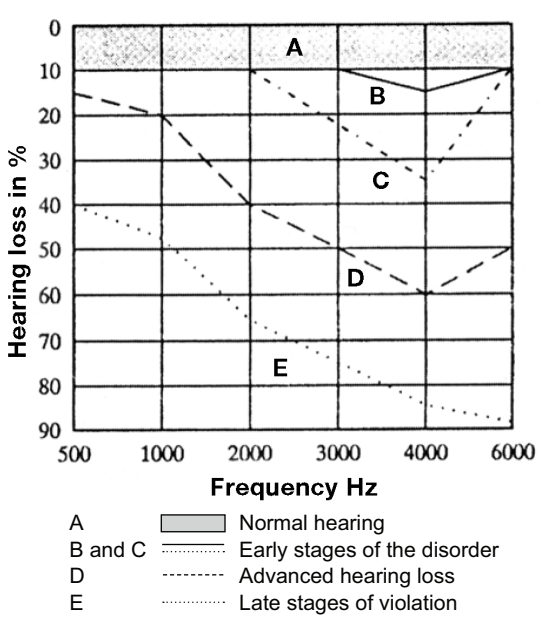


Fig. 2. Audiogram characterizing different stages of hearing loss in noise impact

Tinnitus is a condition that often accompanies professionally conditioned temporary or permanent hearing loss. Described as mild to strong ringing in the ears. Reflects irritation of sensory cells of the inner ear and often precedes hearing loss. Causes irritability, anxiety, sleep disturbances, and is more common in impulse noise.

May and ***acute acoustic trauma*** that occurs when a sudden impact of very intense noise exceeding the threshold of pain (explosion, shooting with powerful weapons and others) on unprotected ears. It is characterized by acute pain and ringing in the ears, hearing loss, occurs occasionally rupture of the tympanic membrane, lesions of the auditory bones in the middle ear, less damage organ of Corti.

Synergies of noise with infrasound and vibration augment auditory effects of noise.

Many believe that any prior damage to the middle ear predisposes to hearing damage from noise. Previous damage to the middle ear, lesions in receptor system, otosclerosis, prior treatment with ototoxic medications - antibiotics, etc., or postcomotio cerebro astenic syndrome, increase the vulnerability of hearing. Some endogenous (uremia, uricemia) and exogenous (with carbon monoxide, carbon disulfide, lead, organic solvents - toluene, xylene, benzene, etc.) intoxications reinforce individual sensitivity to noise. Vulnerability of hearing is significantly higher after 40 years of age.

6.4.3. NONSPECIFIC - EXTRAAURAL, EFFECTS OF NOISE

Violations in the *nervous, cardiovascular, endocrine* and other systems due stressful nature of noise form nonspecific or extraaural its effects, giving rise to Andreeva - Galanina E.Ts. in 1957 to introduce the concept of **"noise disease"** including aural and extraaural effects.

The impact of noise on the central nervous system (CNS) may cause disturbances in *psycho-motor area* (as a rule at intense and very intense noise) and general disturbances in the *mental sphere*. The first result of impaired functional balance in CNS and express themselves in difficulty simultaneously perform mental and motor activities, imprecision of motor actions, no economical spending of energy, changes in facial expression, laughter, speech and gestures. To general disruption in mental sphere refer insufficient alertness, reduced willingness and initiative, feelings of insecurity and discomfort, difficult recovery after work, sleep disorders, agitation and intolerance against others, characteristic and intellectual changes that some call "noise psychopathy ". Establish tendon and supraosteal hyper reflection of functional type.

In very intense noise possible vertigo events due to *dysfunction of the vestibular system* with transient dizziness and gait disorder, originating a threat to individual and collective security. The intense noise causes a narrowing of the field of vision, loss of vision in darkness and in stereoscopic vision, spasm of the branches of a.centralis retinae. Established higher rates of *gastritis, anorexia and pylorospasm* in systemic exposure to intense noise, as a manifestation of *autonomic dysfunction*.

Noise *causes increased secretion of adrenaline and aldosterone, changes in electrolytes* with a credible increase in serum calcium and magnesium and an increasing trend of serum phosphorus. And increases excretion of magnesium and phosphate.

Excess noise increases *cardiovascular risk*. Predominant opinion of a positive correlation between hypertension and noise exposure. Reliable increased serum cholesterol and triglycerides. They have been described in experimental studies, immunological changes, and embryotropic action of intense noise.

The noise worsened qualitative and quantitative indicators of the labour activity.

In recent years (1999), some authors describe ***vibroacoustic disease*** with a variety of pathological changes in flight technicians and aeronaut exposed to noise intensity 90 dB, and low frequencies - below 500 Hz, and general vibrations.

6.4.4. PREVENTION OF ADVERSE NOISE EFFECTS

Guidelines for the prevention of adverse effects of noise include: programs for noise control through engineering and administrative measures and personal protective equipment; conservation programs for hearing and extraaural effects with focus on prevention. Consideration of the actual situation, usually requires the implementation of comprehensive preventive measures.

Programs for noise control. *Engineering noise control methods* include precise localization of noise sources, determine the quantitative parameters of noise and analyzing its spread. The most rational is necessary a noise effect can be provided by **reducing** the noise formation. Easily applicable and cost-effective measures such as: regular and ongoing overhaul and maintenance of noisy machines, aggregates and instruments; replacement of outdated equipment; grinding and lubrication of the friction parts; maintenance of vehicles and their silencing devices; precision static and dynamic balancing machines and engines; replacement of worn, unbalanced parts, etc.

Due to technical features and defects in the machinery and equipment mentioned current measures sometimes are uneffective. Then to reduce the intensity of noise apply changes in engineering design through major construction or simple solutions. With good results have been the replacement of metal parts with plastic, textolite, wood and the like. Effectively reducing the clearances between mating parts by materials that increase the resistance and weaken vibration. Thus air-cushion of vibration be increased by coating the vibrating surfaces with material having high friction such as rubber, resinous substances, felt and the like. Suitably, the replacement of large and slow to smaller and faster machines, hydraulic to mechanical press, etc.

Results were obtained by changing the processes in a manner which eliminates the blow (welding instead hammer, compression rather than stamping, unshuttle weaving machines, and so on) or reduce the number and power of the blows.

Limiting noise is possible by **absorbing** (acoustic) materials and structures such as mineral wool, some highlyporous materials, swelled perlite, ceramist, vermiculite, frothy fabrics of high-molecular compounds, soft porous woody-fiber boards and others.

Sound absorbing structures are combinations of sound absorbing material and protective and decorative coatings - perforated gypsum, plywood, woody-fiber, plastic, metal and other plates or panels. The lining of the premises with acoustic material is economical and effective method primarily with respect to high-frequency noises.

The noise emitted by pneumatic tools, compressors, blowers and the like, can be **isolated** with silencers - active and reactive. Complicated reactive silencers based on the principle of the acoustic filter, are more effective against low-frequency noise. Active filters absorb sound energy with them effectively lower high-frequency component of the noise. The most suitable tubular mufflers that have little aerodynamic resistance. Important is the significance of vibro isolation of compressors, engines and other units by placing them on shock absorbers (steel springs or layers of rubber, felt, etc.), and stable base.

Inhibit the spread of noise is achieved by **reflection** of sound waves from different partitions. For this purpose, so-called encapsulation with acoustic hoods on noise sources or using partitions, screens, shields and so on. For sources with high sound power encapsulation is often the only effective means of suppressing noise. Encapsulation is made easier with turbines, motors, compressors, diesel engines, fans and other equipment, which are automated or remote controlled, but is difficult in presses, hammers, punches, etc., requiring interrupted or continuous manual operation.

It is the encapsulation of various lightweight construction detachable and portable, where sound isolation combined with sound absorption through acoustic treatment of the inner surface of the structure.

Another type of sound barrier between the source and the worker are **screens, portable semi-fixed booths, soundproofed booths for monitoring and remote control** of technological processes.

There have been studies on the use and so-called interference noise reduction by microphone to perceive noise, amplifier, phase inverter and speaker. Sound emitted from speaker reverse of the 180° phase creates a "zone of silence". It is also possible acoustic noise reduction using placed in the airspace bodies of different geometric shape.

Suitable **planning, urbanization and architecture to prevent** the spread of noise to less noisy rooms through rational planning of the production building. Creation of premises soundproofing is greater as a massive wall and higher frequency of noise. This law for the mass can be changed in order to improve fuel economy by a double wall with an

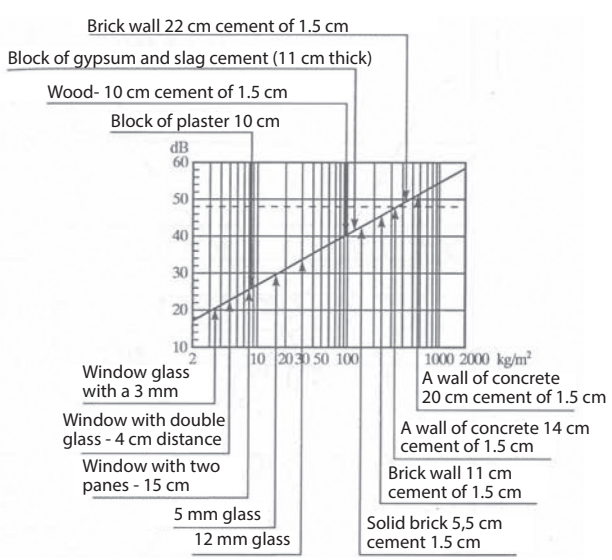


Fig. 3. Noise reduction various by building materials and structures. Law of the mass.

air space between the two walls **(Fig. 3)**.

Noise from infactory transportation may be limited by a ban on audible signals, landscaping and asphalting the territory, construction of highways parallel to the factory walls - barriers, earth mounds and others.

The program for noise control covers a number of *administrative measures* to shift work, alternating types of activities with a view to reducing individual

noise exposure time. It is the reduction of the duration of noise exposure and/or transforming it from continuing in intermittent by shortening the periods of noise impact, extension and/or increasing the number of breaks, periodically passing to operation unrelated to the noise impact. It is recommended that breaks take place in a quiet room with a comfortable microclimate. Our legislation provides for an additional six-day leave at work in conditions of excessive noise in continuation more than four hours a day. American authors propose a scheme for the duration and number of periods of rest in a quiet room to periods of noise with varying intensity

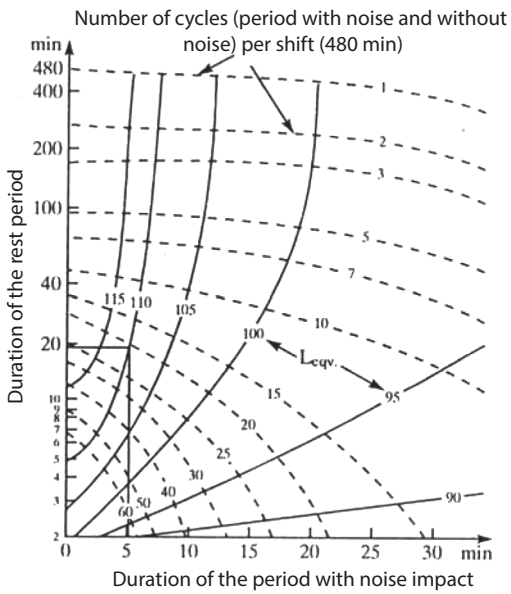


Fig. 4. Scheme of the duration and number of periods of intense noise and rest during the work shift (in Desoille H., J. Scherrer, R. Truhaut - 1991)

throughout the work shift - 480 min **(Fig. 4)**.

Without a generally accepted, opinion exists for beneficial effects on mental state of low-frequency music with less intensity. Auditory stimuli to soften by proportional rhythmic contrast of color combinations of interior.

Individual protection. For protection from the adverse effects of intense noise apply different types of antiphons.

The choice and use of hearing protection are determined primarily by the intensity, frequency spectrum and nature of the acting noise, the duration of noise exposure, microclimate conditions of the working environment and the need to maintain verbal and other acoustic communication. To justify the production and use on their merits, hearing protection must satisfy a number of requirements, basic of which are: to descend into optimum noise **(Fig. 5)**; their use does not lead to a rapid sweating, maceration and infection of the external auditory canal; not to impede speech and other signal-acoustic communication of workers between them and the environment; do not cause foreign body sensation and painful pressure in the ear; they are made of easy to clean, without irritating properties materials; shape and structure them to comply with the morphological characteristics of the ear and skull, maximizing

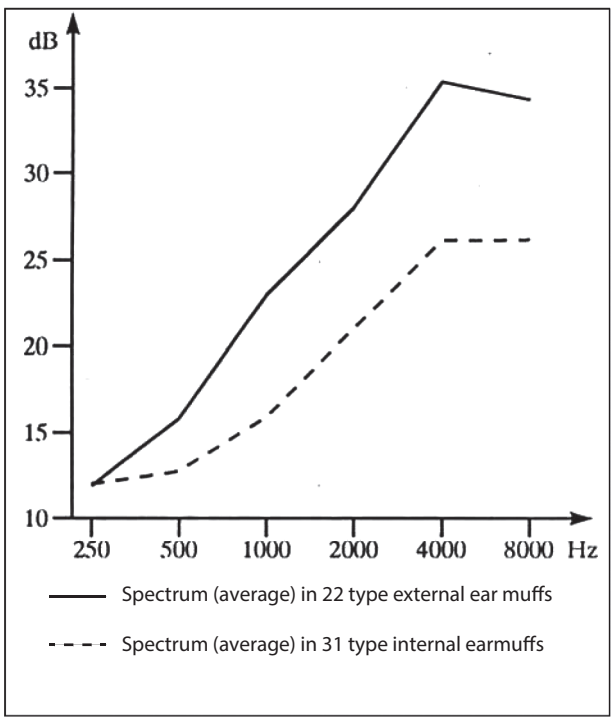


Fig. 5. Loss of noise in internal and external ear muffs

adherence to them.

In practice, use internal and external ear muffs most varied form, structure and efficiency. The **inner ear muffs** (bushings, obturators) are made mostly of plastics or rubber, but can be successfully used those made of cotton, mixed with wax, sponge and others. Good adhesion of ear sleeves sometimes is more important than the quality of material used.

The **outer ear muffs** are made in the form of earphones, headset and helmets. Earphones only reduce air conduction noise and like bushings, lower mainly high frequency components. Apply at high intensity noise (over 100 dB) and in cases where the bushes are underperforming or hardly usable because of ear infections, decubitus and itching in the ears.

In the more intense noises requires the use of **headset or helmets** that prevent both air and bone conduction noise, but they are expensive, heavy and bulky.

To overcome resistance to the use of hearing protection, often associated with an underestimation of the risk of noise effects are required individualized preparation and training for proper use. Individual tools are an important part of prevention programs

for noise disabilities, in many cases they are only effective mean, but they should not replace technical solutions to noise sources.

The program for the conservation of hearing and extraaural noise effects include: evaluation of the worker exposure - average daily, weekly and general; profpathological assessment of the risk, of setting maximum noise levels for a certain period of time; hearing protection; educational programs for staff. Sparing hearing programs apply to all workers in noise over 85 dB/A, used accordinaly noise characteristics. The intervention includes multicomponent educational strategies and features of storing industrial strategy.

Control at noisy environment. Only on the basis of systematic analysis of the parameters of the noise made by means of precision instrumentation and detailed knowledge of the origins and causes of the emergence and spread adequately possible decision on measures to lessen the noise, the frequency of prophylactic examinations and selection of personal protectors. Dose investigations with the use of individual dosimeters precise data on noise loading.

Risk assessment for hearing loss induced by production noise offers standard ISO 1999 (1990), who relate the effect of the intensity of the noise and the total time of exposure. It is assumed that equivalent (about 8 h) noise level of 85 dB/A and less, will no damage auditory sensitivity at 95% of influenced persons for period of impact over 30 years (whole service). BS 14478-82 allowed noise at work places also up to 85 dB/A (equivalent level).

Since 2006 (Decree № 6 of MH and MLSP) the minimum requirements for ensuring the health and safety of workers from the risks related to exposure to noise as hygiene standards is introduced concepts exposure limit values and exposure limits.

Exposure limit values is $L_{ex.8h}$ = 87 dB/A at momentary peak sound pressure (P peak) - 200 Pa or 140 dB/A.

Upper exposure limits for action is $L_{ex.8h}$ = 85 dB/A at P peak - 140 Pa or 137 dB/A.

Lower exposure limits for action is $L_{ex.8h}$ = 80 dB/A at P peak - 112 Pa or 135 dB/A.

Upon exposure limits actual exposure of workers report with noise reduction used personal protective equipment, ie allowed in noisy proceedings **in mandatory use** of PPE impact of noise intensity $L_{ex.8h}$ = 87 dB/A.

Exposure to noise greater than $L_{ex.8h}$ = 80 dB/A, workers also **provide** personal protective equipment, while Lex.8h = 85 dB/A PPE now they **should be used**.

It is not permissible noise exposure above the limit values (87 dB/A). Only in exceptional cases where the nature of work, the use of PPE may cause greater risk to health and safety, they can no be used.

These normative values and BS/ISO 1999-2004 concerning the impact of noise on hearing sensitivity and appropriate prophylaxis.

In business activities related to greater stress - mental work, the greater is the importance of noise as a factor inducing a state of psychic discomfort, decreased work capacity and leading quickly to fatigue. In Decree № 6 MH and MEW in 2006, for indicators of environmental noise, taking into account the degree of discomfort indicates that limits noise - equivalent level for consulting rooms in hospitals are 40 dB/A during the day and 35 dB/A during the night; in classrooms and auditoriums in schools, reading rooms, facilities for research - 40 dB/A. (day and night); workplaces in office buildings - 50 dB/A (day and night). Here at the impact of tonal and impulsive noise values are reduced by 5 dB/A. Also states that production and storage areas and zones limits are 70 dB/A (day and night).

Medical control. In our country these contraindications apply to work in conditions of intense noise: permanent hearing loss regardless of etiology; otosclerosis and other chronic diseases of the ear with apparently poor prognosis; expressed disturbances in the function of the vestibular apparatus irrespective of the genesis; expressed autonomic dysfunction; organic diseases of the CNS, including epilepsy; neuritis and polyneuritis; mental illness and psychopathia; diseases of the cardiovascular system (hypertension, coronary heart disease, angina pectoris and hypotension); ulcers of the stomach and duodenum in the acute stage. In conducting audiometric testing to establish the state of auditory adaptation. The more pronounced is the increase in the auditory threshold after noise loading (direct adaptation), the higher the resistance to noise; more convincing indicator however, for resistance to noise is readaptation (recovery time of hearing threshold to baseline).

Mandatory screening of risk contingents aimed at early diagnosis of noise disability. In Bulgaria was adopted in carrying out their compulsory participation ENT specialist and a neurologist, at indications - and therapist and gynecologist. In excess of the limit of not more than 20 dB is conducted every 24 months, and over 20 dB - 12 months in compulsory audiometric testing and electrocardiogram - at indication. Audiometric changes in hearing threshold over -15 dB impose measures to prevent further deterioration of auditory function, adequate therapeutic behaviour and monitoring of audiometric status of the worker.

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N.Mitrev

6.5 INFRASOUND

A powerful subsonic wave generated by Perbuatan volcano on the island of Krakatoa was described in 1883. Later, during First World War were created infrasound detectors guns. More systematic research in the 30 years of the twentieth century to infrasound phenomena relate to their many natural sources - earthquakes, volcanic eruptions, hurricanes, sea waves, storms and strong winds, waterfalls, landslides avalanches, demolitions, blowing in the wind buildings, trees, bridges, etc.

Objectively infrasound and noise have the same physical nature with different frequency response. Infrasound represents mechanical oscillations and waves of elastic medium in the frequency range between 0.1 and 20 Hz, which is lower than the minimum audible frequencies perceived by the human ear as an acoustic signal. The features of infrasound waves are spherical forme and amplitude of the wave. Their distribution is low absorption, and therefore can travel large distances. Moreover infrasound waves bypass the barriers that stop noise and ultrasound, and easily penetrate into the premises because they expressed diffraction phenomenon. The latter is all the more manifested, the greater is the wavelength. Characteristic of infrasound waves are and their resonant manifestations that are capable of causing vibration of large objects.

There are numerous art manufacturing technical sources of infrasound: compressors, turbines, blast and rotary furnaces, industrial fans, excavators, bulldozers, cranes, pneumatic tools, means of transport (trains, planes, buses, trucks, ships), acoustic warning devices (sirens), space equipment and others. Intense infrasonic waves also arise in chemical and nuclear explosions.

Designed are: subsonic steams (France) for heat demonstrators, which generates five miles infrasonic field, causing severe pain and vibration to the human body; subsonic projector (USA), damaging vision and causing a feeling of fullness and fear.

There are infrasonic generators for ore dressing, cleaning vegetables and metallic and synthetic filters, studies of geological structures and others.

6.5.1. EFFECTS OF INFRASOUND ON THE BODY

It is believed that infrasound is irritant receptors at organ of Corti, vestibular system, Vater-Pacini skin receptors. In phenomena of resonance and stimulate pain receptors in the internal organs, intero- and proprioreceptors, mechanoreceptors are activated different autonomous neural and neurovascular reflex mechanisms. Allowed and auditory detection of infrasound, without this constituting a real hearing. Probably from the middle ear are perceived periodic pressure changes caused by infrasound.

When it comes to the mechanisms of the biological action of infrasound, utmost importance is given to induced resonance - coincidence of infrasound frequencies with the natural frequency of some organ is irritate its inter- and proprioreceptors which information is transmitted to the nerve centers. Resonance of internal organs is infrasound at a frequency of 2-8 Hz, and head - about 17-25 Hz **(Fig. 1)**. It is established the interaction between the frequency of brain alpha rhythm in humans and frequency of infrasound emitted by marine and ocean waves. Intense infrasound at a frequency of 7 Hz, coinciding with the frequency of alpha rhythm brain, is very dangerous, even deadly. Some authors, depending on the intensity and the frequency characteristic of infrasound define "zones of danger/safety" **(Fig. 2)**.

Adverse psycho-physiological effect is reduced work capacity directly dependent on the intensity and length of exposure to infrasound. Infrasound acts directly on the nuclei of VIII and X CBN and indirectly on other organs and systems. Described are weakening auditory sensitivity, balance disorders, nystagmus, worsening the parameters of attention, the accuracy of movement and orientation. The impact of infrasound at a frequency of 20 Hz for 8 minutes and intensity up to 144 dB causes unpainful pressure in the middle ear, which relieves by the test of Valsalva or termination of exposure. Morphological changes in the receptor cells of the three semicircular canals and of the inner hair cells of the spiral

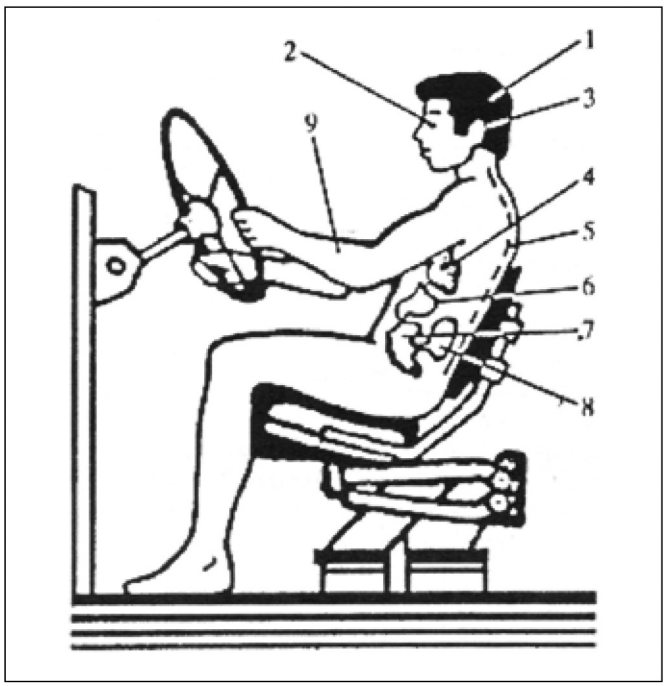
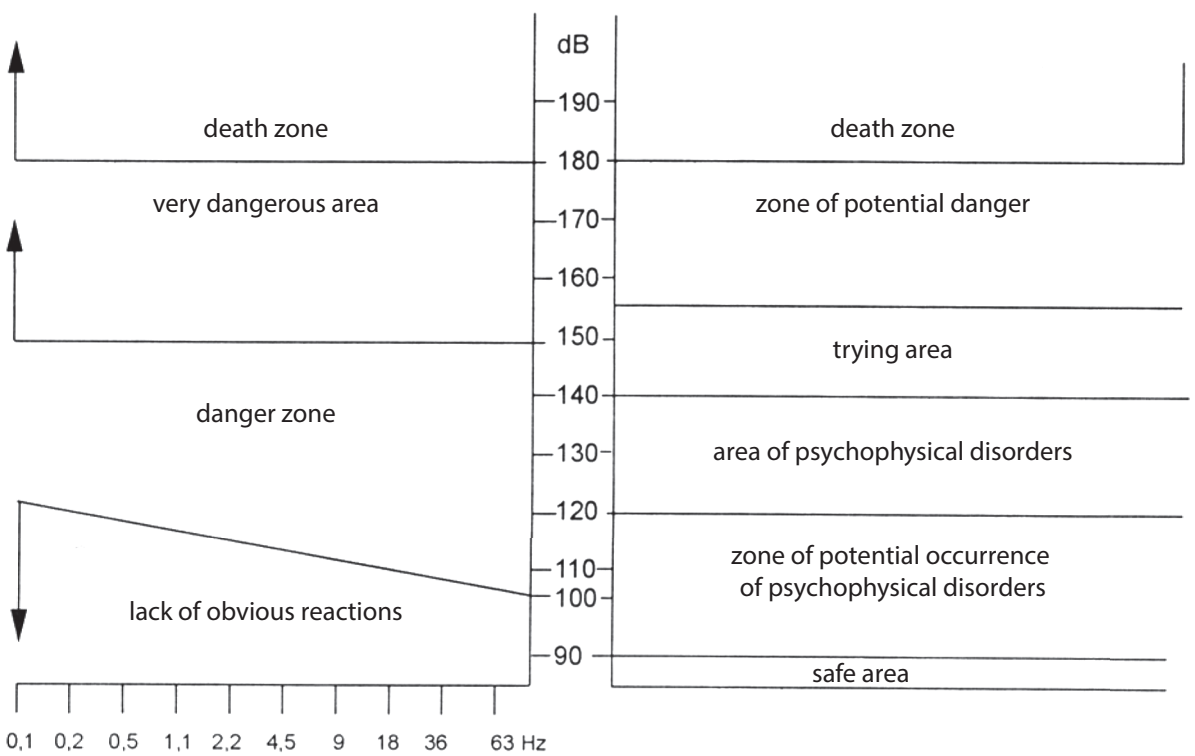


Fig. 1. Own (resonant) frequencies of the human body: 1-head (20-30 Hz); 2 - eyes (40-100 Hz); 3-vestibular system (0,5-13 Hz); 4-heart (4-8 Hz); 5-vertebrae (4-6 Hz); 6-stomach (2-8 Hz); 7-intestine (2-4 Hz); 8-kidney (6-8 Hz); 9-hands (2-5 Hz)



(by A.Stan, 1974) (by E.N.Malishev, 1979)

Fig. 2. Areas in danger of infrasound impact

canal after the subsonic exposure were observed by electron microscopy, with reversible changes in the endoplasmic reticulum and mitochondria.

Biometric indicators for infrasound biological effects, of exposure to infrasound - parameters 4 Hz and 130 dB, in show prevalence of general responses of the human body. Infrasound waves with intensity above 100 dB cause headache, pallor, nausea, weakness, fear, increased sweating, feeling friction and vibration of internal organs (liver, stomach, spleen and chest wall), difficulty breathing, decrease in visual acuity, increase auditory threshold, tinnitus, impaired speech intelligibility and others. The resonance range of brain structures between 5 and 9 Hz, makes these frequencies especially noxogenics. Upon impact of infrasound allow participation of limbic-reticular complex, hypothalamus and other subcortical structures with sensomotor and auton-

omous visceral symptoms. Infrasound is a stressor. It impairs the functions of the pituitary, thyroid and gonads. The intense infrasound change frequency and the rhythm of cardiac contractions with tachycardia in the beginning and bradycardia after. Also disclosed is a peripheral vasodilatation with increase in skin surface temperature and reduced systolic blood pressure. In the experiment established, however, spasm of peripheral vessels and increases blood pressure in unchanging values of the pulse. Studies of the effects of infrasound at a frequency of 8 and 16 Hz with a intensity of 120 to 140 dB in 3 hours a day for 1 to 40 days established an increased functional activity of the myocardium and absence of destructive changes. The acute effects of infrasound (frequencies 6, 12, 16 Hz and intensity 95, 110, 125 dB (lin) in the experiment at people establish reliably increase in diastolic blood pressure and the decrease in systolic without changes in heart rate. This suggests peripheral vasoconstriction with an increase in blood pressure, thus allowing the influence of chronic infrasound exposure at predisposing to hypertension people.

Infrasounds and noise inhibit contractility of the lymphatic vessels.

A combination of subsonic impact to that of electromagnetic fields, noise, vibration, ionizing radiation, biological effects are enhanced.

There are publications that do not show changes in behaviour of exposed to noise and infrasound in aviation and are considered unreliable opinions about behavioural changes in exposed human and manifestations of nystagmus.

6.5.2. PREVENTION OF ADVERSE EFFECTS

Prevention adverse effects of infrasound is difficult, due to these characteristics. Antiphons create weak protection. The most promising way is to reduce infrasound through *structural changes* in the sources - machinery, equipment, tools, vehicles. The effect of efforts to reduce the level of infrasound in the way of its distribution through soundproof enclosures and housings and sound-absorbing cladding is minimal. Better are reactive silencers range in subsonic spectrum. Deployment of sources of infrasound in separate and isolated areas may also give a certain result. Vehicles to be as distant as possible.

It takes frequent (at least once a year) measurements of infrasound level and good technical maintenance of machinery and vehicles. Different authors offered, and in some countries there are very different levels of infrasound. Nixon C.W. recommended level of 120 dB at a frequency of 20 Hz, with an increase of 3 dB for each subsequent lower frequency octave (16-1 Hz). In short duration (up to 8 min) recommended up to 150 dB for frequencies 1-7 Hz; 145 dB-8-11 Hz and up to 140 dB-12-20 Hz. Hygiene standards in Russia (1980) indicate values up to 105 dB for the range of 2-16 Hz, 102 dB-31,5 Hz and total infrasonic pressure - 110 dB. Similar regulations in Poland and Sweden, and in Norway general infrasonic levels up to 120 dB for 8 h shift. In psycho-sensorial labour (CP, administrative offices, design offices) and residential buildings offer lower levels - 80-90 dB (Poland).

In the system of prevention find a place and a number of *medical measures*. In *preliminary medical examinations* are recognized contraindications for work in the sphere of intense infrasound impact: the vestibular and hearing violations, expressed neurotic conditions, autonomic dysfunction, organic changes in the central nervous system. In *annual medical examinations attention* focuses on the constellation of subjective symptoms (headache, fatigue, dizziness, weight, sleep disorder, unmotivated fear, etc.) and objective data on changes in the vestibular and auditory, nervous and cardiovascular system. The establishment of these changes require appropriate vocational rehabilitation. In the periodic medical examination of the risk contingent involved neurologist, ENT specialist and internist (Decree № 3/1987, the Ministry of Health).

Relatively good results are obtained and *rational regime of work and rest* (20-minute rest in a quiet room every 2 hours). It is also periodically endangered stay in prophylactic places and sanatoriums in ensured conditions for recreation.

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N.Mitrev, D.Tsvetkov

6.6 ULTRASOUND

The term "ultrasound" means those mechanical oscillations whose frequencies exceed the limit of perception by the human ear*, namely those in the range from 20 (16) Hz to 1 GHz. The frequencies in the range above 1 GHz is designated as hypersound (1-100 GHz). In nature, ultrasound is generated by earthquakes, volcanic eruptions. Many animals, mostly inhabiting the underground (caves) and underwater or at night, produce and perceive ultrasound - birds, rats, bats, dogs, fish, etc., for exchange of information or echolocation. Eg. dogs perceive ultrasonic frequencies up to 44 kHz, bats - 115 kHz, rats - 72 kHz.

Although relatively new source of energy, multiple properties of ultrasound determined its widely used for industrial, medical and scientific purposes. The first attempts to use are associated with Paul Langevin (1918) - during the First World War for the purpose of hydrolocation. Sources of this type of mechanical fluctuations are varied ultrasonic sirens, magnetostrictive and piezoelectric transducers and others. Ultrasound is applied for cleaning and processing of hard materials and plastics, for the drying of fine powders, preparation of emulsions, ultrasonic fault detection, in hydrolocation and many others. In medicine **(Table. 1)** ultrasonic equipment is used for both diagnostic and therapeutic purposes in surgery (eg. lithotripsy), gastroenterology, cardiology, angiology, urology, physical therapy (eg. drug ionophoresis), obstetrics and gynecology, neurology, ophthalmology (eg. for the treatment of cataracts), dentistry (for removal of tartar) and the like.

A number of noise sources in production and transport are both generators and of ultrasonic frequencies - pneumatic tools, turbines, plasmotronics, compressors, textile machinery (in rotation spindles), gas burners, jets and more. In hygienic assessment of the risk of damage than the ultrasound intensity (measured in dB with a reference level 2.10^{-5} Pa or W/m^2) and spectrum, matter and conducting ultrasonic oscillations in different environments. In this aspect talk about *air and contact ultrasound*. Air absorbs ultrasound. Therefore, the risk for harmful effects of ultrasound by air is more limited but not negligible. The higher is the frequency of the ultrasound waves, the higher the rate of absorption of the air environment.

Conversely, the ultrasound waves are conducted better in a liquid medium (water, biological fluids) and spread easily from water in biological fluids. In such cases, talk about the impact of contact ultrasound. The degree of penetration and conduction the ultrasound in biological tissues depends on their structure (type) and functional status and is greater, the lower is its frequency. Attenuation constant α/in Np/m/ for ultrasound at a frequency of 1 MHz is: air - 15; water - 0.02; liver - 8; fatty tissue - 5-25; blood - 2; skeletal muscle - 10-35; bones of the skull - 115.

Tab. 1. Medical ultrasound equipment - characteristic (C.R.Hill, 1989)

Apparatus - kind	Frequency range (MHz)	Area source (mm^2)	Coefficient of efficiency	Average power (mW)	Spatial - temporal peak intensities (kW/m^2)	Spatial - temporal variations in pressure (MPa)*.
Diagnostics Pulse-echo (pulsed Doppler)	1-20	100-3000	0,001	0,01-80	1-10000	0,1-7,4 (+) 0,1-3,9 (-)
Permanent Doppler stomach / heart vessels	 2-4 5-10	 100 100	 1 0,01-1	 5,0-240 0,01-40	 200-18000 30-19500	 0,01-0,11 (±/ 0,003-0,12 (±)
Treatment permanent pulsatile	 0,75-3 0,75-3	 500 500	 1 0,2	 0-15000 0-3000	 0-100000 0-20000	 0-0,25 (±) 0-0,25 (±)

* Positive (+) or negative (-) relative to the mean value

* According to various surveys the lower limit of auditory perception is 5-16 Hz, and the upper - 16-25 kHz. These limits depend on the age, individual characteristics, methods of research. For practical purposes (audiometry) set thresholds of audibility for tones with frequencies above 12 kHz is also without relevance - very high and variable values

6.6.1. IMPACT OF ULTRASOUND ON THE HUMAN BODY

Arises from the ultrasound biological effect depends mainly on the intensity, frequency and duration of impact. Low-intensity ultrasound in the range of 80-90 dB influence stimulating (positively) on the body, while high intensity (120 dB) is negative, harmful effects. The low-frequency ultrasound produced effects similar to those of the noise. High-frequency ultrasound induces changes similar to those of radiofrequency electromagnetic radiation or extraauralic noise impacts.

The pathogenic action of ultrasound can be explained by four main and interrelated effects: mechanical, thermal, chemical (biochemical) and electrophysical. The mechanical effect is associated with both mechanical stress of intense ultrasound perceived by mechanoreceptors and the phenomenon of "cavitation" - the formation of small bubbles (diameter in micrometers) in water and biological fluids and high vapor pressure in them. Cavitation occurs in intensity of ultrasound over 0,3 W/cm^2, there is a strong destructive (mechanical) effect and is considered that it provokes and chemical effects - formation of new compounds (eg. hydrogen peroxide) and a free radical. In intensity of ultrasound over 1 W/cm^2 the resulting electrical potential is comparable to that of cell membranes and induced depolarization change their electoral permeability. Ultrasound with the suitable power there is and thermal effect - eg. This effect is used in the treatment of malignancies (destruction of cancer cells by hyperthermia - 42-45˚S), or in physiotherapy.

Biological action of ultrasound has been found in studies on: isolated biomolecules, cells, tissues, multicellular organisms (plants, insects, mammals), epidemiological studies in humans **(Table. 2)**.

In humans, pathogenic action of ultrasound is explained mainly by caused the mechanical and thermal effect, occurring disorders in tissues in the form of inflammation, heromagiya, denatura-

Tab. 2. Biological effects of ultrasound

Object	Biological effects / mechanisms
1. Isolated molecule - DNA - solution	1. rupture and destruction / cavitation 2. genetic changes have not been established
2. Isolated cells and cell cultures	1. demolition / cavitation 2. unaffected cell proliferation / without cavitation 3. greater influence in mitosis and relative resistance after mitosis / cavitation
- Genetic effects	1. chromosomal aberrations in joint action on X-rays and ultrasound 2. lymphocytes and lymphoblasts - no comfirming data on chromatid exchanges (mutagenesis)
- Structural changes	1. nonlethal changes - in the cell membranes of tumor cells, thymocytes, endothelial cells / cavitation, electric potential 2. changes in mitochondria, and destruction of lysosomes, cell death / cavitation
- Functional changes	1. changes in membrane permeability 2. the inhibition of motor function in aquatic microorganisms 3. boost: protein synthesis in fibroblasts; restoration of bone structure; angiogenesis in ischemic tissue / non-thermal and nocavitation mechanisms
3. Multicellular organisms - mammals	1. no evidence of gonadotrophic and embryotropic action in single doses under 7,5 kW/m^2 (non-thermal action) 2. prolonged exposure - reduced fetal weight and increased mortality 3. ocular lesions - cataracts, in intensity from 30-400 W/cm^2 and 1-9,8 MHz frequencies 4. stimulate: blood flow in the vessels; tissue regeneration
- insects	1. not established mutations in the gene apparatus of Drosophila melanogaster 2. the increased mortality of larvae of Drosophila in intensity over 100 kW/m^2/cavitation, heat above 42°C
- plants	1. destruction of cells, tissues, chromosomes in the intensity of more than 3 kW/m^2, more strongly manifested effects of cavitation

tion, necrosis, but there are reflex mechanisms and changes in hormonal regulation due to other effects - chemical, electrophysiological. As adverse effects of ultrasound waves concern astheno-vegetative syndrome, hypothalamic syndrome, vestibulopaty, lower visual acuity and changes in peripheral vision, vasomotor instability, dysesthesia, paresthesia, gastrointestinal dyskinesia. There is evidence of abnormal biochemical parameters (protein fractions, enzymatic changes), blood changes - transient eosinophilia, decreased blood pressure and others. Furthermore, the presence of ultrasounds in the mixed industrial noise sensitize the inner ear and can contribute to faster development of hearing damage - described temporarily decrease in auditory sensitivity of action of low-frequency ultrasound (17-37 kHz) and very high intensity - 148-154 dB, and noise in the ears (tinnitus).

All these aural and extraaural effects of **air ultrasound** in the production or related to the joint action of sound (noise), or similar changes may be caused by other physical factors, toxic agents or stress.

In action of **contact** ultrasound are described: ultrasonic polyneuritis; skin changes - hyperemia, hemorrhage, increased skin temperature, aseptic inflammatory reactions; changes in the cardiovascular system - tachycardia/bradycardia, arrhythmias, angina pectoris attacks.

Studies on the risk of genetic, carcinogenic and embryotropic effect in humans no show a statistically significant increased incidence of birth defects and malignant tumors using ultrasound for diagnostic purposes during pregnancy.

6.6.2. PREVENTION OF ADVERSE EFFECTS

Limit levels of sound pressure over 11,2 kHz at work places for 8 h exposure (industrial ultrasound), according to BS 12.1.001-79 are for third octave bands with geometrical mean frequencies: 12,5 kHz-75 dB; 16 kHz-85 dB; 20 kHz and more - 110 dB. The measurement is linear ("lin", not included "A" filter) and does not affect the contact ultrasound. Russian standards from 1983 (GOST 12.1.001-83) indicate levels: 12,5 kHz-80 dB; 16 kHz-90 dB; 20 kHz-100 dB; 25 kHz-105 dB; 31,5-100 kHz - 110 dB for air ultrasound and 110 dB or 0,1 W/cm^2 for contact ultrasonic transmitting on arms. Standards ACGIH (1999) indicate levels of: 88 dB - 10 kHz; 89 dB-12,5 kHz; 92 dB-16 kHz and 94 dB-20 kHz, also for 8 h exposure and threshold of 105 dB for this range. If ultrasound is narrowband (tonal) subjective discomfort can be obtained at levels of 75-105 dB for frequencies 10-20 kHz and must be taken measures for hearing protection and engineering solutions. To contact ultrasound are offered values of 110-115 dB at range of 25-100 kHz (all these values for air and contact ultrasound are at Po-20 μPa). When working underwater thresholds for ultrasound are 167-177 dB at range of 10-100 kHz (but Po-1 μPa).

Prevention of adverse effects of ultrasound includes a number of measures of organizational, technical and medical nature. It is appropriate devices, sources of powerful ultrasound, be placed in separate and insulated rooms and even better in cabins with remote control. Ultrasound generators need to procure passport data on power and frequency spectrum.

Limiting exposure is an important measure for prevention as well as in industrial and in medical use in of ultrasound. Here, as in noise, gradually requires the application of the approach to dosing by measuring the shift and all service dose and their limitation. In the medical use of ultrasound must strictly comply with the emission limit power, energy density and exposure, which is important for both staff and patients.

When working with ultrasonic cleaning baths is forbidden any contact of hands and other body parts with fluid; the same applies to the contact with ultrasonic tools and workpieces. Charging the baths should be performed at off equipment, and it is best that this be done automatically.

Personal protection using gloves with air and pincers (working with ultrasonic baths) and multilayer antiphones type "sandwich" designed to protect the auditory organ. In consider includes protective professional nutrition and rational regime of work and rest of the risk contingents.

Contraindicated for work in conditions of ultra sonication are persons with diseases of the central nervous system, auditory, paranasal sinuses, autonomic nervous system and hematopoietic organs, eye lens, cardiovascular system.

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6.7 VIBRATION

In 1911 G. Loriga first described the Italian workers with pneumatic vibrogenerating tools, who show symptoms of the disease type Raynaud*, and in 1918 A. Hamilton and associates established in hewers in Indiana a high incidence of "spastic anemia of arms" that relate to the cold-impact, compression and vibration of the arm. Subsequently, the comprehensive mechanization of almost all production activities and the pronounced trend of continuous increase in the number and diversity of introduced in use vibrating machinery and equipment have the effect of increasing the number of vibroinflenced persons. Currently, over 70 occupational groups exposed to vibrations **(Table. 1)**. The vibrations are expressed factor in road, rail, water and air transport. It pointed out that in the 70s and early 80s in the United States about 8 mil. workers impacted by vibration, of which about 6.5 million in transport operations (transport, agriculture, construction). Indicate there also 194 thousand impactful people with general vibration in foundries. In Bulgaria during the period 1975-91, the annual reported cases of vibration disease were held 4-5 place in the structure of occupational diseases, half were miners, and the next five professional groups in frequency were: tractor drivers, drivers, crane operators, turners , logging. Over the past 15-20 years, however, there is a strong trend for reducing the incidence of vibration disease in developed countries - up to 64% in England to 1982, 85% in Japan for the period 1978-1991, the 1/3 to half of them working in logging - combined effect of vibration and low temperature air (work outdoor).

6.7.1. GENERAL AND BASIC FEATURES

The term vibrations mean mechanical vibrations of the material bodies or points removed from equilibrium position under the influence of external forces.

The nature and severity of vibration-related injuries are caused mostly transmitted from the source of vibrations in the contact area amount mechanical *(vibration) energy*. This quantity is proportional to the intensity of vibration, the contact area (receptive area) and the time of exposure (exposure). *The intensity* of the vibrations is dependent on the **speed** ($m.sec^{-1}$), the **acceleration** ($m.sec^{-2}$) and **amplitude** (mm). Another important feature of vibration is repetitive or periodic motion, ie *frequency* (Hz).

The vibrations are felt as such at frequency to about 8 kHz - higher frequencies are perceived as heat. The vibrations are measured in the range of 1-1000 Hz, or 8-1000 Hz - for local vibrations and 1-63 Hz for general (these values are the mean of octave bands). Based on frequency vibrations are divided into low frequency, mid- and high frequency, which are respectively at general and local vibration 1-4 Hz, 8-16 Hz and 31,5-63 Hz; 8-16 Hz, 31,5-63 Hz and 125-1000 Hz. Low frequency vibrations propagate more intensely in the human body from the point of contact and high-frequency local vibrations lead more quickly to vasospasm.

Possible **resonance** hardening (even repeatedly) of own vibration of the tissues, organs or whole body under the influence of low frequencies vibration, i.e. increasing the risk of vibration damage. The resonant frequency of the whole body in a sitting posture is 4-6 Hz, and in standing - 5-10 Hz; head 20-30 Hz; heart and liver - 4-6 Hz; stomach - 6-8 Hz.

Depending on the direction of oscillation vibrations are *horizontal, vertical, rotational* and so on. The vertical general vibrations have a more negative impact on the human body in an upright and a sitting position and horizontal general vibrations - in lying. Vibrations characterized depending on the time being - ***periodic, aperiodic (strokes), stochastic (random), declining and short-term (pulse)***. Most often in production are **complex** - the result of superimposing very **simple (harmonic or sinusoidal)** vibrations with different characteristics at the time, direction, movement and regime. According to the contact between man and vibrational sources influential vibrations are **local** (segmental or even hand-arm vibration), **general and mixed** (local and general). In the first case the transmission of vibrations going through your hands when working with hand-held vibrating tools, and the second - they act on the body in standing, sitting or lying position.

* Maurice Raynaud (1862) doctoral thesis "Local asphyxia and symmetrical gangrene of the extremities" - attack and permanent white - fingers (or "dead hand"), with subsequent atrophy and gangrenous changes, now called Raynaud's disease or primary Raynaud's syndrome.

Tab. 1. Sources of above the standards vibrations in production *

Production	Source	Type vibration	Mean rate exceeding standards - K
1. Mining	drills	Iv	2-3,5
	breakers	Iv	4-14
	loaders	Iv	1,5-3
		Gv	2-3
2. Coal	drills	Iv	2-3,5
	breakers	Iv	3-10
	electrobores	Iv	1-2,5
	loaders	Iv	1,5-2,5
		Gv	1-2
3. Open mine	excavators	Gv	1-2,5
	boring machines	Gv	3-8
	electric carrier	Gv	3-10
	bulldozers	Gv	3,5-4
	cargo vehicle	Gv	3-4,5
4. Machine-building	pneumatic hand instruments	Iv	2-4
	grinding machines	Iv	1-1,5
	presses	Gv	2-2,5
	molding presses	Gv	2
	compressors	Gv	1-1,5
	cold treatment	Iv	1-1,3
	cranes	Gv	1-3
5. Logging	chainsaw	Iv	2-5,5
	tractors	Gv	1,5-2,5
	cargo vehicle	Gv	2-3,5
6. Production of ready vibroelements for building	vibrators	Iv	2-3
	vibroplatform	Gv	1-1,5
7. Agricultural	tractors	Gv	1-3
		Iv	1-2
	tractor-driven	Gv	2-3
	bulldozers	Gv	2-3
8. Textiles	textile machinery	Gv	1-1,5

** The data represent mean values from different measurements and should be regarded as tentative. The parameters of vibration strongly influence various factors (mainly technical) and they can be very different under the same machinery and manufacturing processes.*

For hygienic assessment of vibration they are measured in three orthogonal directions (x, y and z). In local vibrations into account the highest measured value in different frequency bands **(Fig. 1)**. In general vibrations **(Fig. 2)** are evaluated separately vertical (z) vibration and the highest values in different frequency bands from both directions (x and y) of horizontal vibrations.*

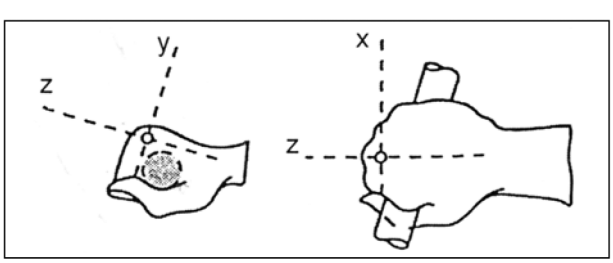


Fig. 1. Standardized position of the axes x, y and z in local vibrations

Determine also the exposure time of the work shift.

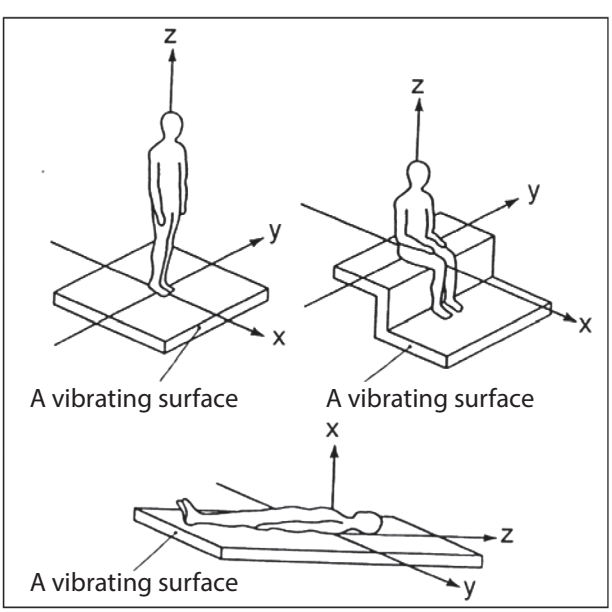


Fig. 2. Standardized position of the axes x, y and z with general vibrations

* Now at local vibrations into account the amount of **simultaneously** frequency-weighted values in all three axes, and in general - also frequency - weghted values, but **separately** for the three axes.

Besides vibrations for the occurrence of vibration mediated pathology significance have other adverse factors. Most important in this respect are static tension of muscles, reverse shot tool, awkward posture, cold impact (local or general), noise, toxic noxal (nicotine, aerosols of heavy metals, organic solvents, carbon monoxide, etc.).

6.7.2. IMPACT OF LOCAL VIBRATIONS ON THE BODY

Local vibrational energy is absorb primarily by upper limb and that is why they are affected most when working with hand-held vibrating tools, but cause and generalized damage. Damage is primarily functions and structure of the nervous, cardiovascular and osteo-articular system.

Vascular disorders are one of the earliest and dominant manifestations of vibration damage. Based on these in Anglo-Saxon literature vibration disease is described as a vibration-induced phenomenon of Raynaud, vibration induced white finger, traumatic vasospastic disease, work-related phenomenon of Raynaud. Since 1983 according to international consensus called **vibration-induced hand-arm syndrome**. In Southeast Europe, Japan and the Scandinavian countries talk about **vibration disease**.

In local vibrations occur varying degrees of markedness changes in the tone of the arterial bed. Disorders in the regulation of increased vascular tone of arteries of medium and small-caliber in upper limb has been proven repeatedly by a number of methods, including. arteriography, infrared thermography, Doppler sonography, fotopletismography, laser-Doppler flowmetry. Expression of these violations has decreased skin temperature of the hands sometimes and asymmetrical, while at generalization of process and in lower limbs, not only during ischemic crises "dead fingers", but in intercrises periods. Attacks are preceded by a latency period whose duration is much shorter, the more intense the vibrations, ranging from years to weeks. The shorter this period is, the progression of the phenomenon is more severe and attacks - more frequent. In the initial period the graying covers skin of the distal phalanges of III and IV, less II and V of the fingers, as the attack is short - from seconds to several minutes.

In advanced cases, the attacks become more frequent and longer, sometimes up to two hours and cover fully all phalanges of the fingers. Classic attacks of sudden blanching and numbness of the fingers with sharp demarcation are often provoked by cooling. Early attacks occur mainly in the early morning in winter, at home or on the way to work, when exposed suffering to cooling. Outside workers are most exposed to early morning attacks. Advanced occurring serious difficulties in the performance of work duties, social activity and entertainment.

There are also atypical forms - describe attacks by graying of the top half of the ear shells and ischemic coronary events in some vibration disease patients. It can be observed development of cerebral angiodystonic syndrome, characterized by headache, neurasthenic manifestations, due to functional dysregulation of brain circulation.

Pathophysiology of vascular disorders. On **Fig. 3** presents the pathophysiological mechanisms of vibration disease from local vibroimpact.

Vasoconstrictor response is determined by the increased activity of sympatho-adrenal system or parasympathetic depression caused by overstimulation of Vater-Pacini corpuscles. There was also increased receptor activity in arterioles and arteries to vasoactive substances with functional dominance of α_2-adrenergic receptors in acral parts. Activation of serotonin (5HT2) receptors in the digital vessels also plays an important role in provoked by cold events type Raynaud. The vibrations damaging local dermal perivascular nerves containing the powerful vasodilator calcitonin gene-related peptide (CGRP).

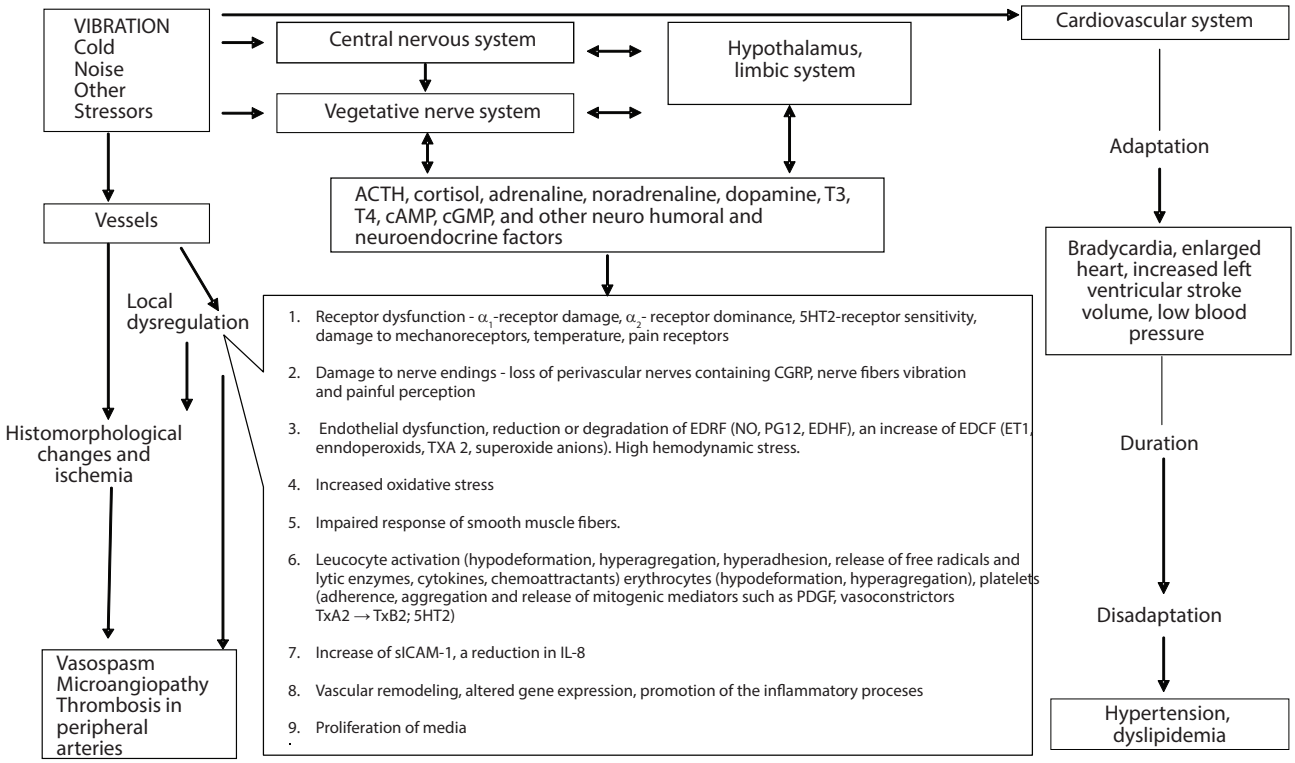
Endothelial vasodilator substances - nitric oxide (NO), vasoactive prostaglandins (prostacyclin) and endothelium-derived hyperpolarizing factor (EDHR) modulate vasoconstriction driven by adrenergic nerves, originating from the blood cells substances and local autocoides. Endothelial cells produce and vasoconstrictor substances, such as prostanoids, thromboxane A2, superoxide anions, endothelin-1 (ET1). Numerous factors secreted by the endothelium and the complex interactions between them and noendotel mediators determined local vasomotor control on vascular smooth muscle cells.

Humoral dysregulation alter hemostasis, fibrinolysis and haemorheology.

It was found impaired permeability of the capillary walls for plasma proteins, increased blood viscosity (hypercoagulable), increase the concentration of plasma hematocrit and hemoglobin.

Vibration exposure causes and intravascular platelet activation which can enhance the release of thromboxane A2 (TxA2), whose metabolite thromboxane B2 is a potent vasoconstrictor. In places of vascular stenosis leukocytes and platelets are subjected to extremely large hemodynamic stress from blood flow (shear stress), which further activates them. Leukotrienes, chemotaxis factors and cytokines secreted by damaged endothelium, activated leukocytes further, on a background of reduced production of prostacyclin.

Fig. 3. The pathophysiological mechanisms of vascular injury



Histomorphological changes in vessel wall grow in the more advanced stages of the vibration disease. There have been intense thickening of the muscle layers of the digital arteries, with a strong hypertrophy of smooth muscle cells without intimal fibrosis, but with the peri-arterial fibrosis and sometimes changes with atherosclerotic foam cells, lipid deposition and fibrous sclerosis. Development of severe morphological changes in the vascular wall with pregangrenous and gangrenous condition of the fingers, described mostly in the past due according to some authors less impact vibration as other accompanying them nonspecific risk factors - trauma, infection or intoxication.

Peripheral nerve disorders. In local vibrations has expressed peripheral and central neural pathologies. Especially pronounced polyneuritic syndrome with sensory and autonomic vasomotor, sudosecretory, thermoregulatory, neurovascular and dystrophic disorders. The symptoms of vibration disease is exept microcirculatory but senseneural - prickling, tingling, burning, numbness, insensibility of fingers with intermittent character initially and subsequently permanent accompanied by pain.

Local vibration damage mainly peripheral nerves of the upper limbs, and sensory nerve fibers are more sensitive and vulnerable than motor.

Establish lowered **vibration sensation (hypopalestesy)** even before the onset of other symptoms of vibration disease. The combined impact of vibration factor with cold increase the risk of an increase in the threshold of vibrosensitivity. A similar combined effects of vibration and physical load, probably as connected with vibroreceptors fatigue and impaired local circulation. On the functional status of specialized mechanoreceptors perception of vibration influenced and some chemical (adrenaline, acetylcholine, novocaine, drugs, caffeine, fenamin etc.) and physical factors (ultrasound, noise, ionizing radiation, etc.), age changes (atherosclerosis, hypotrophy of the skin, changes in mechanoreceptors, etc.). The earliest notable and most pronounced is hypopalestesy under the influence of vibration frequency of 125-250 Hz, probably due to the increased vulnerability of specialized FAII receptors (i.e. inclusion Vater Pacini corpuscles), who perceive these frequencies, and later affected and low-frequency range perceived by the FAI and SAI receptors (cell complexes Merkel and corpuscles of Meissner). Hypopalestesy is most pronounced in the distal sections of limbs, especially volar side of the tips of the fingers and plantar of foot.

The vibrations harm the superficial sense of touch and pain. **Hypesthesy** *(hypalgesia)* of distal type and unlike of hypopalestesy is more pronounced at impact of low frequency vibrations. In the early stages of the vibratory disease hypalgesia settled in the fingertips, during disease progression "climb" proximal covering successively wrist and lower third of the forearm (type "gloves"). In the third stage can acquire segment character and cover the shoulder girdle and part of the chest (type "short jacket" or

Vascular	Neurological
Tests Adson and Alain	Tests of Tinel and Fallen
Hyperabduction and bone-clavicle movement	Abduction-external rotation
Doppler sonography	Muscle strength into a fist
Laser-Doppler flowmetry	Deep sensibility
Finger blood pressure before and after a cold provocation	Discriminatory sense
	Manipulative dexterity
Plethysmography before and after the cold stress test	Palestesiometry
Cold test	Temperature perception
Capillaroscopy	Nerve conduction

Tab. 2. Clinical and laboratory tests

"breastplate with sleeves"). Tactile sensation is less disturbed, but according to some authors, exposure to high-frequency vibrations can cause a temporary decrease in the sense of touch even full loss. Damage and complex sensation- *discriminatory sense* to the extent of the incapacity of the patient to perform fine manipulation.

In local vibrations occur, and abnormal **temperature sensitivity** with an increase in the thresholds for thermal and, to a lesser grade to the cold. In patients with vibration disease is established and **hypotermestesy hypotermalgesia** not only in the points of direct contact with the vibrations, but also in other body parts. In some cases termanestesy may create a risk of burns on contact with hot objects.

A study of the **functional state of the neuromuscular apparatus** in most patients is established and reduction of the lability and increase in the thresholds of excitation. In the early stages decreased muscle tone and endurance. Muscle strength changes phase - increased at the beginning and progressively reduced. Possible **trophic muscular changes** in upper limbs and shoulder girdle and painful hardenings type myofasciculus or fibromyositis. In advanced stages are possible hypo- to atrophy of individual muscles or muscle groups.

For musculoskeletal disorders have a dominant importance and accompanying vibration static and dynamic intension, return punch tool, heavy physical labour that lead to mechanical traumatisation.

Be established **tendovaginitis** most of the flexors of the hand, radial and ulnar *epicondylitis, periarthritis* predominantly humeroscapular joint it. A common finding is *tendomyositis* of extensor group muscles of the hand.

An important place in the clinic of vibration disease hold **degenerative-dystrophic changes in the osteo-articular system** in the upper limbs, shoulder girdle and spine. The character of bone-articular changes too diverse - among the most typical are *enostosis*, regional *osteoporosis*, *osteochondrosis* and *deforming spondylosis, bone cysts and deforming osteoarthosis*. Surveys indicate most common degenerative-dystrophic changes enostosis (63.3%), cystic flashes (58.3%), regional osteoporosis flashes (24.5%), deforming osteoarthrosis of the interphalangeal and metacarpo-phalangeal joints (31.1%). More frequent and *aseptic necrosis* of carpal bones on the type of disease Kienbock.

In the insertions of muscles and tendons grow hardening and calcium deposits.

On **Table. 2** are recommended clinical and laboratory tests to diagnose pathology of overstrain and vibrations in working conditions.

In the differential diagnosis in patients with vibration disease should be excluded a number of diseases as the primary phenomenon of Raynaud; secondary Raynaud's phenomenon with different etiology - collagenoses (scleroderma), endocrine, metabolic, cardiovascular diseases, intoxication (vinyl chloride); polyneuritis and polyneuropathy with other genesis, syringomyelia, disseminated sclerosis, cerebrovascular disease, pernicious anemia, compression and neuro-vascular syndromes in upper limbs.

On **Table 3** presents senso-neural, microvascular and musculoskeletal criteria for determining the stage of vibration disease.

6.7.3. EFFECT OF GENERAL VIBRATION ON THE BODY

General vibrations are regarded as non-specific common stressors that can act multiform on a parts and organs of the human body depending on the vibration characteristics. In addition to musculoskeletal system general vibrations affect the reticular formation, cerebellum, spinal cord and brain and endocrine glands and cause various psycho-sensory, neurovegetative and somatic reactions of the body.

The main approaches to the evaluation of the relationship cause/effect of general vibration is most often limited to: subjective assessment of tolerance to vibration and a feeling of comfort; reliability of the tasks (a performance); integrated assessment of functional status of the human body with the objective physiological indicators.

Intensive general vibration impact and duration of exposure worsen proportionally results of the visual-motorial tasks.

Subjective evaluation of vibration is based on the

Stage	Symptoms
1SN	Intermittent numbness and tingling in fingers
2SN	Intermittent or persistent numbness, tingling and hypoesthesia of hands
3SN	Intermittent or permanent numbness and tingling, grown hypoesthesia in the hands of pain in the fingers and upper extremities, decreased sensation discriminatory
1V	Cold fingertips with or without numbness, attack after provocation
2V	Attacks phenomenon of Raynaud (RP), comprising the distal phalanges of
3V	one or more fingers with numbness
4V	RP expressed all phalanges of the fingers, frequent ischemic attack in summer and in winter Graying full of all fingers of both hands in summer and in winter, trophic changes
1M	Intermittent or persistent numbness and pain in the fingertips
2M	Impossible fine movements in everyday life such as fastening buttons or
3M	handling small objects. Expressed pain in the upper limbs. Constant pain in the arms, upper limbs and elbows.

Tab. 3. Stockholm criteria for diagnosis of vibration disease (1987) *

* O [SN, V, M] – exposed to vibration, without clinical symptoms

threshold of sensation, tolerance and discomfort (pain, anxiety and fear). Studies in production conditions established a number of subjective complaints - pain in the lumbar and cervical spine, epigastralgia, nausea, decreased visual acuity, insomnia, spasms in the colon, neurotic symptoms.

Changes in the functional state of the ***central and peripheral nervous system*** - increased fatigue, headaches and dizziness, sleep disorders and irritability, tremor of the eyelids and hands anisoreflection, disturbed cerebral hemodynamics, activation of the reticular formation, mesodiencephalic and brain stem structures. Damage to the peripheral nerves and the formation of vegetative polyneural syndrome are possible in general vibration impact.

Damages and *musculoskeletal system* (MSS). When exposed to intense general vibration establish degenerative changes in the spine (lumbar and cervical segments), osteoarthritis of the radio-ulnar joint, aseptic necrosis of semilunar bone, pain syndrome in the lumbar-sacral part of the spine, ischioneuralgia pain, reduced muscle strength, muscle cramps and myalgia.

Common in general vibration impact are the reactions of the *vestibular and auditory analyzer* - occurrence of discomfort, dizziness, nausea and drowsiness at the end of the workday, changes in the regulation of posture in stabilography, greater increase in hearing threshold in exposed to noise and general vibrations.

General vibration can also affect the function of the *visual analyzer* with signs of decreased sharpness of vision and worsening of peripheral vision and color vision.

There are changes on the part of the *cardiovascular system* - slowing heart rate, lower blood pressure, changes in reographic and Doppler sonographic indicators, increased tone of cerebral vessels in tractor drivers and combine operators, peripheral vascular disorders in the legs with operators of excavators and tractor drivers. One reason for gynecological pathology in women exposed to intense general vibration are disorders in blood circulation to the organs in the pelvis.

General vibrations cause a number of *endocrine, hormonal and neurohumoral effects*. They affect adreno-cortical system, increase of testosterone and growth factor and reduction of cortisol, as well as higher concentrations of glucose and insulin, and many others.

More common among women of professions with vibration exposure are the emergence and exacerbation of inflammatory *gynecological diseases*, spontaneous aborts and premature births, gestational toxicosis and post-natal complications. Animal experiments suggest disabilities and in fetus.

To assess the impact of general vibrations during the last 10-15 years, many researchers use indicators of morbidity with temporary disability. In such analyzes an increased frequency of accidents and diseases MSS, gastrointestinal, cardiovascular, urinary and reproductive system of tractor drivers, operators of excavators and steam-navvy machines and disc disease in tractor drivers and crane operators. In the structure of the cardiovascular pathology most common are hypertension, followed by chronic ischemic heart disease.

The presence of numerous themselves nonspecific changes in the impact of general vibrations have their further study with a view of complex evaluation and forecasting the state of the body.

6.7.4. PREVENTION OF ADVERSE EFFECTS.

Preventing vibration pathology includes control and regulation of vibration, measures to weaken the vibrations at source and limit their spread; measures against other factors and conditions of the working environment of etiological significance for vibration pathology; rationalization of the regime of work and work organization in vibro dangerous professions; measures with medical prophylactic character.

Control and standartisation of vibration. Under Decree № 3 of MLSP and MH (2005) in accordance with BS ISO 5349-1:2004 and BS ISO 2631-1:2004 (identical to ISO 2631-1:1997) - according to local vibrations (vibration transmitted to the hand) and general vibration (vibration on the whole body) on the health hazards of workers exposed to vibrations, states:

In local vibrations (hand-arm system):

1. The daily exposure limit value for a period of 8 h - to 5 ms^{-2}

2. The daily exposure value for taking action, for a period 8 h - to 2.5 ms^{-2}

In general vibration (whole-body):

1. The daily exposure limit value for a period of 8 h - to 1,15 ms^{-2}

2. The daily exposure value for taking action for a period 8 h - up to 0,5 ms^{-2}

The above values to **local vibrations** are common (a_{hv}) vibration values, determined by the sum of together reported frequency weighted square root values of acceleration (in ms^{-2}) measured on three axes - x, y and z, of the vibrating surface.

For a period of 8 h (vibrations actually do not effect 8 h, and the fewer, but similar to the effects of noise or chemical noxae here use "time-weighted averaging") the daily exposure value $A(_8)$ is defined as:

$$A(_8) = a_{hv} \sqrt{T/T_O}$$

where T - total daily exposure s (h)

T_O - exposure 8 h (28.800 s)

In **general vibrations** daily exposure $A(_8)$ is defined as:

$$A(_8) = k.a_w \sqrt{T/T_O}$$

where a_w is the vibration magnitude (square root) of the weighted average frequency accelerations in each of the three axes - x, y and z.

T_O - 8 h (28.800 s)

T - real duration of exposure in h (s)

k - factor of 1.4 in x and y axes and 1.0 on z axis

Daily exposure is regarded as the highest of the calculated the three axes.

Workers should not be exposed to levels exceeding the exposure limit values of general or local vibrations. The employer is obliged to take action (or change them) in order to avoid exceeding them. Allowed some exceptions (eg ships, helicopters, air screw planes) when possible accidental increase, but with a proven lower risk than that of excess. Also, the employer provides health surveillance in accordance with Ordinance № 3/1987 on mandatory testing of workers in the presence of a health risk (contact vibration), while exceeding the daily exposure values is obliged to provide it, and exceeding the limits (at specified exceptions) is obliged to provide enhanced health surveillance.

According to BS/ISO 5349-1:2004 (identical to ISO 5349-1:2001) evaluation of **local (hand-arm) vibration** changes:

1. Estimated total value of vibrations, while in the previous standard ISO 53491:1985 permissible vibration acceleration of 2 ms^{-2} is compared to the direction (x, y and z axis), where frequency-weighted rms value is greatest.

Therefore now the measured value (total value) is greater by 1.0 to 1.7 times (typically 1.2-1.5 times) than the highest, but only one of the axes (respectively in comparison with previously measured values they must be multiplied by the appropriate factor - mostly 1.3 to 1.4).

2. The daily assessment of the impact of vibration is based on the equivalent energy (vibration acceleration) for 8 h. In ISO 5349-1:1985 it treated the period of 4 h. Transforming 4 h equivalent values to those for 8 h requires multiplication by a factor of 0.7.

3. Retain in general showed in standard ISO 5349-1:1985 compliance between the development of the syndrome of "white fingers" and the impact of vibration (vibration acceleration), but correction are made due of reference now to the equivalent period of exposure 8 h and multiplication values by a factor of 1.4.

According to BS (ISO 2631-1:2004 (identical to ISO 2631-1:1997) estimate of **general vibration (on the while body)** changes compared to the first edition of ISO 2631-1:1985, as:

1. The evaluation is based more on frequency-weighted mean square (rms) acceleration value of the vibrations and their impact on health, comfort and perception, as well as diseases of the travelling (malaise travel - vomiting, nausea, headache, dizziness and etc.). **Not included:** "exposure limit", but assessment methods can be used as a basis for the limits with appropriate action (measures); also the concept of "decreasing performance (fatigue)" under

the effect of vibrations because it much depends on ergonomics and working conditions of the operator.

2. The range of vibration is extended under 1Hz:

– from 0.5 to 80 Hz - for health, comfort and perception

– from 0.1 to 0.5 Hz - for diseases of the travelling.

3. The main method of assessment is frequency weighted square root value (rms) of acceleration. When vibration ratio peak above 9 (including accidental shots) is optionally available using two other methods:

– method for assessing the current r.m.s values or determine the so-called MTVV (maximum value of the previous vibration);

– method of fourth degree of vibration dose VDV.

Emphasize that the risks to health impairment always evaluated by using the main method, but in case of suspicion of misjudgment to use and the aforementioned methods.

Measures to weaken the vibrations at source and limit their spread outside source. The weakening of vibrations in the source of their generation can be achieved in the way of constructive and technological measures: change of kinematic scheme and the working cycle of hand power tools; improving the production of components, their installation and the parameters of vibrating machines and tools; the use of materials with high internal friction; careful balancing of moving parts and so on.

The elastic suspension of part of corps of the chainsaws eg., playing the role of dynamic vibration reducer leads to significant damping effect and reduce the frequency of vibration damage. Appropriate suspension or isolation systems in machines and vehicles with fixed base by inserting elastic springs or other damping material between the source and the person, reduces vibrations significantly. Such solutions include springy seats in vehicles with rough ride and airplanes.

The spread of vibrations outside their source may be hindered more by anti vibration processing bases of machines and foundations using rubber pads, insulating joints of vibroisolated materials, removing the solid connection between the vibrating platforms and structural elements of the premises, isolation of workshops with vibrating machines and equipment, etc.

To isolate the hands of workers in condition of vibration effects often used gloves of vibration-absorbing materials, that protect from moisture and cold.

Results in the fight against vibration are achieved and the way of improvement of technological processes. In this regard, consideration includes the replacement of machines and tools, working on stroke principle, with those that exclude the stroke, founding in molds with liquid self-hardening mixtures, allowing it to be discarded pneumatic rammers and molding machines; attaching the processed details on vibration pad, etc.

The most radical way to combat the harmful effects of vibration is a remote control vibrating machinery and equipment, thereby limiting the influence of noise, cooling and physical load.

Meaning have such measures as compulsory passport of vibration sources, inclusion in their documentation rules for installation, operation and maintenance.

Measures against other factors and conditions attendant vibrations. To eliminate the cooling apply various methods: using warmth- and moisture protective gloves, clothing and shoes; making handles and other parts of contact with hands on materials of suitable coefficient of heat or lining the same with such materials; create a comfortable microclimate conditions on the premises; ensure during the cold seasons of heated transport and space heating for outdoor work; change the direction of the exit of the exhaust air of pneumatic tools, cooling hands and others.

To reduce the physical load required handles and levers have a shape that is not only comfortable for the job, but also evenly distributes the force of pressure, reduce the contact area with the tool and the machine and preventing jams.

Rationalization of the regime and work organization. The organization of complex brigades and rotation of manipulations, performed significantly decrease the incidence of vibration disease, by reducing the exposure time. In Bulgaria are distributed special instructions for prophylactic regimen of work in jobs threatened by local vibrational effects; determinated admissible exposures of a single time vibrations, duration of pauses and limit a total exposure per shift, depending on the intensity of vibration; a total maximum exposure for shift should be up to 90-120 min. When operating vibrating tool should stick slightly loose, avoiding large static effort and optimal angle of supination between the arm and forearm between 90 ° and 150 °.

Measures with medico- prophylactic character. The professional selection for work in conditions of vibrations is held in strict observance of contraindications: organic diseases of the central nervous system, incl. epilepsy; marked autonomic dysfunction, diencephalic events; Raynaud's disease and Raynaud-like syndromes, autonomic polyneuropathy; neuritis and polyneuritis; diseases of musculoskeletal system, disorders of motor function; diseases of the cardiovascular system; diseases of the endocrine

glands with permanent impairment of their function; ulcer; permanent hearing loss, otosclerosis and other ear diseases with unfavorable prognosis for hearing; chronic diseases and expressed anatomical changes in the female genital area.

At risk and affected by vibration disabilities should be cautious in order to avoid extra work vibrations (i.e. long riding a motorcycle, operating motor mowers, power saws, drills, etc.), and in particular in conditions of cooling, soaking and moisture. It is also using the beneficial effect of specialized production gymnastics, regular nutrition and suitable professional diet (diet A - rich in animal protein, biologically active substances, vitamins B_1, PP and C); must be eliminated smoking, alcohol abuse.

Among the means of individual prevention of injuries from local vibration most recommended are warm hydroprocedures. Recommended and gentle self-massage after the procedure and wearing warm gloves during the cold seasons.

Prophylactic examination is held once a year (oto-rhino-laringological - at two years) by a neurologist, therapist, orthopedic and ORL specialist, and at indication - and by obstetrician and rheumatologist.

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6.8 REDUCED ATMOSPHERIC PRESSURE

Globally noted an increasing number of people who work and live in conditions of reduced atmospheric pressure. That is due to the rapid development of aviation and aerospace, and also the conduct of a number of economic activities in heights - construction and mining sites, scientific expeditions, weather stations, mountain tourism, farming and agriculture and others. At the present time over 25 mln. people live and work at altitudes above 3000 m above sea level, most of them are not adapted to height. Reduced atmospheric pressure found in natural conditions when climbing in height and in artificial conditions (eg. in hyperbaric chambers).

With increasing altitude, barometric pressure (PB) reduces, the temperature dropped by about 6,5° C at 1000 m. **(Table. 1),** reduce the cloud, dust, and the pressure of the water vapors. Ultraviolet radiation increases by about 4% at 300 m. (additional snow reflect UV rays)

6.8.1. PHYSIOLOGICAL EFFECTS OF REDUCED ATMOSPHERIC PRESSURE

With elevation of the height due to the reduced barometric pressure, as a result of the decreased partial pressure of oxygen in the air occurs differently pronounced hypoxia in the tissue. According to the etiology and pathogenesis are 4 main forms of hypoxia:

Hypoxic hypoxia - Causes can be diseases violating oxygenation of venous blood in the lungs. In healthy people this occurs in elevation height without extra breathing oxygen - i.e. so-called ***altitude hypoxia***.

Chemical hypoxia - occurs when a drop content of hemoglobin in the blood (blood loss, poisoning, etc.). It is characterized by a reduction in the PO_2 in the venous blood and tissues in a normal PO_2 of arterial blood.

Circulatory hypoxia - develops in conditions violating O_2 transport to the tissues - eg. reduced inflow of arterial blood in cardiovascular disease.

Histotoxical hypoxia - is determined by the reduction in the utilization of O_2 tissues (arterial blood PO_2 is normal and venous PO_2 is increased). The main indicator for the development and severity of the hypoxic condition is the magnitude of the partial pressure of oxygen in the alveolar air - PaO_2 and closest to him magnitude of PO_2 in arterial blood. The critical value of PaO_2 according to a number of authors from 27 to 33 mmHg.

Altitude hypoxia is ***acute*** or ***chronic***. In rapidly climbing high altitude (4000-5000 m.) the body does not have time to adapt and acute hypoxia occurs. Of such are threatened primarily aviators and mountain rescuers. In a smooth ascent to high (hikers, climbers and others.) provides time for acclimatization, which is a protection against acute hypoxia. By appearances of chronic hypoxia threatened longer or permanent residents of height. As a result of acute hypoxia occurs altitude sickness, and as a result of chronic - mountain sickness. Despite the importance of acclimatization, training, inherited properties, speed of ascent, a major factor in the occurrence of adverse effects and the ability to experience remains altitude **(Fig. 1)**.

Height (m)	Atmospheric pressure		Temperature (°C)
	(mb/hPa)	**(torr/mmHg)**	
Sea level	1013	760	15.00
100	1001	751	14.35
200	989	742	13.70
300	977	733	13.05
400	966	724	12.40
500	954	716	11.75
1000	898	674	8.50
2000	795	596	2.00
3000	701	525	- 4.49
4000	616	462	- 10.98
5000	540	405	- 17.47
10 000	264	198	- 49.90
15 000	121	90	- 56.50
20 000	55	41	- 56.50
25 000	25	19	- 51.60
30 000	11	8	- 46.64
40 000	2	2	- 22.80
50 000	0.8	0.6	- 2.50

Tab. 1. Relation between altitude, air pressure and temperature

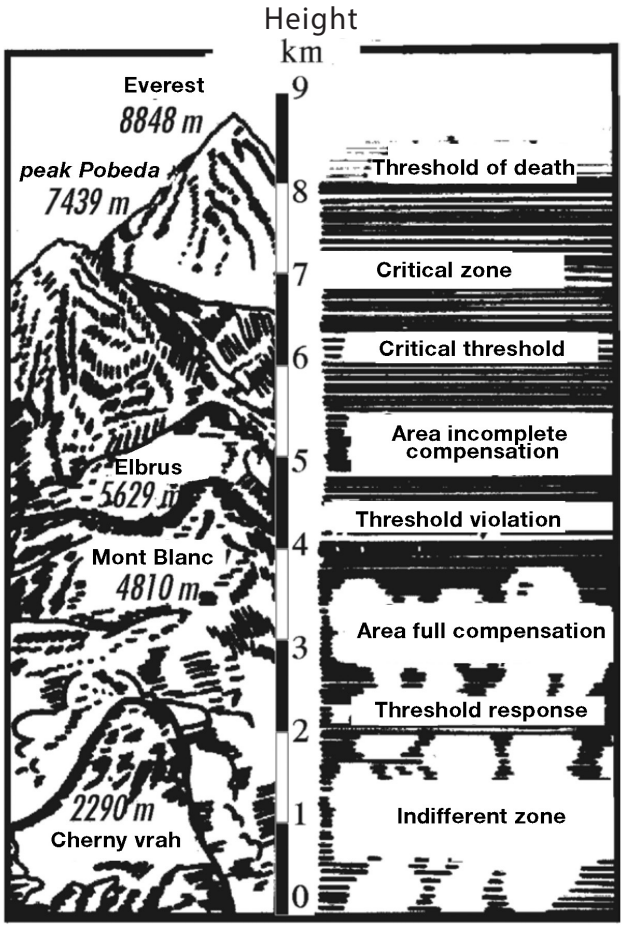


Fig. 1. Zones of tolerance height

The decrease in the partial pressure of O_2 in the arterial blood causes irritation chemoreceptor cells - a highly sensitive neuro-similar cells in carotid bodies of synocarotid and aortic vascular reflex zones. Hard afferentation of chemoreceptor is trigger of very reflective adaptive responses, that indicate the increase in cardiac output, stimulation of the pituitary-adrenal system and bulbar formations of the brain, including the cortex of the hemisphere.

The impulses sent through ninth cranial nerve directly turn to the respiratory control center in the cerebral cortex (the brain stem-medula oblongata). The last effect is to increase the frequency and depth of respiration, respectively quantity of ventilation air through the lungs, which helps restore arterial PO_2.

The complex functional changes to increase the ventilation is called **ventilatory acclimatization - Fig. 2.** Ventilatory acclimatization protects arterial PO_2 against the effects of reduced oxygen levels in the ambient air.

On the one hand, increased ventilation is a way to raise the arterial PO_2 to its values at sea level. However, increasing the release of CO_2 as a result of which the blood, and then the alveolar partial pressure of CO_2 falls. The decrease in alveolar PCO_2 allows to increase alveolar PO_2 and as a result, arterial PO_2 also increases **(Fig. 3)**.

On the other side, however, an increased release of CO_2 reduces the concentration of hydrogen ions (H^+) in the blood, determining the development of alkalosis. This "protective" alkalosis, respectively. disturbed acid-base balance (ABB) inhibit hypoxic ventilatory response.

Ability to compensate for respiratory acidic losses and balancing the ABB has increased excretion by the kidneys of bicarbonate, but this is a slow process - a few days. This explains less pronounced increase of ventilation with increasing altitude. The time that is necessary to complete (full) ventilatory acclimatization increased progressively with increasing height **(Fig. 4)**. Note and individual differences in time for acclimatization.

A longer period of residence (several years) in height can be determined reduced hypoxic ventilatory response associated with the development of other adaptive mechanisms for preserving tissue oxygenation: increasing capillary density, increased the capacity of tissues for gas exchange, increasing the number of mitochondria.

When height acclimatized people return to sea level, the process is "turned" - PO_2 increases to this sea levels and ventilation decreases. Reduced PO_2 at high altitude greatly reduces O_2 diffusion in the blood - **Fig. 5.** At sea level is counterbalanced terminal capillary PO_2 to alveolar PO_2 while at Everest (8848 m) for example full equilibration is not realized. This difference leads to greatly reduced **diffusion gradient** between alveolar and venous PO_2. With exercise or sport cardiac output and blood flow can rise and thus reduce the transport time of blood cells in the alveolar capillaries.

Difficulty in oxygen delivery to the tissues can be enhanced through: the adaptation of the cardiovascular and circulatory systems; increased production of erythropoietin and an increased concentration of Hb; increased tendency of O_2 to bind to hemoglobin; displacement of the oxyhemoglobin dissociation curve to right - the accelerated release of oxygen to tissues; increased acceptance and utilization of O_2; by biochemical changes that lead to increased mitochondrial function (increased levels of myoglobin).*

Research shows and other physiological effects of high altitude such as: changes in *brain function* - delayed motor, sensory and cognitive abilities, incl. reduced ability to learn new tasks, difficulty in presentation of verbal information, which leads to poor self-esteem and irritability; when residents of height

* Myoglobin concentration increases with exercise (at sea level) and correlated with the aerobic capacity of the muscle cells.

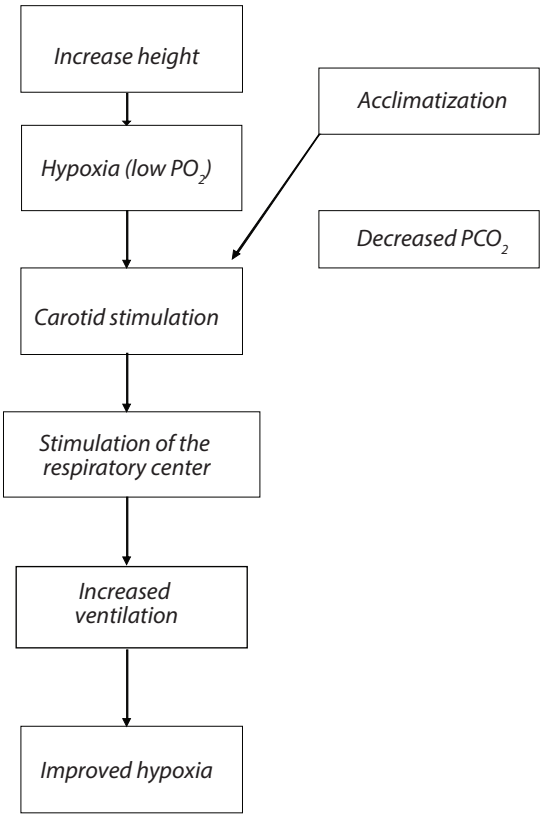


Fig. 2. Course of acclimatization (by J. T. Reeves)

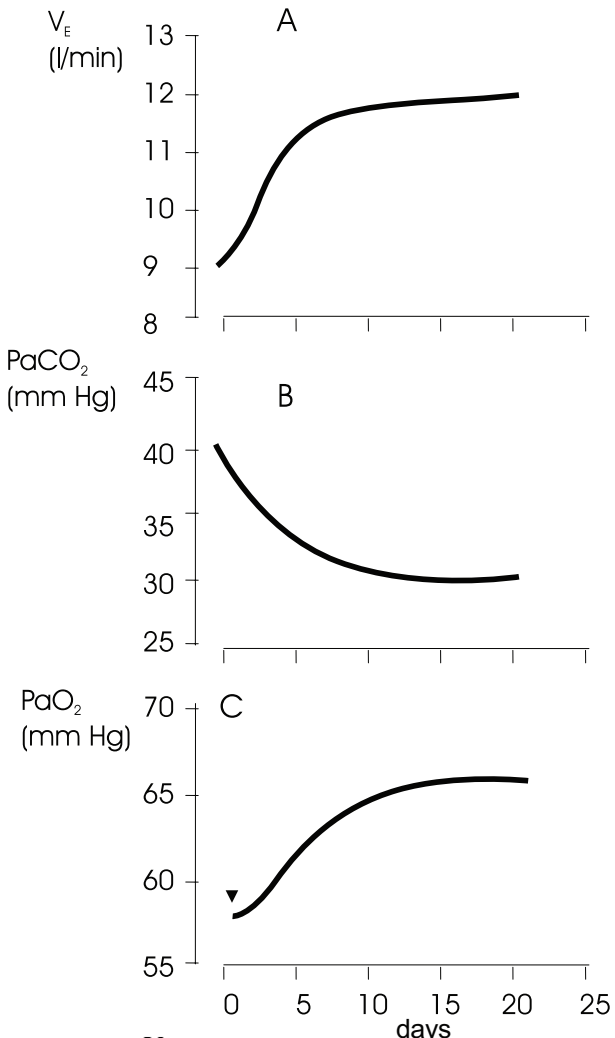
settled *periodic (Cheyne-Stok's) breathing* often during sleep - characterized by periods of increased respiratory rate (hyperpnoea) followed by periods of cessation of breathing (apnea), leading to hypoxia. This is more pronounced in subjects with higher ventilatory sensitivity to hypoxia. In acclimatization periodic breathing improves. The use of drugs and alcohol that suppress ventilation exacerbate hypoxia during sleep.

6.8.2. PATHOLOGICAL CONDITIONS AND DISEASES

Decompression disorders. Occurred in very quickly climb over 8000 m. The reason is the transition of tissue nitrogen from liquid to gaseous state. Characterized by pain in the large joints in the tissues around them, which disappear in lowering height. They also establish puffiness of the skin, rash erythematous character, itchy skin as from insect bites, neurological symptoms - headache, pallor, increased sweating, heaviness in the heart area and others. This condition is typical for working under increased pressure with subsequent decompression.

Explosive decompression. That is a peculiar form of decompression disorder, ie decompression occurring in a very short time (seconds), most often

Fig. 3. Ventilatory acclimatization at 4300 m above sea level (by J. T. Reeves)



V_E- ventilation
$PaCO_2$- alveolar CO_2
PaO_2- alveolar O_2

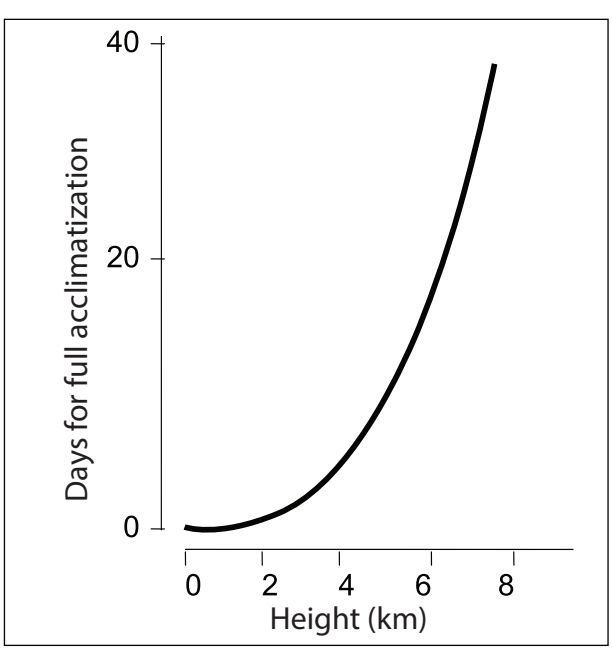


Fig. 4. Height and time for acclimatization (by J. T. Reeves)

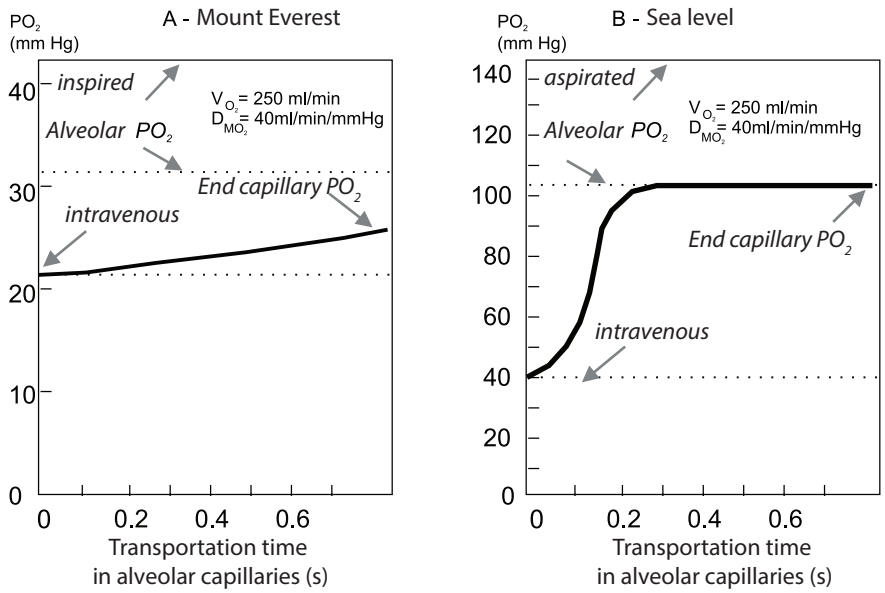


Fig. 5. Oxygen diffusion in the alveolar capillary

in sudden dehermetisation in the aircraft and spacecraft. The sudden transition from high (incabin) to low (outcabin) pressure occurs sharp expansion of gases in the body and mechanical damage to tissues and organs.

Disbarizam. In impaired hermetisation of the aircraft is possible sharp drop in atmospheric pressure, which can lead to increased pressure in a cavity spaces of the bodies containing gas, with development of meteorism, aerosinusitis, aerootitis, also altitude tissue emphysema (vapors in the tissues).

In a quick and chronic repeated decompression arise states of **disbarizam** long latency period as aseptic osteonecrosis and increased threshold of auditory sensation. These conditions are characterized mainly during decompression from high to normal atmospheric pressure. As manifestations of chronic disbarizam (but insufficient epidemiological support) must also disclose: signs of emotional instability and impaired short-term memory; EEG abnormalities; changes in the pigment and the vessels of the retina; reduced vital capacity.

At the **height meteorism** stomach swells, diafragmeta shifts up, the heart takes a horizontal position, breathing is difficult; reflex irritation of mechanoreceptors in the gut causes various gastrointestinal and autonomic disorders - infringement of coordination of movements, pallor, sweating, palpitations and others.

Boiling of the liquid (blood). At a height of 19,200 m in lowering the air pressure to 47 mmHg and a temperature of 37 ° C water (resp. blood) boil. Boiling occurs more quickly in regions of the body with low pressure - veins, right ventricle of the heart, the pleural cavity, subcutaneous tissue, which occurs at high altitude subcutaneous emphysema. The size and shape of the body will change, but clinical manifestations are often insignificant.

Retinal haemorrhages - many spread and self-injury at high altitude - sometimes affecting up to 40% of people at height 3700 m. Most often no symptoms, more common in people with other symptoms (most commonly headache) and at a sharp physical loading. No can prevent from Acetazolamide or Furosemid. Most often the absorption is spontaneous and occurs in about 2 weeks.

Altitude sickness (AS).

This concept join all the pathological conditions in humans quickly raised at altitude (mountain climbing, high-altitude flights, rise in hyperbaric chambers, etc.) associated with reduced PO_2. Appearance depends on many factors, such as frequency of ascents, length of stay, level of physical activity, individual susceptibility.

The development of AS to establish a number of disturbances in metabolism, with accumulation in the cells of lactic and pyruvic acid and violation of intracellular homeostasis- decrease in pH and changes to the selective permeability of cell membranes, which determines the early development of morphological changes. AS occurs most often in the first few hours after rapid ascent to a height of over 2,500 m. Described are two main forms of altitude sickness: **precolapse**, which occurs at relatively moderate deficit of O_2 - at height 5000-6000 m, at and a **collapse** - occurring in significant deficit O_2 at heights around and above 7000-10000 m.

Precolapse form of AS occurs in healthy people of the 5th to the 30th minute after climb 4000-6000 m. Persons noted poor self-esteem, headache, ma-

laise (fatigue); "warm feeling" in the body or head, vertigo, tinnitus; shortness of breath, wheezing; loss of appetite, often accompanied by nausea and vomiting. Changes in appearance and behaviour, facial features sharpen, eyes appear sunken, increased in early locomotor activity and euphoria are replaced with common inhibit, posture became rigid, all commands and instructions are perceived slow and perform reluctantly. If you do not provide O_2 feeding status could sharply deteriorate in loss of consciousness. Serious indications of deterioration are the reduction in heart rate and blood pressure, a decrease in cerebral blood flow.

Collapse form. In the mechanism of the development of the collapse response, plays an essential role and hypocapnia, which may explain the absence of therapeutic effect within the first minutes after O_2 feeding.

Loss of consciousness occurs suddenly or may be preceded by clonic cramps. Around and over 15,000 m hyperacute disease occurs, with loss of consciousness of 12-15 s after the rise. A height of 7000-7500 m course of the disease depends on the individual resistance - from 2-3 to 20-30 min or more.

Collapse form of AS develop without expressed harbingers - person loses adequately respect to the surrounding environment, does not notice the mistakes made in the implementation of psychophysical tests or solve simple arithmetic problems, noted a violation of the handwriting. Often found retrograde amnesia - do not remember the circumstances prior to loss of consciousness and even reported that they felt better.

Serious complication of AS is **high-altitude pulmonary edema**. It affects 0.5 to 2.0% of those over 2700 m, and is the most common cause of death from altitude sickness. It grows 6 to 96 hours after the climb. The pathogenesis associated with increased pressure in the pulmonary vessels and increased permeability of the same (pulmonary hypertension as a result of altitude hypoxia, is present in all individuals of high altitude, incl. and those who did not develop pulmonary edema).

The symptoms are like in AS, combined with reduced tolerance to physical loading and prolonged recovery period, shortness of breath, persistent cough, cyanosis of nails and lips. Early diagnosis of pulmonary edema is very important to reduce mortality.

The most severe complication of altitude sickness is a **high-altitude cerebral edema**. The pathogenesis is the same as AS - hypoventilation, increased cerebral blood flow and intracranial pressure, leading to cerebral edema. During disease progression was added neurological symptoms, incl. increased irritability, insomnia, ataxia, hallucinations, paresis, coma. Ophtalmoscopic examination may show papilloedema, retinal haemorrhages. Brain edema can be combined with pulmonary.

Treatment of AS: The most effective is lowering the patient at a lower height and to be carried out at a moderate speed. At impossibility is necessary to ensure oxygen or carbogen (oxygen with 3-7% CO_2) to overcome hypoxemia and restore breathing. Humidified oxygen therapy with 100% oxygen begins initially with a mask or nasopharyngeal probe. In severe cases are shown endotracheal intubation, artificial or assisted mechanical ventilation for a prolonged period.

Upon occurrence altitude pulmonary edema is shown giving large doses of corticosteroids - 30 mg/kg Urbazon (Methylprednisolone) for 6 hours for 48 to 72 hours. We need a proper water balance, normal import of liquids and in increased pulmocapillary pressure-diuretic.

Mastery of hypercapnia (if any) is achieved by correct disobstruction, applying respiratory analeptics - eg. Mikoren 1-4 ampoules at 250-350 ml. glucose-salt solution. Good effect is achieved with novphylline - 1-3 ampoules added to this mixture.

Cardiotonics should be used carefully, since in hypoxemia their therapeutic width narrows - recommended that about 1/3 of the normal dosage.

To reduce the risk of thromboembolic complications are recommended anticoagulant agents - heparin 5000 E in 6:00 h under the skin for 8-10 days, after which continues with indirect coagulant - antistenocardin 3x2 table or 3x1/2 tablets. Symptomatic drugs - analgesics and vitamins of the E, C, B_1, B_6, PP (for stimulation of the oxidation processes and to facilitate the resynthesis of codehydrase and cocarboxylase systems in the brain).

Mountain sickness.

For the first time - in 1590, data for this disease presents J. de Acosta to residents in the Andes at 4000 m - now called Monge's disease * - chronic mountain sickness at insufficient acclimatized residents, but not in permanent Andes residents. Men are affected more often than women. It is characterized by a plethora, cyanosis, an increase in the mass of red blood cells, neurological symptoms - headache, dizziness, somnolence and memory impairment. Sufferers often develop corpulmonale, due to increased pressure in pulmonary vessels and reduction of oxyhemoglobin saturation. The pathogenesis is not fully understood - studies show low hypoxic ventilatory response, pronounced hypoxemia,

* Carlos Monge Medrano (1884-1970), Peru. Institute of Biology of the Andes

increasing during sleep, high-HB concentration and increased pulmonary artery pressure. Many of the symptoms can be managed when driving at sea level. This change (decrease) hypoxic stimulus for the production of red blood cells and pulmonary vasoconstriction. The therapy involves the submission of O_2 during sleep (to control the hypoxia), Medroxyprogesteron (respiratory stimulant) - at 10 weeks therapy of it improves ventilation, hypoxia and reduces the number of red blood cells.

6.8.3. PREVENTION OF ALTITUDINAL DISABILITIES

Increasing resistance to human hypoxia means preliminary training in natural and artificial hypoxic conditions - alpine climbing or training in hyperbaric chambers. Stay in alpine conditions for several days or weeks to develop height resistance allowing trained to rise to a height of 8500 m without oxygen and also endure oxygen starvation of height 7000 m within a few days. So acquired acclimatization in individual fluctuations kept for relatively short period of time and gradually disappears. Before or during the climb high ASL is useful adoption of Diamox (Acetazolamid) - it inhibits the action of carbonic anhydrase in the kidneys, which lead to acidosis in the blood and stimulate breathing.

Health considerations in organizing work in high altitude. Workers at high altitude have to spend a few days of 3000-4000 m, and then a few days at sea level. The most acceptable options to shift modes are: 7 days work and seven days rest (or 10 days working with 10 days rest). Thus not reached full acclimatization at high, and also full disaclimatization at sea level.

Medical contraindications for work at high altitudes are chronic bronchitis, COPD and asthma; hypertension, anemia, polycythemia; diseases of the gastrointestinal tract; mentally unstable persons. Appropriate tests are: spirometry, lung X-ray, assessment of physical fitness, ECG when resting and after physical load. Altitudinal testing the flight staff carried out in hyperbaric chambers. Assess tolerance to moderate hypoxia, imitate the conditions of a height of 5000 m. The rating is "good" and "reduced - I, II, III degree." At a reduced assessment is not allowed airmanship and repeat the test.

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6.9 INCREASED ATMOSPHERIC PRESSURE

The normal functioning of the human body is in a relatively narrow range of barometric pressure. At high atmospheric pressure leading to physiological stress and related illnesses are subjected and ground workers at digging tunnels requiring use of compressed air or operating caissons (laying foundations of bridges, water towers, oil drilling, etc.). Also personnel of hyperbaric chambers used to treat patients (operations with hyperbaric oxygenation, presence of treatment with recompresion etc.). More common is the high barometric pressure under water - divers (eg. removing the pearl mussels, athletes) divers using aqualungs, divers performing various activities at different depths (eg. 300 m, and more in the exploitation of oil drilling in the sea).

Historically one of the first practical methods for underwater work are created by Smeaton (1778) underwater bells predecessors of current caissons - used then and now as laying the foundations of bridges (vertical caisson), and in digging of underwater tunnels (i.e. horizontal caissons). In 1819 in England created the first suits with air supply pressure - from the surface to perform underwater work by divers. At the same time appeared the first messages (Triger, 1841) for decompression disabilities (caisson disease) when working in horizontal caissons. In 1908 the English physiologist J.S. Haldane developed the first decompression tables for the prevention of caison disease, known as the DCIEM tables * - based and currently used tables.

6.9.1. TOXIC EFFECTS OF INCREASED ATMOSPHERIC PRESSURE

It is normal atmospheric air contains 20.93% oxygen. The human body is adapted to breathe atmospheric oxygen at a pressure of about 760 Torr (760 mm Hg) (160 Torr partial pressure of O_2) at sea level. The organism is able to withstand a relatively wide range of variations in pressure of oxygen without significant disability for a short period of time. Prolonged exposure can lead to **oxygen toxicity**.

On the surface of the earth person can breathe continuously without risk 100% oxygen between 24 and 36 hours. Then oxygen exerts its toxic effect on the lungs (the effect of Lorrain-Smith). Symptoms of this toxicity are substernal chest pain; dry, nonproductive cough; decline in vital capacity; loss of production of a surfactant. In continuing exposure establish microhaemorrhage with subsequent formation of permanent fibrosis in the lung. All stages of oxygen toxicity to microhaemorhagic state are reversible formation of fibrosis, the process becomes irreversible.

Oxygen can be breathed at a pressure lower than 0,6 ata* without ill effect. When 100% oxygen is inhaled under 2 ata (10 m pressure seawater), early signs of oxygen toxicity is manifest after about 6 hours. The measurement of vital capacity seems to be the most sensitive indicator of early oxygen toxicity.

At a pressure higher than 2 ata, pulmonary oxygen toxicity loses its primary importance on account of cerebral oxygen toxicity, a major manifestation of which seizures. If one should breathe 100% oxygen at a pressure of 3 ata for more than 3 h, the probability to get fit type grand mal is significant. There are some factors that enhance the toxicity of oxygen and reduce the seizure threshold: physical exercise, use of steroids, temperature conditions, cooling, acceptance of amphetamines, hyperthyroidism.

Taking medicines that suppress the production of seizures, such as phenobarbital or phenytoin, did not reduce damage to the brain and spinal cord, if the pressure or time are exceeded **(Table. 1)**.

Carbon monoxide can be polluting the atmosphere at work in caisson or under water. Most often sources of internal combustion engines used for compressors or other devices near them.

Early symptoms of carbon monoxide poisoning include headache, nausea, dizziness and general weakness. Carbon monoxide binding to hemoglobin block oxygen transfer and has a lethal effect. Recent studies indicate, however, the direct effect of CO on cell membranes, and induced lipid peroxidation (a major cause of death) can be interrupted only by inhalation of oxygen.

* DCIEM - Defence and Civil Institute of Environment Medicine

* Ata - absolute atmosphere equal to the sum of the pressure of the air column at sea level and that of the water column (ie. additional pressure - ati). 1 atm (physical or normal) = 1,013 at (technical) = 1,033 kg/m^2 = 10,33 m of water column = 760 Torr (760 mm Hg) = 1,013 bar = 1013 hPa (unit SI)

It must be ensured taking air at a height above the tailpipe of the compressor. To monitor for possible change in wind direction.

Carbon dioxide is a normal product of metabolism and is eliminated through the lungs. Different types of respirators, however, can disrupt its separation or cause high concentrations in the breaths of air divers.

At high concentrations (above 3%) it can cause errors in the ability to assess what initially appears as unjustified euphoria, while prolonged exposure such as depression. If the concentration continued to increase (above 8%) could be observed loss of consciousness **(Table. 1)**. At a partial pressure above 40 mm Hg, carbon dioxide and deteriorating exacerbating the effect of the impact of nitrogen narcosis. When breathing 100% oxygen at a pressure above 2 ata, with the increase of the CO_2 concentration the risk of obtaining a seizure (convulsion oxygen) greatly increases.

Nitrogen is an inert gas that is related to the normal metabolism in the body.

By increasing the atmospheric pressure, the concentration of nitrogen in the blood increases, and it acts as an aliphatic anesthetic (nitrogen narcosis), the level of anesthetic effect depends on the ratio of the solubility of the oil/water. Nitrogen causes a slight swelling of the cell membrane of the neurons, which increases its permeability with respect to the ions of sodium and potassium. Violations during normal depolarization/repolarization is the basis of clinical symptoms of **nitrogen narcosis**.

Work in water depths up to 50 m can be achieved with compressed air (78% nitrogen). At depths greater than 50 m, for avoiding effects of nitrogen narcosis is most commonly used mixture of helium and oxygen.

6.9.2. OTHER EFFECTS OF THE INCREASED PRESSURE

In the pressure change in the external environment during compression there occur the so-called. **disbarizam** - no mean while change in the pressure

Table 1. Toxic effects of gases on divers

Gas	Reasons (sources)	Symptoms
Narcosis of inert gas	■ Increased partial pressure of inert gas	■ Dizziness ■ Diminished ability to think clearly ■ Loss of fine dividing power ■ Euphoria ■ Slowed reflexes ■ Impaired ability to assess ■ Paresthesia ■ Progressive depression ■ Auditory/visual hallucinations ■ Memory loss ■ Loss of consciousness
Oxygen toxicity ■ CNS (acute) ■ Pulmonary (chronic)	■ > 1,3 ata partial pressure of oxygen ■ The risk increases with increasing partial pressure ■ Prolonged exposure of> 0,5 ata oxygen	■ Disturbances in vision ■ Reduced visual acuity ■ Nausea/vomiting ■ Twitch ■ Irritability/fatigue ■ Dizziness ■ Convulsions (may occur without prodromal symptoms; no long-term consequences) ■ Chest pain when inhaling ■ Breathing difficulties ■ Cough Progressive dyspnea
Hypoxia	■ Hyperventilation prior to diving and hold air ■ Wrong gas mixture	■ Loss of consciousness (without symptoms)
Toxicity CO_2	■ Constructioin of tools ■ Increased gas density ■ The increased partial pressure of oxygen blunted response to CO_2 ■ Increased content of CO_2	■ Increased respiratory rate ■ Asthma ■ Headache ■ Loss of consciousness
Toxicity CO	■ Using inappropriate means to grease the compressor (flammable) ■ Error compression ■ Incorrect position of the hole for air intake (eg. near the compressor)	■ Headache ■ Nausea/vomiting ■ Loss of consciousness

of the external environment and cavities of the human body - primarily in the middle ear and the skull as a whole. This is expressed with symptoms - pain in the ears, in the eyebrows and forehead, barotrauma - bleeding and rupture of m. tympani, bleeding from the nose and ears. These conditions of the middle ear occur most frequently in abnormal conduction of the Eustachian tubes (inflammation, allergy). The earliest symptoms are decreased auditory sensation, followed quickly by severe pain in the ears (to intolerance). It may also be observed and dizziness, headache, nausea, irritation as a consequence of the inner ear.

When operating at high pressure (and its alignment) man adapts relatively quickly, but there are changes in voice timbre (tone deaf), a decrease in auditory sensitivity, as well as skin sensitivity, dryness of mucous membranes of the upper respiratory tract. The diaphragm is lowered, increased vital capacity, reduce the frequency of breathing, heart rate and cardiac output (at rest). Efficiency is slightly increased, there was a slight increase of muscle strength and endurance.

As regards the influence of compression on the **cardiovascular system** describes a decrease in systolic pressure and increase in diastolic (during decompression - back). Are described and ECG changes with reversible character - bradycardia, a decrease of P and T increases peak.

Studies on the **blood changes** establish so-called **caisson anemia** - reduced content of HB, erythrocytes in an increased number of leukocytes and lymphocytes and the development of reticulocytosis. These results, however, not always be confirmed. There are also conflicting results regarding changes in metabolic processes with high atmospheric pressure. Some authors reported and amplified gastric motility at a reduced secretory activity; reduced diuresis at relatively low compression (1-2 ati) and increased at higher pressure (4-5 ati); changes in the absorption of phosphorus and glucose in the small intestine, etc., i.e., a variety of changes, but not always verified and substantiated pathogenetically exactly.

6.9.3. DECOMPRESSION

Decompression disorders may impair working under high pressure through one of **two primary mechanisms**. **The first** is due to the adoption of the gas during compression and formation of bubbles in the tissues and vessels (venous, arterial) during and after subsequent decompression. It is generally accepted that metabolic gases, oxygen and carbon dioxide have no attitude (and relevant) to the formation of blisters.

During the compression tension of breaths and located in the arterial blood inert gas (mostly nitrogen) increases. The inert gas (es) is transferred to the tissues until a balance between its tension in the inspired air, arterial blood and tissues. The time for this ranges from 30 min to more than one day, depending on the tissue type and the inert gas, and also depends on:

- ✓ Blood supply to the tissue;
- ✓ The solubility of the inert gas in the blood and in the tissue;
- ✓ The spread of inert gas through the blood to the tissue;
- ✓ The temperature of the tissue;
- ✓ The workload of the tissue;
- ✓ Tension of tissue carbon dioxide.

In subsequent decompression process is reversed, and the rate of release of gas is determined by the above factors, but the process proceeds more slowly compared with its receipt. In slow decompression gas is exhaled, but in fast form bubbles in the tissues and blood vessels.

The second primary mechanism by which decompression can cause disabilities is barotrauma (it can occur both compression and decompression).

Classification. Until recently decompression diseases are classified as:

– barotrauma

– cerebral arterial gas embolism

– decompression disabilities – 3 types: Type 1 (pain and rashes on the skin); Type 2 (all other events); Type 3 (appearances as cerebral arterial embolism, and the decompression disabled) - **Table. 2.**

A new classification of decompression illness was established because of the difficulty in the distinction between cerebral arterial gas embolism and brain decompression complaints and between decompression disabilities Type 1-3. Now all decompression illness only be classified as such **(Table. 3).**

Barotrauma. During each **compression** space filled with gas can be included in barotrauma and this applies particularly to the ears. Most often damage the tympanic membrane and the middle ear. This type of injury occurs more frequently in workers with pathology of the upper respiratory tract and Eustachian tube dysfunction. The possible consequences are increased pressure in the middle ear and/or rupture m. tympani. Often occur ear pain and conduction deafness. Upon entering the cold water in the middle ear in impaired membrane can occur nausea, vomiting and dizziness transient. More often, vertigo (and possible also sensoneural deafness) is the result of barotrauma of the inner ear.

Paranasal sinuses are also often affected, usually due to blocked foramen. To local pain here is added

Tab. 2. Clinical picture of the main types of decompression disorders

Type 1	Type 2	Type 3
Mainly affects the limbs and skin. The most frequently manifests as: ■ Acute pain around the big joint. Can begin immediately or 12th hour after decompression. ■ Sometimes it is accompanied by hives and itchy skin	*It affects the central and peripheral nervous system.* The most frequently manifests as: ■ Stabbing and needles "paresthesia ■ Hypoesthesia ■ Dizziness ■ Hyperreflexia ■ Ataxia ■ + Babinska ■ Paralysis or weakness in the limbs ■ Headache ■ Seizures ■ Vomiting ■ Loss of vision or visual field deficit ■ Incontinence ■ Impaired speech ■ Tremor ■ Coma *Pulmonary System:* ■ Substernal pain ■ Chest tightness ■ Severe cough ■ Dyspnoea ■ Pulmonary edema *Cardiovascular:* ■ Arrhythmia ■ Hypertension May have significant consequences such as: ■ Obstruction of vessels and tissue infarction, which is sometimes accompanied by: – haemoconcentration, – changes in osmotic pressure or fat emboli, – hypercoagulable blood	*Aseptic necrosis of bone, the most common* ■ The head or the body of the humerus ■ More rarely, the long end of the femur or tibia head It was found 6 to 60 months after decompression and is asymptomatic, the inclusion of the joints - mostly symmetrically. ■ Increased frequency of violations in memory ■ Retrograde amnesia ■ Emotional instability and others neurotic and psychotic symptoms in divers spent decompression sickness

and epistaxis. Rarely are damaged lung, teeth, intestines and others.

Decompressive barotrauma occurs rarely compared with compressive, but there heavily consequences. It operates primarily in the lung and inner ear. The gas discharged from the damaged lung may pass through the interstitium to the hilus of the lungs, mediastinum and even to the underlying tissues of the neck and head. This interstitial gas can cause dyspnea with substernal pain and cough with sputum with blood. The compression of the heart is extremely rare. Gas from the traumatized lung can pass also in the pleural space (pneumothorax) or in the pulmonary veins (and possibly to obtain arterial gas embolism).

Decompression disorders ("caisson disease") due to blistering in the tissues or blood vessels, is typically manifested within a few minutes or hours after decompression. The natural history of many of these decompression illness ends with spontaneous improvement. In some cases, however, spontaneous recovery is partial and needs treatment.

The most common manifestation of decompression disease is a form of influenza-like illness. Other common complaints are different sensory disturbances, local pain, especially in the extremities and other neurological symptoms associated with sensory and motor fatigue (fewer prejudice skin and lymphatic system). For some groups, the most common complaint is pain - in fixed joint or joints,

Tab. 3. Revised classification system for decompression illness (in Dees F. Gorman, 1998).

Duration	Evolution	Symptoms	
Acute	Progressive	Musculoskeletal	
Chronic	Spontaneous recovery	Skin	
	Static	Lymphatic	Decompression
	Repeated	Neurological	disease + or -
		Vestibular	Availability of
		Cardiac and respiratory	barotrauma

back pain and rarely can be observed unspecified crumble pains.

Manifestation of decompression illness is diverse. Any disease of working in conditions of high pressure that occurs to 24-48 h post decompression may be associated with decompression (until proven otherwise).

The development of treated decompression illnesses is different. In some cases the residual phenomena can be overcome in the next 6-12 months, while others complete recovery does not occur.

The treatment of the majority of decompression illness is rekompresiya in hyperbaric chambers. An exception to this rule is barotrauma (when not cause arterial gas embolism). In pa**tients with ear barotrauma** required number of outpatient examinations, application of means, ability to relax the nose, pain, and suspicion of barotrauma and inner ear - compulsory treatment of bed.

In barotrauma to the lung of applying 100% oxygen at the level of the atmospheric pressure has a beneficial effect.

The pressure (rekompresiyata) to treat **decompression sickness** it is not definitely known, although a conventional first choice is 2,8 bar absolute pressure (282 kPa). In low affect of symptoms is prolonged by a further compression of 4 to 6 bar absolute pressure. Mixtures of oxygen and helium may be more effective in comparison with ambient air or 100% oxygen.

Again exhibiting symptoms of decompression sickness are frequent and most workers have to be recompressed. This continues until deviations are removed or until two consecutive courses of treatment had no positive effect. One year after it is held X-ray search disbaric osteonecrosis (aseptic necrosis) of long bones.

As a general effects of decompression sickness can be identified (in decreasing frequency): depression; problems with short-term memory; sensory symptoms; micturition and sexual offenses; and vaguely aches and pains.

Many of those working in conditions of high pressure can return to their work at least one month after decompression sickness. They should not be allowed to work but workers with pulmonary barotrauma or evidence of chronic severe injury to the inner ear.

6.9.4. PREVENTION OF COMPRESSION AND DECOMPRESSION DISABILITIES

To prevent **compression barotrauma** of the ear and paranasal sinuses should not be allowed compression at inflammation of nasopharynx, poor conduction of the Eustachian tubes, rhinitis. Compression early to be slow by systemic pressure equalization airways cavities with external pressure. In manifestation of deaf or ear pain, compression stops and reduced. It is repeated when symptoms disappear. At the reappearance of complaints compression is terminated.

To prevent the occurrence of **decompression barotrauma** to the lungs is necessary to meet the following requirements:

- use of properly scuba equipment;
- do not hold breath at exits on surface;
- do not allow people under water with cough, regardless of its nature;
- be strictly observed decompression schemes.

Medical prevention. Underwater work allowed people practically completely healthy, high physical and neuro-psychological resistance. Current medical examinations were 12 months. The commission includes therapist, ORL specialist, neurologist and ophthalmologist at indication and orthopedist and radiologist. It must be investigated Hb, by indication - leukocytes, B.S.R., X-ray of lung and large joints, oscillography.

Decompression. Schemes of decompression **(Table. 4)** determine the time for decompression of a person who has been exposed to high pressure, depending primarily on the depth, and duration of exposure, the gas mixture and others. None scheme of decompression can not guarantee absolutely avoid decompression sickness (bubbles form in any normal decompression), but the longer full decompression is, the more rare is decompression sickness.

Most modern decompression schemes are based on mathematical models that anticipate the development of decompression sickness <1-5% for workers.

Habituation or acclimatization was observed in divers and those working with compressed air, which makes them less sensitive to compression and decompression effects after repeated exposures. Tolerance can be seen in daily exposure for one week and lost after an absence from work between 1-4 weeks or at sudden increase in pressure (“addiction” does not mean a complete adaptation of the body).

Other preventive measures here are: quantity and quality of supply air pressure to meet the regulatory requirements, the use of pure oxygen or helium-oxygen mixture to a rapid decompression and removal of nitrogen narcosis; preventing cooling of the body; annealing and training; technological and sanitary-technical means for removing other harmful - oil vapors and aerosols, dust, metal aerosols (welding), insufficient lighting, noise, vibration, risk of physical injury, fire, etc .; protective professional nutrition with weight control; the possibility of constant medical supervision and use of hyperbaric chambers.

Tab. 4. Decompression tables. Norms for decompression in one-shift operation in caisson (a S.V. Alexeyev and V.R. Usenko, 1988; V.K. Navrotskiy, 1974)

Pressure in the working chamber in ati	First phase of decompression		Second phase of decompression	Total time in minutes
	Up to what level is reduced pressure in ati	Duration in minutes	Duration in minutes	
1,3	0,3	4	8	12
1,4	0,4	4	10	14
1,5	0,5	4	12	16
1,6	0,6	4	14	18
1,7	0,6	4	16	20
1,8	0,7	5	18	23
1,9	0,7	5	21	26
2,0	0,8	5	24	29
2,1	0,8	5	27	32
2,2	0,9	5	30	35
2,3	1,0	5	33	38
2,4	1,0	5	36	41
2,5	1,1	5	39	44
2,6	1,1	5	42	47
2,7	1,2	5	45	50
2,8	1,3	5	48	53
2,9	1,3	5	51	56
3,0	1,4	6	54	60
3,1	1,4	6	57	63
3,2	1,5	6	60	66
3,3	1,6	7	63	70
3,4	1,7	7	66	73
3,5	1,7	7	69	76
3,6	1,8	7	72	79
3,7	1,9	7	74	81
3,8	1,9	7	76	83
3,9	2,0	7	78	85

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6.10 GRAVITY, ACCELERATION, AND MICRO-GRAVITY (WEIGHTLESSNESS)[1]

Nowadays, aviation and spaceflight are essential high technological international activities. Data show that from 1960 to 1989 the average annual number of passengers in civil aviation regular underway flights has increased from 20 to 900 million. people. Now data are: 928 mill. in EC for 2015 - growth 22% to 2009; 1,7 bill. in World for 2012 - annual growth 5%. Air Force Aviation is a high part of the armed forces of most nations. Advances in aviation technology contributes to the development of space programs involving humans.

Impact on the health of the crew, passengers and astronauts than the reduced oxygen concentration in the air, have a number of physical factors such as reduced air pressure, heat stress, **acceleration, gravity, weightlessness**, vibration, noise, ionizing radiation and others.

6.10.1. GRAVITY AND ACCELERATION

Combination of gravity and acceleration occurring during flight in the atmosphere has a variety of physiological effects on the crew and passengers.

On the surface of the Earth gravitational forces act on all forms of human physical activity. The weight (mass) per person corresponds to the force with which the earth's gravitational field acting on it. The magnitude of the acceleration of freely falling object on Earth's surface g = 9,8 m/s^2.

Acceleration occurs when an object in motion increases his speed (accelerated). *Deceleration* leads to acceleration of the opposite direction.

There are three types of acceleration:

1. Linear - changes in speed without a change in direction;

2. Radial - change in direction without a change in the speed - an example of this type of accelerations are those that arise in the course of virage (flight in a closed circle) or making figures of the complex and aerobatics example loop.

3. Angle - changes in speed and direction.

In aviation applied accelerations most often expressed in the unit G, expressing the attitude of the applied acceleration to the gravitational constant (9.8 m/s^2).

The direction of acceleration against a human body is described through the three axes – x, y, z, where z is a vertical axis (parallel to the long axis of the body), x is pointing forehead/nape and y is oriented from side to side. Accelerations can be categorized into two main types: permanent and transient.

Constant acceleration. Prolonged changes in velocity leading to acceleration lasting more than 2 s may be established for changes in the direction of flight and speed of the aircraft.

The biological effect of constant acceleration is the result of deformation of the tissues and organs of the body, changes in blood flow and the distribution of body fluids.

In civil aviation acceleration axis z (G_z) can occur randomly and moderately during take-offs or landings or flying in turbulent environment. In these cases, passengers may fall short in a state of weightlessness, due to sharply downgrade (negative G_z). In unfastened seat belts that can lead to injuries.

Positive or directed to head acceleration axis z ($+G_z$) is the largest physiological problem.

Management of high-speed military aircraft and aerial acrobatics can generate much higher linear, radial and angular accelerations. Indicated are the positive accelerations - from 5 to 7 G lasted for 10 to 40 s. The crew can experience a sense of increasing the weight of the tissue and limbs even at relatively low levels of acceleration of +2 G_z.

The most important system of the body to determine its tolerance limit in response to + G_z is cardiovascular. The impact of positive acceleration on vision and mental performance due to reduced blood supply to the eyes and brain. This can cause temporary loss of peripheral vision to complete loss of vision or complete loss of consciousness. Cardiac arrhythmias occur after exposure to high levels of persistent + G_z acceleration, but they are usually minimal clinical importance in healthy and trained people.

Negative G_z rarely establishes for the design and implementation of aircraft. Physiological effects associated with -G_z originally lead to an increased vascular pressure in the upper half of the body, head and neck.

Constant accelerations acting at right angles to the long axis of the body are called transverse ac-

[1] As a problem of aviation and aerospace medicine

celerations and are relatively rare in most situations in aviation - for example in ejection. Accelerations in these operations are relatively small and the effects are less pronounced than those of the acceleration G_z.

The lateral accelerations axis ± G_y are much rarer than those in experimental aircraft.

The biological effects of acceleration on the degree of markedness are minor functional changes to severe functional and structural damage, loss of consciousness and death. There is a variety of subjective symptoms - feeling overrides, difficulty in movement to impossible to move, pain sensations, etc. Change are functions of external breathing - shortness, increased pulmonary ventilation, but can be monitored and atelectasis and puffiness in the lower parts of the lungs, decreased blood saturation with oxygen, developing hypoxia. As a stress factor acceleration causes early changes in the central nervous system - changes in EEG with phase character and appearance of delta waves. Described are the early phase changes in the functions of: endocrine glands - an increased level of catecholamines and norepinephrine; gastrointestinal tract - changes in the secretory and motor functions; urinary system - oliguria, but and polyuria; biochemical and morphological blood changes.

Transient acceleration. Many factors influence the tolerability of man to short accelerations. These are: the magnitude and duration of the applied forces; initial magnitude of the applied force; its direction and site of application. People can withstand much greater forces when they are perpendicular to the long axis of the body (the axes of x and y).

Physiological responses of the crew and passengers of brief (transitional) accelerations are essential for the prevention of air accidents and to protect the crew and passengers.

Human tolerance to sudden deceleration (often less than 1 s) of occurrence of mechanical* disabilities resulting for deformation of tissues and organs and consequently for blow to the structural parts of the aircraft's is variable value. The nature of the mechanical trauma depends on the applied forces (whether involve penetration or blunt impact). The forces are divided into manageable, traumatic and fatal. Tolerated forces lead to mild traumatic injuries (scrapes and bruises) and traumatic forces - to moderate or severe injuries, which, however, does not always lead to complete loss of capacity. It has been found that acceleration of 25 G for approximately 0,1 s are tolerated limit for the axis + G_z, those of 15G for 0,1 s are limit to the axis - G_z.

* Mechanical damage (injuries) are essential in the short-term accelerations

6.10.2. PREVENTIVE MEASURES

To enhance crew tolerance to the effects of acceleration and hyper gravitation use complexes of physical and physiological methods and means:

Physical (mechanical) methods:

- safety (anti over load) clothings;
- adjustable seats;
- systems for breathing oxygen under pressure;
- immersion systems.

Physiological methods:

- hardening and physical training;
- training in a centrifuge;
- muscle tension;
- procedures (maneuvers) M-1 and L-1 (controled intension breathing);
- adaptation to hypoxia;
- pharmacological agents;
- professional selection and medical supervision.

Preliminary medical examination of crew members to detect diseases are essential in aeromedical program.

Crews must be trained to recognize different physiological conditions, which can reduce their tolerance to G stress - fatigue, dehydration, heat stress, hypoglycemia and hypoxia.

The crews of high-speed aircraft used three types of procedures (maneuvers) to reduce the harmful effects of constant acceleration during the flight. *Forced contraction of the muscles*, especially in the legs, abdomen and shoulder girdle increases pressure on blood vessels, contributing to a return venous blood, reducing its landfill in the lower half of the body, resulting in increasing resistance to accelerations in 1.5-2.0 G. *Exhaling against a closed glottis*, called the Valsalva maneuver (or M_1 procedure) can increase the pressure in the upper half of the body and increase it in the chest. These results, however, are short-lived and may even be harmful if the procedure lasted longer due to reduced return of venous blood to the heart. **Breathing against positive pressure** (50-70 mm Hg of air mixture with 40-50% oxygen) is another method of enhancing intrathoracic pressure. Through it increases blood flow to the eyes and brain and reduces hypoxia.

Military crews use a range of training methods to increase the G-tolerance as a spin in a centrifuge that generates different + G_z acceleration. This workout helps to increase tolerance to accelerations with 1,6-1,8G. Hardening of the body and physical exercise, and especially to increase the endurance of the body are also considered effective.

Adaptation to hypoxia (in alpine conditions or hyperbaric chambers) also supports resistance to

acceleration - indicate elevations up to 2.0-2.4 G. Developed methods and criteria for professional selection of persons with individual qualities of reduced susceptibility (increased resistance) to accelerations.

Developed and protective clothing (pneumatic inflatable anti-G suits) to reduce the effects of exposure to + G_z. Under the impact of the inflated air in the suit is increased tissue pressure on the lower limbs. This keeps the peripheral vascular resistance, decreases the collection of blood in the abdomen and the lower limbs and the displacement of the diaphragm down, which prevents an increase in the vertical distance between the heart and brain. Survival in plane crashes depends on efficient restraint systems and maintain the integrity of the cabin and cockpit. Seats that reduce energy and lining on the back of the seats are part of the structure of the aircraft, which restricts injuries. Another technology involves creating components that absorb energy and improve the structure of the seats in order to reduce mechanical damage, and improving tolerance to accelerations.

The use of pharmacological agents (vasoconstrictors and vasodilators, ganglioblockers, stimulants CNS, hormones) as well as of immersions systems (insertion of the body into a container with liquid - water) are experimented and has not found practical application.

6.10.3. MICROGRAVITY (WEIGHTLESSNESS)

As a result of spaceflight has published a large amount of literature that reflects the physiological changes at teams during and after the flight. The reason for these deviations indicate exposure to weightlessness or microgravity. These changes are transient - a full recovery from several days to several months after return to Earth, however, can not predict the consequences after a flight lasting 2-3 years and more. The most significant deviations can be categorized into several major groups - cardiovascular; musculoskeletal; neuro-vestibular; haematological and endocrinological.

Cardiovascular system. Microgravity can induce instability of blood pressure and pulse, but they are not very significant - not harms or work crew during the flight. Almost half of the astronauts, however, immediately after the flight complained of dizziness and stunning, some receive fainting (syncope) or are near to fainting (pre-syncope). The reason is the drop in blood pressure in vertical position due to the impact of the Earth's gravitational field and abnormal compensatory mechanisms of the body. These conditions are transient and without consequences, but after a very long flight, they can be much more serious (even fatal).

Preventive measures for these conditions of the cardiovascular system are primarily focused on physical exercise during the flight of a walking path, exercise bike and simulator rowing, they are combined with creating a negative pressure in the lower half of the body (in astronaut spacesuit). Used appropriately designed and drinks with high saline content.

Musculoskeletal system. In all astronauts settled a loss of muscle mass (atrophy) irrespective of the duration of the flight. Particularly affected are the muscles of the arms and legs - decrease muscle strength and endurance. Explanation for these changes is that work in conditions of weightlessness are carried out almost no muscle effort.

Prevention here include: exercise during the flight, electrical stimulation of the muscles and anabolic drugs (testosterone or testosterone-like steroids). These means, however, only postpone muscular atrophy.

It establishes a slow but continuous loss and bone mass (about 300 mg per day, or 0.5% of the total content of calcium in the bones, in a month). Calcium loss and bone erosion may be a limiting factor during the flight. Bone loss may lead to an increased risk of fractures, and the release of calcium in the urine increases the risk of lithiasis.

So far, not been found effective measures for preventing the loss of calcium. Have tried a number of means including exercise (walking path, exercise bike and rowing simulator) and additional intake of calcium, vitamins and medications (eg. diphosphonates - drugs helps prevent bone loss in osteoporosis). The most effective, but practically difficult to achieve is the creation of an artificial gravitational field, either continuously or temporarily turning the aircraft.

Neuro-vestibular apparatus. More than half of the astronauts and cosmonauts suffer from a condition called motion sickness in space (SMS - Space Motion Sickness). Symptoms arise due to violation of the functional interaction between analyzers. Neuro-vestibular complaints are caused by disorders in the function of the inner ear due to microgravity. CNS to be sent wrong signals by analyzers or recent interpreted incorrectly. Symptoms include heartburn, nausea, vomiting, headache and drowsiness. The complaints are exacerbated when rapid movement of the head. SMS is manifested several minutes to several hours after start and recover fully within 72 hours. Therefore, the first three days of the flight is not planning any work in outer space.

Increasing adaptation of astronauts before flight by training with rotating chairs with little effect by preventing SMS. Antihistamines administered by in-

jection, are particularly effective as a treatment, but its efficacy as a prophylactic is not proven.

Other neuro-vestibular symptoms are dizziness, balance disorder, illusions of astronaut himself for time, spatial perception, movement, colors, making it difficult to work during the flight and its movement for a short time after the flight. The mechanisms for these phenomena are complex and not fully understood. They have not been found effective preventive measures.

Hematologic. Microgravity effect on red and white blood cells. Astronauts decreased by 7-17% of the number of red blood cells at the beginning of the flight. This condition remains to normal 4 to 8 weeks after the flight. This phenomenon is not of clinical significance, but would become a serious problem during long flights, as it will lead to the development of anemia.

Microgravity also affects white blood cells. There is an overall increase in their number, mainly of neutrophils and reduction of lymphocytes. There were no illnesses directly related to them. It is not known whether also at a long flight of these changes will deepen, which would lead to disorders in the immune system. It has been described in some cosmonauts changes in haemostatics and thrombocytopenia in post-flight period.

Endocrine system. It was found that during the flight occurs several changes in the liquid medium and minerals in the body, which are due to changes in the endocrine system. In general, there is a loss of the total volume of liquids in the body, as well as sodium, calcium and potassium. Although the exact mechanism of this phenomenon has not been defined, some explanation give found changes in the levels of various hormones - growth secretion of adrenaline, renin and aldosterone, but as the results of the tests at various astronauts are not unidirectional, it may be formed single hypothesis about the causes of these physiological disorders. It is not proven and deterioration of health of astronauts and quality of their work on the basis of the changes described. It is not known and what could be the consequences of these violations in a very long flight.

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7 CHEMICAL FACTORS

D. Tsonevsky

7.1 INDUSTRIAL TOXICOLOGY - BASIC ASSUMPTIONS, PARAMETERS OF TOXICITY. PREVENTION OF OCCUPATIONAL POISONING

Impact of chemicals on workers is possible in many professions during the production and processing of raw materials in the preparation of industrial production, operating in transport, in agriculture. Effects of toxic substances on the health status of workers studied the production toxicology.

Toxicology is a science that studies the interaction of toxins in the body (from toxicon - poisons and logos - knowledge). In practical terms toxicology science that prevents, detects and removes early proximal and distal consequences of the action of harmful substances. All substances are potentially toxic. The concept of poison defined more by Paracelsus (1493-1541), which states that **"All substances are poisonous and nothing is devoid of toxicity. Only the dose makes the poison unnoticed. "** Poisons are defined as substances that come through physiological pathways in the body in relatively small amounts, enter the chemical or physico-chemical interaction with tissues and under certain conditions violate health*. American Association of Industrial Hygienists - ACGIH gives quantitative valuation of poison. According to experts the association, of poisons are accepted substances which, after treatment of experimental animals (white rats weighing 200-300 g) for the solutions and emulsions in doses of 50 mg/kg of weight or in smaller quantities, and to gases and vapors that at concentrations of 2 mg/l air and exposure of 60 min, after 48 hours causing the death of 50 percent or more of the animals treated.

The objectives of clinical and experimental studies in toxicology are to determine: a) the harmful effects of substances, their toxicity; b) doses, when occurring toxic effects i.e. the relationship between the dose-response; c) the potential for injury or disease, depending on specific conditions, ie to assess the health risk.

In toxicology distinguish between the concepts degree of toxicity and health risks. Highly toxic substances that are hermetically sealed, creating little risk or presents no danger. When these substances are used by workers in a confined space in impaired pressurization of equipment, without proper precautions, the health risk becomes significantly larger. Ie. how dangerous a substance in the environment is determined by technical equipment, processes, pressurization of equipment, ventilation of production premises, subject to the requirements relating to technical safety, the use of personal protective equipment, etc.

Manufacturing poisons are substances that can be raw materials, intermediate or finished products must waste from industry and agriculture. Health risk are exposed workers of: the metallurgical industry (CO, sulfur gases and metal aerosols); ore mining ("explosive gases" - CO, CO_2, NOx); metal products and machinery (toxic gases, vapors and aerosols in acid treatment of metals, galvanization, nickel, chromium); chemical industry (chlorine, ammonia, sulfur gases, toluene, phenol, formaldehyde, styrene, nitrogen oxides, acids and bases); agriculture (different types of pesticides, as PhOC, ChlOC, carbamate and dithiocarbamate preparations, nitrophenols, mercury compounds) and others.

Classification (in Ch. Becker). For this purpose, the following important criteria: **a) their origin** - the manufacturing poisons are mineral and biological-plant, animal, microbial; **b) a chemical structure** (primary classification) are divided into inorganic and organic substances. Among the most commonly occurring inorganic substances - metals and non-metals, in manufacturing concern: halogens (chlorine, bromine, fluorine), sulfur compounds

* Poisons are marked also as xenobiotics (from Greek: xenos - foreign and bios - life)

(sulfur dioxide, hydrogen sulfide, sulfuric acid), compounds of nitrogen (ammonia, nitrogen oxides, nitric acid), the compounds of the cyano acid (hydrogen cyanide, potassium cyanide), heavy and rare metals (lead, mercury, manganese, chromium, cadmium, vanadium, etc.). The most common organic substances include: hydrocarbons: aromatic compounds (benzene, toluene, xylene, phenol), butyric compounds (gasoline, methane, ethane, ethylene, acetylene, chlorinated aliphatic hydrocarbons) and alcohols of butyric row (methyl, ethyl alcohol, ethylene glycol, etc.); **c) a physical condition** are divided into: solids, powders, gases, vapors, aerosols, liquids (lead for example is harmless in the solid state, with intermediate toxicity is as a powder and is highly toxic as an aerosol) **d) in the medium of the action.** Some toxic substances are found in specific environments. For ex. sulfur and nitrogen oxides - into the air, trihalomethanes - in water and nitrosamines - in food. Mid of action to some extent determines the degree of risk and risk population; **e) specific toxicity** - substances are divided primarily neurotoxic, hepatotoxic, nephrotoxic, cardiotoxic etc. f) **mechanism of effect on the body** - toxins are divided into cause: tissue asphyxia (cyanide, CO), hypoxia (inert gas), blocking enzyme systems (PhOC, carbamates), etc .; **g) as the dominant health effects** are divided into: toxic active (acute or chronic poisoning); allergic acting (chlorinated hydrocarbons, mineral oils, diisocyanate, toluene); gonadotropic(chloroprene, manganese, carbaryl, formaldehyde, phenol, lead, mercury, dichloroethane, carbon disulfide, granozan, sevin, etc.); teratogenic (dioxins, lead, mercury, manganese, chloroprene, vinyl chloride, thiocarbamates, derivatives of phthalic acid, ethanol, etc.); mutagenic and carcinogenic (3,4-benzpyrene, nitrosamines, urethane, benzene, acrolein, vinyl chloride, formaldehyde, aflatoxin, lead organic compounds, naphthylamine, asbestos, vinyl chloride, arsenic, chromium, nickel, beryllium, benzidine, chlorinated hydrocarbons, etc.).

7.1.1. FACTORS DETERMINING THE TOXICITY OF SUBSTANCES

The toxic effect depends on the condition of the organism during its interaction with the toxins in environmental conditions. On these processes affect these groups of factors:

1. The duration, frequency and route of exposure. The severity of toxicity impairment most often related to the duration and frequency of exposure. Route of administration of substances in the body are also often determines the toxicity - eg. ethylene glycol is toxic when ingested, but of work places constitutes no threat to workers unless it is heated.

2. Environmental factors. The toxicity of the substances are influenced by temperature, humidity and atmospheric pressure. High temperature violates the metabolism in the liver, increases lung ventilation, reduces imunnobiologichnata reactivity of the body, and combined with high humidity accelerates the absorption of toxic substances. In this connection, as a rule, high temperature and humidity enhance the toxic effect. Low temperatures in some cases enhance the effect of toxins (CO, benzene, acetone, dichloroethane), in others - reducing toxic effects due to inducing the activity of OMF* (naphthylamine, aniline). The increased atmospheric pressure also associated with an increased partial pressure of the substances promote the entry of more poison in the body (eg. nitrogen oxides). Under reduced pressure, however, is often also observed to potentiate the toxic effects - eg. hypoxic poisons (CO, benzene, benzine) as a result of reduced oxygen partial pressure.

3. Individual factors. Individual factors that influence the body's sensitivity to poisons are: gender, age, race, genetic background, body weight, diet, lifestyle, immunological and hormonal status, transitional diseases, stress. These factors are dependent on one another. For example, genetic factors determine a significant number of other individual factors, and diet can significantly affect the immunological status. Gender - women establish more sensitive to some poisons (lead, mercury, benzine, benzene, PhOC), particularly during pregnancy, lactation or menstruation; compounds of boron are selectively toxic action to the male gonads. A number of studies have shown that the fetus, children and the elderly have a greater sensitivity to a variety of poisons as a result of the lower efficiency of metabolic poisons. On susceptibility to toxins can also affect the length of service and intensity of physical labour.

Malnutrition usually alter the normal response of the body to toxins by influencing the absorption, storage and bio-transformation. Individual sensitivity also depends of health status, functional activity of different systems, the character of work - eg. heavy physical work enhances the processes of respiration and circulation and thus accelerates the entry of toxins in organs and tissues. Increased sensitivity to certain toxic substances - vicyanin, arsenic, naphthalene, described in subjects with deficiency of glucoso 6-phosphate-dehydrogenase, sickle cell anemia, alpha-antitrypsin deficiency.

* The OMF - mixed function oxidases

In the reactions of the organism in toxicity of poisons observed stages: Stage 1. The primary reaction; 2. Stage of "adaptation"; 3. Stage of compensation/ decompensation. The described adaptation of the organism to some poisons- chlorine, ammonia, formaldehyde, is often apparent and is connected to the intension of the biochemical and physiological responses, which leads to hidden damage to tissues and organs. These lesions can be detected sometimes after adequate functional load.

4. Physical properties of toxic substances. The toxicity of the substances exert certain influence their solubility, volatility and physical condition. As a substance is more soluble, it is more toxic. Particularly important to assess toxicity has solubility in water, biological fluids and fat. The effect of drugs depends on the distribution of their distributing coefficient (DC) = solubility in oil/solubility in water - rule of Overton and Meyer. As this ratio is larger, the penetrating ability and their accumulation in lipid-rich tissues is larger. Physical condition is also important for toxicity - metallic mercury as a liquid is non-toxic, but in the form of vapour is very dangerous. The same applies to a finely dispersed solids. In equally other conditions higher volatility - with a lower boiling point, determines most toxic of substances. For example parathion and sistoks have the same toxicity but sistoks is much more volatile and therefore is much more dangerous than parathion*. In this connection, in toxicological practice has been introduced the concept of effective toxicity. It is determined by the product of absolute toxicity and volatility and well-characterized manufacturing health risk. In case a particular appears introduced by I.V. Sanotskiy term - coefficient of possible inhalation poisoning (CPIP), which is determined by the ratio $C^{20^\circ}/ CL_{50}^{120}$, where C^{20° is the maximum real concentration at 20°C, and CL_{50}^{120} is the concentration causing 50% mortality of white mice in single exposure of 120 min.

5. Chemical composition and structure of substances. To a large extent the toxic action of the substances is determined by their structure and chemical composition. Thanks to that today can largely be predicted toxicity and pharmacological activity of newly synthesized chemical compounds, based on some known their physical and chemical properties and determine their tentative MAC in the air of the environment or the working environment.

It should'nt, however, be absolute opinion that knowing the chemical structure of a substance can quite accurately determine the nature and intensity of its effect. There are many examples which show that two completely different chemical structure substances can have similar toxic effect and vice versa. The reason for this is that very often the body responds with a non-specific, universal effects of the action of substances that are quite different in chemical structure. By practical standpoint importance have dependencies that allow to predict the change in the toxic action of the substances in a type of modification of the chemical structure. Most manifest the relationships between chemical structure and biological activity in homologous series.

Some of the established patterns between chemical structure and toxicity are: **1.** The rule of branched chain. This rule is fully valid as a homologous series of unspecified fatty hydrocarbons and the aromatic hydrocarbons - ex. propylic and butylic alcohol are stronger drugs from isopropylic and isobutylic; **2.** Substances with higher atomic weight are more toxic as compared to substances with less atomic weight; **3.** The toxicity of the organic compounds increases with increasing number of double bonds - eg. acetylene is more toxic than ethane and ethylene. Narcotic effect increases at acyclic hydrocarbons with an increase of double bonds (cyclohexane $\rightarrow$ cyclohexaene $\rightarrow$ cyclohexadiene). **4.** By increasing the number of carbon atoms in the homologous rows of methane is increased toxic effects of the compounds (rule of Richardson). For example, there are pentane CL50 - 300 mg/l, the hexane - 100 mg/l, hectan - 40 mg/l, and octane - 30mg/l; **5.** The spatial location of the radicals in the molecule also affects the strength and character of toxicity - paraisomer have the highest biological activity, follow by meta- and ortoisomers. This dependence is observed for example in the isomers of nitrophenol, nitrotoluene, nitrobenzene and nitroaniline; **6.** The molecular symmetry is also important for the toxicity of the substances. It is understood that the compounds which have a symmetrical structure of the molecule are more toxic than the compounds of asymmetrical construction; **7.** The organic compounds of low valency when combined with metalloids - sulfur, arsenic and phosphorus, increase their toxicity; **8.** The character of the action of the substances is altered by introduction of halogen atoms, methyl, amine, hydroxyl, and nitro groups. The introduction of chlorine, bromine and fluorine in a molecule increases the irritating and their general toxic action and the introduction of amino and nitro groups converted into methemoglobinferous compounds, increases their allergic action. **9.** The change in the valence of the elements may change their toxicity and the nature of the action. Trivalent arsenic compounds for example are more toxic than quinquivalent, and

* above applies to the toxicity of the substances entering the inspired air

in chroma, contrast, hexavalent are more toxic than trivalent (trivalent chromium compounds, however, have a carcinogenic effect); **10.** Dependence of toxicity by electronic stability. It was found that a reduction of the electronic stability increases the toxicity of inorganic compounds; **11.** Depending on the type of crystal lattice. Substances with a volume centered cubic lattice are the least toxic, and those with low grade symmetry of the lattice are less toxic. As examples can be mentioned titanium and tungsten, which have a weak pneumosclerosis effect and free silica (with hexagonal lattice) is a high pneumosclerosis effect.

7.1.2. TOXICOCINETICS

Toxicocinetics studied the movement of toxic substances in the body, ie their absorption, distribution, metabolism and excretion and the relationship between the dose which is received and the level of toxic substances in biological media.

Ways of penetration of toxins in the body. Poisons enter the body in three main ways: through the lungs, the skin and the gastrointestinal tract. By airway penetrate gases, vapors and dusts. By penetrating the skin mostly organic substances and through the gastrointestinal tract - emptied poison on your hands (in production gastrointestinal tract have the least importance for receipt of poisons).

Basic and the most dangerous way for receipt of poisons in the body is still **breathing**. The large surface of the lungs from 90 to 120 m^2, and their rich blood supply, allowing toxic substances to do quickly in the blood and quickly spread to all the organs and systems. It is estimated that 80-90% of acute poisoning in the industry are due to gases and vapors. Their entry into the body through the lungs is subject to certain mechanisms. Gases and vapors according to their absorption in the body is divided into reactants and non-reactings. **To the reactants** refer sulfur oxides, nitrogen oxides, ammonia, chlorine, phosgene, diphosgene, acids, bases. Their practical saturation of the blood with the poison does not occur, as is always the amount of the received gas is directly dependent on the volume of respiration (as a consequence, the risk of poisoning is much greater as greater exposure). Very well soluble and chemically reactive in water, gases and vapors such as ammonia, acids and bases, damaging mainly the upper respiratory tract and have irritation. Slowly soluble chlorine and sulfur gases totally damaged airways and slowly soluble - nitrogen oxides, phosgene, diphosgene, reach the lung alveoli and cause toxic bronchopneumonia and pulmonary edema.

To non-reacting gases and vapors includes all hydrocarbons from aromatic and fatty rank and their derivatives such as gasoline, benzene, ether, acetone, acetylene and others. They enter the blood through the lungs under the law of diffusion, ie due to the difference in partial pressure of gas at the alveolar air and blood. At the beginning of exposure saturation of blood is fast, then gradually slows down and eventually terminates (the partial pressure equalization). It is important to conduct in providing first aid to the victims, since their export on fresh air will quickly come desorbtion. The toxic effect of these toxins depends mainly on their solubility in the blood (water) and much less than their exposure. Their solubility in the blood is determined by the partition coefficient (K) of Henderson and Hagar.

K = substance concentration in arterial blood / substance concentration in the alveolar air.

For example, K ethanol is 2000, 12.9 of the ether, benzene - 10.3, benzine - 2.1, acetylene - 0.84. As smaller this ratio, the faster the poisoning occurs.

Dermal absorption of toxins is limited. Ability to pass through an intact or damaged skin have only lipoidsoluble substances such as organic solvents, some pesticides, chlorinated hydrocarbons (dichloroethane, trichlorethylene, carbon tetrachloride), amino- and nitro compounds (nitrobenzene, trinitrotoluene, TNT, dinitrobenzene) and others. The absorption increases with larger contact surface with the poison, in moist skin, substances that cause increased blood supply to the skin. It depends on the individual and racial characteristics of the skin and the skin area of the body (skin structure). For the action of toxic substances penetrating through the skin matter and their solubility in water, ie it depends on the partition coefficient (K) of the substance in the oil and water: K = solubility in the oil/ water solubility - (law of Overton and Meyer)*.

Some electrolytes such as lead and mercury in small quantities can penetrate through the follicles of the skin. Major importance for skin penetration are the consistency and volatility of the substances. For example substances of low volatility, and an oil-like consistency, such as aniline, nitrobenzene, or readily volatile, but incorporated in oily creams and pastes, easily penetrate the skin and be retained longer on it.

In ophthalmic absorption toxic substances within the body, through the conjunctiva, in which bypass the liver and can cause severe intoxication (PhOC).

The presence of toxins in the body through the

* For substances with low K, ex. gasoline - 2.1, poisoning through the skin does not occur, due to its rapid excretion via the lungs.

digestive tract is possible for non-rules of personal hygiene, such as nutrition and smoking with contaminated hands. Some poisons can be absorbed already in the mouth, for example lipophilic compounds, such as cyanide, nicotine, phenols and the like. A number of substances undergo chemical transformations in the stomach - for example iron passes from the two-to-trivalent, insoluble salts of lead in more soluble, and other poisons under the action of acid medium pass into more toxic compounds. In certain specific conditions in the digestive tract (elevated pH values, the presence of nitrites more) low or medium-toxic amines, and dithiocarbamates may be nitrosated to carcinogenic nitrosamines.

Distribution of poisons in the body. The toxic substances are transported to the various parts of the body predominantly from the blood (some of them can be transformed by the lymph, and other by macrophages). The distribution depends on the physicochemical properties but affect cardiac output and regional especially blood flow. In tissue distribution and penetration into the cells toxins are divided into two groups: non-electrolytes and electrolytes. **Non-electrolytes** are distributed mainly in the organs with good circulation. As the partition coefficient of the substances in the oil and water is greater (law of Overton and Meyer), so their penetration capability through lipoproteinic cell membranes is greater. **Electrolytes** penetrate into the cells, depending on the charge of their membranes. If the surface of the cells is negatively charged, they do not miss anions and a positive charge not miss cations. Therefore, distributions of electrolytes in the tissues is very unequable. As a feature considered their ability to accumulate in some tissues and organs and form deposits - material accumulation. This creates an opportunity for the introduction of small amounts of a poison in the body occur chronic intoxication (eg. entrance of 0.2 to 1mg of lead per day at about six months exposure leads to chronic poisoning). Lead in larger amounts accumulate in bones, manganese - in the liver and heart, mercury - kidney, cadmium - in the kidneys and liver, arsenic - in nails and hair, etc. Material accumulation is assessed by factor of accumulation (Kk).

Kk = DL_{50chr} (for 3 months)/DL_{50ac} (single exposure).

When Kk less than 1 there is overcumulation, at odds of 1-3 - a high accumulation of 3-7 - medium and 7-10 - weak and over 10 - very weak.

Besides the material accumulation is known and functional cumulation, which showed accumulation of physiological effects, ie if the concentration of the toxic substance is below the threshold, changes in the body do not occur, regardless of the duration of exposure. A typical example in this respect are organophosphorus pesticides (PhOC) who gradually penetrates into the body inhibits cholinesterase activity (CheA). The clinical manifestations of toxic action of PhOC are registered in inhibiting CheA more than 50%. Lowering CheA 30-50% is seen as premorbid condition.

Metabolism of toxins in the body. Only a minor part of become trapped in the body substances is removed unchanged (inert gases, acids and alkalis, some aliphatic hydrocarbons).

Enzymatic reactions performed in metabolic biotransformation of xenobiotics can be divided into four basic types: oxidation, reduction, hydrolysis and conjugation (conjugation is always final reaction).

The reduction and hydrolysis usually prepare toxics substances make harmless, while oxidation reactions are the most common. The oxidation in the body is subjected to alcohols, aldehydes, acids. Benzene, for example is oxidized to phenol and pyrocatechol, alcohols of the fatty rank to water and CO_2 (with the exception of the methanol, which is oxidised to formaldehyde and formic acid). The salts of the metals typically form insoluble complex compounds with tissue albumin. In the liver is done conjugation of toxins with glucose, cysteine, methionine, sulfuric acid - sulfates, glutathione. As a result of the biotransformation of toxins in the body to form less toxic and less soluble compounds that are readily excreted by the kidneys. Known are, however, many cases where products of metabolic biotransformation are more toxic than the parent compound, for example tetrachloromethane metabolized to more toxic radical trichloromethane, methyl acetate hydrolyzed to methyl alcohol and acetic acid and methyl alcohol to high toxical formaldehyde and formic acid. In the oxidative desulfuration of tyophosphates (PhOC) is obtained P = 0 analogs which are also hundreds of times more toxic. The biotransformation of xenobiotics in the body (reduction, hydrolysis and oxidation) is carried out in most cases involving hydrolyzing enzyme system, known as a mixed function oxidases (OMF), mainly in the liver and to a lesser degree in blood plasma, kidneys, skin, lung or other tissues. In OMF includes about 25 enzymes (cytochromes, esterase, reductase), at which leading importance for cytochrome - P-450. Biotransformation reactions without involving OMF are distinguished by pronounced substrate specificity - eg. histaminase only participate in the metabolism of histamine, serotoninase - serotonin, monoamine oxidase - primary amines and catecholamines, alcohol dehydrogenase - methanol and ethanol, etc.

Excretion of toxins from the body. The excretion of toxins from the body is carried out by the kidneys, lungs, gastrointestinal tract and skin. Toxic substances excreted as metabolites or unchanged. Excretory organs except the lungs eliminate more efficient water-soluble compounds. Kidneys are the most important organs that eliminate most polar compounds and their metabolites. The excretion of toxic substances in the urine going through glomerular filtration, active secretion and passive tubular reabsorption. Alkalization or acidified urine can significantly alter excretion of some toxic agents - eg. when the primary urine is alkaline, weak acids are excreted faster because they are ionized and passive tubular reabsorption is reduced. Many toxic substances metabolised in the liver is excreted in the bile and then excreted in feces or reabsorbed into the blood and are eliminated through the urine.

Trans-lung eliminate inert gases and vapors. The rate of release depends on the distribution coefficient oil/water. As this ratio is smaller, so the poison is removed faster at organism. For example, much faster are separated gasoline, benzene and chloroform and much slower ethanol and acetone.

In the stomach and intestines were removed insoluble metabolites, metals, bipolar ions which can not cross the epithelial barrier.

In the skin and mucous membranes may be eliminated mercury, cyanides, arsenic, chromium, serodovorod etc. . Some toxic substances in small amounts can be separated, and in breast milk, saliva, sweat glands (lead, mercury, manganese, etc.).

7.1.3. TOXICOMETRICS

Toxicometrics studied quantification of potential and actual risk from the effects of toxic substances. Toxicometrics parameters include: lethal, thresholds and limit doses and concentrations coefficients digitally define specific damage in certain path of administration and level of impact:

1. Zero lethal dose or concentration $DL_0/CL_0/$ - represents the maximum tolerated dose or concentration, that does not cause death in any of the test animals.

2. Minimum lethal dose or concentration $DL_{min}/CL_{min}/$ - lowest dose or concentration that causes death of at least one of the treated animals.

3. Medium lethal dose or concentration $DL_{50}/CL_{50}/$ - is that dose or concentration that causes death of at least 50% of the tested animals.

4. Absolute lethal dose or concentration $DL_{100}/CL_{100}/$. This is the minimum dose or concentration that causes death of all treated animals. They are set using a statistical treatment of results, the most common method of J. Litchefield and F. Wilcocxon - defined doses at low and high mortality - DL_{l6}, DL_{50}, DL_{84}.

5. Threshold of acute effect in a single treatment of experimental animals - Lim_{ac} - represents the minimum dose or concentration causing in acute effects changes in some (most sensitive to specific conditions) of those of surveyed indicators for body vital activity (integrated, immunological, biochemical, historadiographycal etc).

6. Threshold of chronic effect (Lim_{ch}) - represents the minimum dose or concentration that causes chronic effects change of surveyed indicators. Based on the threshold of chronic effect of substances in inhalation exposure is determined experimental admissible limit concentration (MAC).

7. Ratio of the stock (RS) - the ratio of the threshold of chronic effect to the relevant MAC.

8. Factor of accumulation (FA) - the ratio between total DL_{50}, determined in one month oral experience with the daily introduction of 1/5 - 1/20 and less of DL_{50} single (defined in single time experience) - to DL_{50} single. The more big FA, so the risk of accumulation and toxic effects is smaller (back in FA <1 - "over accumulation").

9. Zone of acute action $Z_{ac} = CL_{50}/Lim_{ac}$ - is the ratio of median lethal concentration to the threshold of acute action.

10. Zone of chronic action $Z_{ch} = Lim_{ac}/Lim_{ch}$ - is the ratio of the threshold of acute action on the threshold of chronic action.

The determination of these areas is important for occupational risk. As the area of the acute effect is smaller and the area of chronic effect is greater, the toxicant is more dangerous, respectively for deaths in acute impact and intoxications - in chronic.

The maximum admissible concentration (MAC) represents the maximum amount of a substance in the air of the working environment, which in everyday work, but also throughout total service does not cause among workers, except in cases of hypersensitivity, somatic or mental illness or changes in their health state out of the limits of adaptive physiological reactions, identified with the modern methods of investigation, immediately or in remote periods of life, on present or future generations.

In determining the MAC starting from:

1. Physico-chemical properties of substances - they justify tentative MAC, valid for two years or to carry out the necessary toxicometrics experimental studies of toxic agent.

2. The results of experimental studies - they justify the temporary MAC.

3. Data of hygiene observations of conditions in the manufacturing and health of workers for a peri-

od of three years after the introduction of the substance into production are use if necessary for correction of the temporary MAC.

In justifying tentative MAC account the structural formula of the substances, molecular and specific weight, melting and boiling point, volatility, solubility in different media.

Output value for determining temporary MAC is a threshold of a chronic effect or dose-effect. Most often MAC is set at 10-100 times lower than the threshold of chronic action - i.e. ratio of the stock (RS).

The stock factor increases with increase in the absolute toxicity, with increasing volatility, by reducing the area of the acute effect, by increasing the area of chronic action, to reduce the rate of accumulation, with an increase in skin-resorption action, with the presence of teratogenic, embryotoxic , gonadotrophic, mutagenic or carcinogenic effects - in recent cases, RS can reach 1000 or xenobiotic no permit to the production (eg. asbestos).

In experimental studies in recent years, is selected exposure level based on the critical effects which is **the highest level investigated, whereby it is not established "any adverse effect." No effect** (level of no observed adverse effects - LNOAE) is the highest experimental dose of a chemical substance in which there are no statistically proven and biologically significant alterations in the frequency and severity of adverse effects among individuals in the group subjected to exposure, as compared with a suitable control group. A toxic agent can cause more than one toxic effect. Used indicator for the critical effect in the evaluation of toxicity dose/response is deviation showing the lowest value in determining the no active dose.

MAD is the standard dose, derived from no effect dose by the application of magnitude of the factors of uncertainty (FU), which reflect the different types of data sets, used to determine the limit levels. Sometimes used and modifying factor (MF), which is based on the professional judgment of the entire database relating to the toxic chemical **(Table. 1)**.

MAD = no effect dose/uncertainty factor x modifying factor

Basically, MAD is level (reference dose - RfD) with uncertainties, lying within one order of magnitude of daily exposure of the population (including and sensitive groups), causing a noticeable risk of disability throughout life.

The reference dose is expressed in mg/kg per day.

Determining the MAC of inhaled pollutants is more complex than determining the MAD - in oral exposure. In determining the MAC should keep in mind the differences in anatomy and physiology of itself respiratory system in animals and humans, and the physicochemical properties of the chemical. Furthermore, the critical health effects for the respiratory system depends more on the kind of chemical deposition in it, than the concentration of the substance in the air.

Doses or exposures lower than the MAC and MAD most often not associated with significant health risks which are not taken into account in the standardization, but the last (ie compliance with regulations) does not mean the absence of a health risk.

According to the duration of the impact of toxic substances, under Regulation N 13 (SG. 81/1992) the limit concentrations are divided into:

1. Mean-shift MAC (MSMAC) - this is the average concentrations of harmful substances 8.5 h work time of no more than 40 h work week;

2. Short-time maximum MAC (STMMAC) - this is the highest allowable concentration for up to 15 min., no more than 4 times per work shift, at intervals over 60 min. (for substances without specific STMMAC allow one-off increase over 3 times MSMAC for 30 min.);

3. Exposure MAC (EMAC) is used to work time over 8,5 h. Determined by the formula:

EMAC = (MSMAC x 8)/h. (h. - have extended hours of working day);

Table 1. Factors of uncertainty and modifying factor

Uncertainties factor (FU)	
Question for discussion	**Value**
1. Take into account the biological variability of the whole population in order to protect sensitive groups	10
2. When extrapolating from animals to humans	10
3. When the no-effect dose was established as a result of subchronic instead of chronic studies in determining chronic MAD	10
4. When used threshold of chronic action instead-no effect dose (LNOAE)	10
Modifying factor (MF)	
1. To take into account other deficiencies, which are not included in previous factor of uncertainties (mutagenic, carcinogenic, etc.).	< 0 до 10

4. Biological MAC (BMAC) - the maximum content of harmful chemicals or their metabolites in blood, urine or breath, which does not expect any damage to the health of workers and their offspring (BMAC is assigned to the value of the MSMAC). BMAC gives important information about occupational risk for the introduction of xenobiotic more than one of the three physiological path way;

5. Limit concentrations of chemical irritants, contact allergens and substances with skin-resorbtive systemic action in stocks and raw materials that have direct contact with people;

6. Concentration immediately dangerous to health (CIDH) - the maximum concentration of the toxic substances that no-threaten the health and life in case of emergency, in effect no more than 30 min. (CIDH determines the need for appropriate use of masks).

An ordinance № 13/2003 in place to limit concentrations (MAC) for the protection of workers from the risks related to exposure to chemical agents at work, introducing concepts:

- **limit of chemical** agents in the air at the workplace (in mg/m^3 or number of particles/cm^3) exposure 8 h or 15 min;
- **biological limits** of the chemical agents and/or their metabolites in biological media, as well as biomarkers of effect.

The values for various chemical agents under this ordinance do not differ from those indicated in Decree № 13/1992 years - respectively mean-shift MAC (about 8,5 h and 40 h the work-week); MAC short-time maximum (to 15 min); biological MAC (BMAC).

The values of chemical agents in the air at the workplace and in biological media (them or their metabolites) should not exceed the limits.

The presence of hazardous chemical agents in the working environment requires the employer to be informed of their existence and take various adequate measures to reduce the health risk to workers. These include technological and technical changes to reduce the amount of their, replacement with other less toxic or the removal thereof; awareness of workers about risk, training for the safeguards, their health status; health surveillance of occupational health services, etc. The main responsibility for this is the employer, of course, and persons (including workers), the relevant authorities and agencies involved in reducing health risks among workers exposed to chemical agents.

This approach to hygiene regulation of chemical agents at work - ie. *occupational exposure standards for airborne contaminants*, developed in the United States and is accepted in European and other countries. Developed in the US called. **TLVs** (Threshold Limit Values) by **ACGIH** (American Conference of Governmental Industrial Hygienists); **RELs** (Recommended Exposure Levels) from **NIOSH** (National Institute for Occupational Safety and Health) and **PELs** (Permissible Exposure Limits) from **OSHA** (Occupational Safety and Health Administration), which are officially authorized, recommended limits for various chemicals in the air of working environment and in biological media and accepted in many countries, incl. in Bulgaria with/or without some adjustments to specific chemicals.

The essence of this approach is that there is no environment without health risk but *"safe"* is the environment where the level of risk is acceptable under specific conditions, ie applied the principle **ALARA** (As Low As Reasonably Achievable).

In the production Toxicology to measure exposure through biological monitoring more often used **biological markers** that are three types: markers of exposure, effect markers and markers of sensitivity.

Markers of exposure are exogenous substances, their metabolites, interaction between xenobiotic agents and target molecules or cells, tested in any part of the body. For example, the concentration of lead in blood reflects the exposure as a result of inhalation of lead in the air, and as a result of the direct or indirect absorption through food, water, dust or soil. Also, there is a linear relationship between exposure to CO and carboxyhemoglobin levels in the blood. It is assumed that usually the marker of exposure is more useful for appraiser if in biological material is measured the substance, not its metabolite.

Markers of efficiency are measurable biochemical, physiological or other change in the body that depending on its magnitude is recognized as an established or potential deterioration of health. Markers of effect can be useful for assessing exposure, if signal for preclinical or presymptomatic stages in the development of the disease (eg. inhibiting the ChEA of PhOC) that are specific to the substance. Other markers of effect can report only adaptive changes that by themselves are not pathological.

Markers for sensitivity are indicators of inherent or acquired limiting the body's ability to appropriate response to exposure to systemic toxic substance. For example, the measurement of the reactivity of the airways to inhaled bronchoconstrictors can be used as a marker for susceptibility - the increased non-specific reactivity of the airways is a characteristic of most asthmatics and plays a role in the development of the disease.

In recent years there has been great progress in developing a new class of biomarkers, namely measuring the labeled DNA molecules and proteins. These new techniques are only possible under

these chemicals or their metabolites which can bind covalently with biological molecules (eg. DNA and proteins) - i.e. labeled molecules into target or peripheral tissues. For detection and quantification of labeled molecules using methods of gas and liquid chromatography/mass spectrometry, as well as immunological assays with labeled with radioactive or fluorescent molecules antigens.

7.1.4. TOXICODYNAMICS (MECHANISMS OF TOXICITY)

Toxicodynamics study the impact of toxic substances on the body, ie studies the relationship between the received dose and the measured biological response, the latter is linked to the concentration of toxic substances at the place of action eg. where the toxic agent received through the mouth causes vomiting and diarrhea, it is rapidly eliminated and does not reach the existing level so-called toxic bioavailability.

In recent years, when literature deals with the mechanism of action of poisons, toxic effects are discussed at various levels - from primary effect at the molecular level to the highest levels - cells, tissues, systems, organism.

All these events constitute **toxicodynamics**, which in turn determines the clinical picture of intoxication.

In critical cell concentration occurring biologically significant adverse functional changes in the cell due to damage at sensitive to toxic substances structures (usually these are organelles in which the receptor is a structural element). Critical cells and is the first population of cells in which poisonous substance reaches a critical concentration (due to readaptation changes next populations are less sensitive).Critical tissue concentration is the average concentration reached in the organ (tissue) for the period during which in the corresponding cells is a critical concentration. Critical organ is the first organum in which the poison reaches toxic concentration under specific conditions of exposure. His knowledge is important for determining the threshold of chronic action, hygienic standardization and early diagnosis of occupational diseases.

Selective impairment of the critical organ is also used for the characterization of toxins such as: hepatotropic (carbon tetrachloride, halothane, ChlOC, etc.), neurotropic (organic solvents, certain compounds of bromine), damaging hematopoiesis (benzene, aniline, etc.), Nephrotropic (compounds mercury, cadmium). In all these cases range between doses of the substance damaging the critical organ and other organs and body systems is significant (the range is much smaller in poisons that haven't distinct effects). Exposure leading to subcritical concentrations in critical organ can cause biochemical changes (subcritical effects) associated with a compensatory intension. They are a manifestation of premorbid condition of the body and serve as a harbinger of danger of causing critical effects - that defines great importance to the prevention of occupational diseases.

The critical effect is a biological change caused by certain exposure. It is connected to the primary toxic effect, as may coincide with it, or be the result of its development eg. primary toxic effect of PhOC is the inactivation of ChEA. When the degree of inactivation reach 50% there is a critical effect.

The critical effect directed to investigate the most sensitive to the toxic effects function of critical organ - eg. critical effect for occupational exposure to cadmium is proteinuria - the result of the development of the primary toxic effects in cells critical to the critical organ (the epithelial cells of the proximal renal tubules).

To adopted today molecular mechanisms, specific to the operation of the whole groups of toxic substances include: a) forming in the body of oxidants - free radicals by lipoperoxidation (carbon tetrachloride, iodoform, dibromomethane, dimetilaminoazobenzol etc.), b) coupling with the sulfhydryl groups of enzymes (i.e. thiol poisons - metals - mercury, lead, cadmium and their compounds, arsenic, styrene, benzene, dithiocarbamates, chloroprene, etc.), c) changes in the enzyme activity (heavy metals, PhOC, carbamates, and ChlOC etc.). These changes are divided by the authors of the primary and secondary. The primary changes are mostly linked with structural modifications in the molecule of the enzyme, leading to partial or complete loss of function. Secondary changes may be manifested by an increase or decrease in the activity of enzymes, d) a biologically alkylation and decupling of oxidative phosphorylation (dinitrophenols, arsenic, aluminum salts, dieldrin, etc.), e) changes in the regulation of biochemical processes in the intoxication (ethanol, nitroglycerin, etilenglikoldinitrat etc.). Changes in the regulatory mechanisms here are associated most often with a change in the content of biogenic amines - adrenaline, noradrenaline, serotonin and others.

Received in the body manufacturing poisons cause **acute, subacute and chronic poisoning**. Acute poisonings often occur in groups and in random accidents. They are characterized by the following important features: a) short-term effect of the poison; b) the poison enters the body in relatively large amounts, often at high concentrations in the

air of the working environment, after mistakenly ingestion or heavily contaminated skin and mucous membranes; c) a clear clinical picture immediately after receipt of the poison in the body or after a short-lived latent period. As a rule, in development of acute poisoning has two phases: the first phase is nonspecific and is manifested by headaches, general weakness, dizziness; the second phase - specific, which showed symptoms characteristic of the poison.

Chronic poisonings occur after long duration exposure and at significantly lower concentrations of toxic substances, particularly those with cumulative properties.

Poisons occur **mainly general or mainly local action**. In local reactions (in the place of receipt of the poison) always establish common events - leukocytosis and fever at chemical burns, histamine - like reactions, etc. Poisons have mostly general resorption action that occurs after the release of substances in the blood and tissue fluids. This is due to the disruption of cell and tissue metabolism resulting impact of toxins on enzyme systems.

Production poisons may cause not only a specific acute, subacute and chronic poisoning, but also reduce imunnobiological reactivity of the organism, may contribute to the development of non-specific diseases - catarrh of the upper respiratory tract, tuberculosis, diseases of the kidneys, cardiovascular system and so on. A number of industrial poisons cause allergic diseases, have gonadotropic, teratogenic, mutagenic and carcinogenic effects.

Allergens belong to various chemicals - inorganic (nickel, cobalt, mercury, arsenic, chromium, beryllium); organic compounds (heterocyclic turpentine, aldehydes, aromatic nitro- and amino- compounds. Very strong allergens are diamines as paraphenyl-diamine (yrsol). Other chemicals have the ability to sensitize the skin to UV radiation, so-called photosensitizers - anthracene, coal tar.

Gonadotropic effect relates by changes in genital tissue and gonads, with a change in their ability to reproduce. Gonadotropic activity have a number of xenobiotics such as carbon monoxide, sulfur oxides, nitrogen oxides, lead, mercury, benzene, nicotine, acrolein, tetrachloroethane.

In **embryotoxic and teratogenic effect** observed damage to the fetus in the uterus after transfer of the placental barrier to chemical substances. This effect was observed in the exposure during the first trimester, the most common in: lead, mercury, manganese, derivatives of carbamic and dithiocarbamic acid, phenol, formaldehyde, benzene, dinitrobenzene, chlorinated hydrocarbons, a number of drugs, ethanol and the like. Embryotoxic effect in which the observed death of the cells of the fetus occurs in exposure to higher doses or concentrations of chemical substances, or when they enter the body through the first 15-20 days of fertilization. The teratogenic effect was found in exposure of pregnant women at low concentrations and after 15 days from the beginning of fertilization.

Mutagenic effect associated with the occurrence of changes in the hereditary information that can be transmitted to future generations. Mutagenic activity have several hundred inorganic and organic compounds - organochlorine pesticides (DDT, hexachloro, dieldrin, aldrin), organic mercury compounds, derivatives of dithiocarbamic acid, chlorinated hydrocarbons, polycyclic aromatic hydrocarbons, nitroso compounds and others.

The carcinogenic effect is associated with two major mechanisms of action: genotoxic and epitoxic. It is estimated that 80-85% of malignant tumors are caused by exogenous chemical substances: 1. Polycyclic aromatic hydrocarbons - more than 100 substances of a basic agent 3,4-benzpyrene, which are contained in petroleum, coal tar, soot, smoke ; 2. Aromatic amines - naphthyl amine, benzidine, amino derivatives of stilbene; 3. Azo- and nitro compounds; 4-metilaminoazobenzol, nitrozodialkilamin, nitrosamines; 4. Other carcinogens - beryllium, nickel, chromium, arsenic, epoxy resins, radioactive substances, vinyl chloride, acrolein, mycotoxins, saccharin, hydrazines, safrole, benzene and the like.

In production conditions very often found **combined effect** of certain chemicals on the body. In metallurgy frequent combinations CO and content of sulfur dioxide, the CO and NOx, in the chemical industry - vapour from benzene with toluene and hydrogen sulphide, in agriculture - combinations between PhOC and dithiocarbamates, PhOC and ChlOC, PhOC and nitrophenols or synthetic pyrethroids . There are three main types of combined action: a) **synergy** - when one substance potentiates the action of the other; b) **antagonism** - where a substance attenuates the other; c) **total or additive effect** (mostly) - where the effect of the substance added. The mechanism of action in the potentiation or reduction of toxic effects is associated with the ability of certain substances to inhibit or induce the enzyme systems involved in metabolic biotransformation. For example, as inducers OMF appear some of ChlOC's - lindane, melbex. Their combination with PhOC reduces the toxic effect - antagonism.

For hygienic evaluation of air environment under conditions of additive effect of toxic gases (eg. sulfur oxides and nitrogen oxides) apply the formula of Averyanov.

$$K = \frac{C_1}{MAC_1} + \frac{C_2}{MAC_2} + \frac{C_3}{MAC_3} + \frac{C_n}{MAC_n} \leq 1$$

where the sum of the relationship of the actual concentrations to their MAC must be equal to or less than one.

7.1.5. PREVENTION OF OCCUPATIONAL POISONINGS. GENERAL PRINCIPLES.

Most effective measures for the prevention of occupational poisoning can take place in the design phase of industrial enterprises.

To hygiene failures associated with acute and chronic poisoning of workers in manufacturing most often include: disruption of the normal technological regime, failure of equipment, ventilation, tools and apparatuses, violating occupational safety and hygiene, poor instruction and insufficient training of workers coming into contact with toxic substances, improper work organization and workplace, absence or malfunction of personal protective equipment and special clothing. In this connection, the main measures for the prevention of occupational poisoning can be grouped as follows:

1. Introduction of new technology and preserving optimal technological regime. Here importance is the replacement of more toxic substances with less toxic (eg, the use of gasoline instead of benzene, of zinc paints instead of lead) and hygiene standards of the most important chemical raw materials in order to prevent highly toxic substances in basic composition of chemical products (plastics, paints). Violation of normal technological regime not only leads to failure of machinery, but serious accidents causing acute group poisonings.

2. Provide complete seal hermetically of equipment and communications.

3. Provide sufficient air exchange in manufacturing premisses. This is achieved with natural and artificial ventilation as the most dangerous places separate high concentrations of gases must be installed local hoods or aspirators. In the workshops, which emit heavier than air gases and vapors it is necessary to provide additional aspiration of air into the lower part of the premises.

4. Continuous mechanization and automation of production, including remote control and regulation of processes, accurate and reliable operation of CMG (check-measuring gauges) and automation. On today much of the main aggregates, like electro-filters, steam-utilitarisators, cameras for combustion, columns of synthesis and precatalysis are out in the open, away from production areas and employees control their work remotely, i.e. from a control room through appropriate instrumentation. More dangerous places need setting up of alarm with siren or warning lights, related to the control room.

5. Regular monitoring of the content of poisons - gas and dust in the air of production premises. This can provide by using gas analysers and detectors for automatic control.

6. Organization of effective measures for personal prevention. This includes providing employees: reliable personal protective equipment and special clothing, protective professional nutrition, resources for maintaining personal hygiene - bathrooms, wardrobes, laundry for cleaning and maintenance of clothing, rooms for personal hygiene of women. In chemical plants, metallurgy and mining sites is mandatory setting up of gaz-saving stations.

7. Organization of effective therapeutic and prophylactic measures. These include mandatory conducting preliminary professional selection of incoming work in harmful and dangerous industries, conducting periodic preventive examinations and dispensary of diseased, registration and analysis of occupational diseases and poisoning. The registration of professional poisonings - acute and chronic, inquiry of each case and the analysis of their causes are mandatory.

8. Organizational and administrative measures. They are regulated by labour legislation and include: shortened working day, additional leave, reduced total length of service, preliminary and ongoing sanitary control, technical and labour hygiene instruction for newcomer workers. Instruction includes introduction with harmful production factors and prevention measures, proper use of personal protective equipment, methods of self-help and mutual assistance in the poisoning, health-education measures.

In all industries where possible poisoning, requires extreme readiness of medical personnel and adequate basis for fast rendering first aid to the victims of acute poisoning.

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7.2 METALS AND NON-METALS

Antimony (Sb)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 0,5 mg/m^3

GENERAL CHARACTERISTICS

Antimony is a silvery-white metal with a pronounced sparkle and crystalline structure. Atomic weight - 121.75; density 6,69 g/ml at 20°C, a melting point of 630,5°C, boiling temperature - according to different authors vary from 1325 to 1635°C.

Chemical is poorly active element. Low migrates into the environment. Diffuse dispersed metal with an average content in the lithosphere 4-5.10^{-5}%. In native condition is very rare. There are over 90 mineral containing antimony: complex sulphide, oxides and oxichlorides. Manufactured has mineral antimonyt (Sb_2S_3 - antimony 70%, sulfur 28%). In ores as a rule except antimony containing gold, silver, lead, copper and arsenic. The average content of antimony ranges from 5 to 20%. The largest deposits of antimony in the world are located on the territory of the former USSR. His most famous organic compound is antimony potassium tartrate (Antimony) that causes vomiting. It is used as a medicament. The majority described antimony poisoning are caused precisely by him.

In natural conditions, the largest quantities of antimony are found in the waters of the mineral springs (up to 0,5 mg/dm^3) and at sea.

USE

World annual production of antimony is about 100 thousand tons. In pure antimony is not used except for the semiconductor ultra-high purity (impurities no more than 1.10^{-4}%). Antimony alloys are very hard, corrosion-resistant, excellent friction properties and the ability to expand during hardening. Used for the production of bearings, plates for batteries, parts for pumps, equipment for the chemical industry, in typography.

Antimony trioxide is used in the chemical industry, textile and glass industry, the manufacture of paints and varnishes. Antimony pentoxide is used in the chemical and pharmaceutical industry. The compounds of antimony with sulfur is used to produce rubber (coloring agents and plasticizers), matches, in pyrotechnics. Halogenated antimony are used for the preparation of rubber, synthetic fiber and drugs. A number of preparations containing antimony is used for the treatment of parasitic diseases distributed in tropical countries.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Occupational exposure is mainly related activities upon receipt of antimony compounds and pure metal.

Markedly toxic to humans is antimony trichloride. The nature of toxicity is determined by the forming in hydrolysis hydrochloric acid.

High toxicity features is SbH_3 (stibine) - gaseous compound. There are reports of poisoning workers with stibine. It is experimentally proved that stibine induce acute inflammatory changes in the trachea and bronchi, pulmonary edema, necrobiotic changes in the myocardium, toxic dystrophy and necrosis of the renal tubules and others.

Unprofessional exposure concerns individuals using antimony preparations for treatment.

The presence of antimony in ambient air is associated with anthropogenic pollution and settled in areas of extracting antimony and lead companies. With waste gases from production in the atmosphere is ejected dust with a complex composition (antimony, arsenic, iron, lead, carbon, silicon dioxide) and spread over large distances (up to 20 km from the source).

The main sources of pollution of the aquatic environment with antimony are wastewater from concentration plants containing more than 10 mg/dm^3 antimony.

METABOLISM AND MECHANISM OF ACTION

Antimony is a nonessential toxic ultramicroelement to humans. For pentavalent its compounds supposedly essential role.

Enters the body by inhalation (for workers) and oral (mainly food). In the gastrointestinal tract are absorbed only 5-15% of the amount absorbed. For about two hours, the antimony passed from the blood into tissues. In a study of the distribution of the antimony in the body it has been found that antimony is rapidly detected in the liver, kidney, lung, thyroid, adrenal, pancreas. Regardless of the path of entry into the body, the nature of the distribution of the antimony is uniform, differences were observed only in the level. This pattern was also observed in complex receipt of antimony in the body (in all pos-

sible ways of simultaneously penetration). Pentavalent antimony is deposited in bones. It is assumed that in the body antimony subject of biomethylation.

Excreted primarily in the urine and faeces, but in the stools amount of antimony is 2-3 times more than in urine.

Exposure assessment recommended determination of antimony in urine, blood and hair.

Individual medicinal preparations (organic compounds) have a pronounced antigenic and cause skin reactions.

PREVENTION

Specific prevention of chronic poisoning with antimony in production include: technological and technical measures to limit dust- and gas emission in the production, isolation and hermetisation; preliminary and periodical medical examinations involving therapist, neurologist, radiologist and dermatologist; dispensary of persons in contact with antimony; testing for the presence of antimony in the blood, urine and other biological media; personal protective equipment (clothing, dust masks or respirators) and good personal hygiene.

ARSENIC (As)

HYGIENE STANDARDS

Air of the working environment.* Arsenic and inorganic compounds (diarsenic trioxide, diarsenic pentoxide, gallium arsenide) - Mean-shift MAC - 0,05 mg/m^3. Carcinogenicity to humans (Group 1) teratogen for animals.

Arsine (AsH_3) - Mean-shift MAC - 0,05 mg/m^3.

Air settlements: arsenic (inorganic compounds without arsine calculated as arsenic) - Daily average MAC - 0,003 mg/m^3.

Arsine - Daily average MAC - 0,002 mg/m^3.

Drinking water (BS 2823-83): Arsenic 3^+ in mg/dm^3 no more than 0,05 mg/dm^3.

Flowing surface water: I category - 0,02 mg/dm^3, II category - 0,05 mg/dm^3, III category - 0,2 mg/dm^3.

Food products: from 0,05 mg/kg in infant food milk-based, to 5,0 mg/kg in the sea fish.

Soil: 25 mg/kg.

GENERAL CHARACTERISTICS

Arsenic is widely distributed in nature element, though rarely occurs in native form. The average content in the earth's crust is 1,7.10^{-4}%. Opened as an impurity (average 2%) in all the sulphide ores in complex iron and manganese, precious and non-ferrous metals: copper, zinc, lead, gold and silver of amounts to 5-10% (in the richer of arsenic ores). Under natural conditions formed arsenates (As 5^+ - salts of arsenic acid), arsenite (As 3^+ - salts of arsenous acid). Alloys of arsenic with metals called arsenide.

Arsenic is a multivalent element, formed over 180 minerals. Manufactured are: arsenopyrite (FeAsS), auripigment (As_2S_3), with an average arsenic content 46% and 61%. Vigorously migrates into the environment. When volcanic activity in the composition of volcanic gases off into the air. With deep thermal water is exported to the surface simultaneously with iron, sulfur, selenium, antimony, cobalt, nickel, copper and others. and precipitated.

There are several allotropic forms of arsenic. The most common form are grey crystals with metallic luster that are oxidized by air. The density of the arsenic is 5.727 (at 14°C), melting temperature of 817°C to 615°C sublimate. Insoluble in water is. When heating is oxidized to As_2O_3. Aqua regia and nitric acid oxidated arsenic to arsenates.

Arsine (AsH_3) is a colorless gas. In its pure form is odorless, in the presence of impurities has a characteristic smell of garlic that probably due to dietilarsine - AsH $(C_2H_5)_2$. The density of arsine is 3.50, dissolved in water. When heated above 500°C is decomposed into hydrogen and arsenic.

White arsenic (As_2O_3) is an amorphous, glassy or crystalline solid, with melting point 313 ° C (crystal form), a boiling point of 456 ° C, density 4,15. Slightly soluble in water, the aqueous solution has an acid reaction.

USE

Arsenic is used to obtain a semiconductor (gallium arsenite), as an additive in the alloys, in glass industry, leather industry, the chemical industry (initiator, catalyst), in paints for underwater installations, in pyrotechnics, for the preservation of wood, in dentistry and medicine (currently limited).

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Arsene form and paid in the following cases:

- In contact with metals contaminated with arsenic acids, in electroplating and metal extraction by electrolysis;
- Contact FeSi with water (in warehouses for storage); metal containing arsenic with water in the metallurgical industry; in the preparation of acetylene by calcium carbide; at contact of aluminum arsenite with water if aluminum is used for cleansing lead from arsenic;
- In the chemical industry in the manufacture of aniline;
- In wet rooms painted with arsenic paint under the influence of developing fungus.

* Here and further for work environment, according to Decree № 13/2004 on be used instead mean-shift MAC, short-time maximum MAC, biological MAC, respectively - **limit for 8 h, limit for 15 min., a biological limit**. Specific values are the same - for MAC/for limit.

Conditions of increased absorption of arsenic by workers exist in the extraction of metal ores and especially in their heat treatment during the production of copper, lead, zinc, iron.

A major pollutant of the environment with arsenic are metallurgical enterprises. In less arsenic is released during combustion of coal (TEPP). Arsenic is present as an impurity in some superphosphate fertilizers and detergents.

In the air of clean areas contained by 0.0009 to 0,003 mg/m^3 arsenic. In the area of source of air pollution with arsenic concentration reaches 0,006-0,026 mg/m^3. In surface and groundwater contaminated with arsenic amount is about 0,010 mg/dm^3, in mineral waters reached 0,125 mg/dm^3. In anthropogenic polluted areas arsenic in surface waters varies widely: from 0,20 mg/dm^3 to 10,4-89 mg/m^3. Soils in uncontaminated areas in the country contain arsenic average of 5-10 mg/kg. Round sources of pollution (ironworks) soil layer of 30 cm contain 8-200 mg/kg arsenic, soil layer 60 cm - 4-55 mg/kg arsenic.

The natural content of arsenic in food is up to 1 mg/kg: vegetables and fruits - from traces to 0,5 mg/kg, beans - from 0,016 to 1,210 mg/kg. An exception to this rule are fish of the sea and some types of mushrooms that concentrate arsenic in amounts corresponding to 166 mg/kg and up to 356 mg/kg. In our established the following amounts of arsenic in food products of anthropogenic polluted areas: corn - 0,250-0,400 mg/kg of raw product; potatoes - 0,007-0,1 mg/kg of raw product; pears - 0,017-0,60 mg/kg and maximum in alfalfa - 1,175-2,250 mg/kg.

Industrial discards arsenic is more toxic trivalent form which has a greater carcinogenic activity. In the soil, it is oxidized to the pentavalent form weakly active, which is found normally in the surface soil layer. Increasing the trivalent form is a direct indication of intense contamination with arsenic and violation of the processes of self-purification in soil. These changes in biotope affect and biocoenoses, as occurs phytotoxic action, suppresses to the growth of microorganisms, higher organisms die. Due to the selective accumulation of arsenic in the body of aquatic life, possible inclusion in the food chain.

METABOLISM AND MECHANISM OF ACTION

Arsenic is nonessential and toxic to human element. The toxicity of arsenic compounds depends on the degree of oxidation of the metalloid, its chemical form and its solubility in biological media. It decreases in the following order: arsine, inorganic and organic compounds of trivalent arsenic (arsenites), inorganic and organic compounds of pentavalent arsenic (arsenates), metallic arsenic.

The total amount of arsenic in the body under normal conditions amounts to about 1 mg, due to everyday entry - about 47 μg.

In pure state arsenic is insoluble and poorly absorbed in the body. Arsenic compounds are absorbed easily in the gastrointestinal tract (80-100%), and therefore oral entrance is the most important quantitatively way of penetration. The work environment arsenic enters the body mainly by inhalation. About 35% of infiltrating the lungs arsenic stays and 50-80% is absorbed. It is possible penetration and skin contact.

Repeated absorption subtoxical doses of diarsenic trioxide leads to the development of tolerance, possibly by way of reduced absorption of intestinal contents.

Arsenic in serum is protein bound and his fraction in erythrocytes is almost entirely associated with globin. Metalloid quickly passes from the blood into the organs. In exposed workers have shown elevated levels in the lungs, liver and kidneys.

Arsenic accumulates partially (about 30%) in the skin, hair, nails and skeleton. Is eliminated mainly by the kidneys in urine (70% of absorbed arsenic) and to a lesser extent by the hair, nails, feces, sweat and failing epithelial cells. Arsenic crosses the placental barrier and is secreted in milk. Blood brain barrier is impermeable to the inorganic forms.

Determination of inorganic and organic forms of arsenic in urine is recommended as a specific and sensitive method for biological control.

PREVENTION

Integrated application of all known measures to reduce exposure to arsenic in the workplace is required by proven its carcinogenic potential.

Preliminary and periodic medical examinations are required. It recommended diet D for protective professional feeding and giving additional vitamin C 100 mg, vitamin E 20 mg, vitamin B_2 - 2 mg, enriched with pectin, fruit drinks and fruit purees, mousses.

BERYLLIUM (Be)

HYGIENE STANDARDS

Air of working environment: Mean-shift MAC - 0,002 mg/m^3.

Surface water: I category - 0,0002 mg/dm^3, II category - 0,0002 mg/m^3, III category - 0,002 mg/dm^3.

GENERAL CHARACTERISTICS

Beryllium is a light, pale-grey metal. In the compounds is divalent. Are known isotopes of beryllium having an atomic mass 7, 8, 9, 10, and 11. Melting point 1284°C, a boiling point of 2970°C, a density of 1,85 g/cm^3 at 20°C, insoluble in water. Forms salts that are soluble in water.

In the lithosphere and crust contain from 6.10^{-4} to 4.10^{-4}% beryllium. Currently, there are known 54 beryllium minerals, most silicates, phosphates, oxides,

borates, arsenates and carbonates. The most common is berilat, also known as emerald, aquamarine, etc., Containing 10,5-14,3% Be. Manufactured significance have more: fenakit (45,5% BeO), chrysoberyl or alexandrite (18.1 to 20.7% BeO), bertrandit (39.6 to 42.6% BeO) and others.

Typical satellites of beryllium in these minerals are opal, chalcedony, gypsum, mica, quartz and others. The natural content of beryllium in the ore is less than 1%, and therefore must be subjected to enrichment for the production. Pure beryllium (99.98%) was obtained by distillation.

Beryllium vigorously migrated in the environmental objects. Concentrated beryllium is found in coal and oil. By products of their combustion spread into the environment. The contents of beryllium at objects with low pH (acidic pH): soil, mineral waters, containing sulphates and fluorine, is higher. Rarely found in sites with pH above 7.5 to 8.0. Around deposits of beryllium are formed biogeochemical provinces with a surplus of beryllium, which was observed growth of malformed conifers.

USE

Production of aircraft, missiles and spacecraft is unthinkable without beryllium and its alloys. They are used in all elements of the construction of spacecraft, compressors, air- and gas turbogenerators, brake systems of airplanes. Beryllium is recognized as the most suitable material for making VHF antennas of radars, telemetry devices to transmit signals and mirrors of telescopes. "Transparent" for X-rays, it is used in the manufacture of windows of X-ray tubes. In nuclear reactors used for reflection and slow flows charged particles and radiation.

In the machinery used beryllium bronze (4%) for the production of electricity contacts, explosion-proof tools and bearings.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Due to the high biological activity, high toxicity and specificity of the biological action of beryllium (allergen and a carcinogen group 1), the professional contact with a high risk. Especially dangerous are the professions and jobs associated with obtaining beryllium and it's alloys; welding beryllium alloys; production and processing of beryllium windows for X-ray tubes. Through automation and pressurization of equipment and production processes possible content of beryllium in the air of the working environment is reduced to 0,01-0,36 μg/m^3 (at hygiene standard 2 μg/m^3).

Sources of pollution of Be in air, water, soils are power plants (of coal and oil), motor vehicles and companies extracting or using beryllium and its compounds. The content of beryllium in the air of cities reached 0,0009 μg/m^3. The total import of beryllium in the body for 24 hours is around 10-20 μg.

METABOLISM AND MECHANISM OF ACTION

Beryllium is a nonessential ultramicroelement, toxic in extremely small quantities. Critical toxicity has Be^{2+} which has general toxic, allergic, carcinogenic and embryotoxic action. For soluble compounds is typical irritation. The most toxic are the following beryllium compounds: chloride, fluoride, oxyfluorides, acetate, sulfate and oxalate.

Beryllium enter in the body mainly by inhalation or through skin. At inhalation of beryllium in lung develop process of formation of specific granulomas. The disease is called berylliosis. Observed changes in the immunological condition of the body and the activity of enzymes catalyzing energy processes in the body, since beryllium enter into competitive and often antagonistic relationship with ions of biologically important divalent metals (magnesium, calcium, manganese, etc.) which are natural activators of enzymes. Attempts to quantify toxic effects shown that beryllium is a potent, tissue necrotizing substance without correlation between dose and response of the body.

Inhalation of soluble compounds principal amount of beryllium by lung passes into the blood and deposited in bone. After oral ingestion absorption is negligible.

Beryllium crosses the placenta and harms the fetus. Secreted by the mammary glands and is dangerous for infants. Easily overcome and hematoencephal barrier.

PREVENTION

To prevent chronic professional and non-professional poisonings apply complex preventive measures: cut off sources of air pollution to Be; insulating buildings, which works with Be or its alloys containing more than 20 percent Be; maximum mechanization, automation, effective ventilation, preliminary and periodic medical examinations, personal protective equipment.

Containing beryllium solid waste reprocessed. If processing is impossible, they are collected and disposed of appropriately, mostly buried in concrete repositories.

Industrial wastewater containing beryllium subject to compulsory treatment.

Protection of ambient air on beryllium apply pollution systems of multistep purification of air with the mandatory use of high efficiency electrostatic precipitators as a last step.

VANADIUM (V)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC for vanadium - oxides and inorganic compounds - 0,05 mg/m^3.

Air settlements: Daily average MAC of vanadium pentoxide - 0,002 mg/m^3.

Surface water: I category - 0,01 mg/dm^3, II category - 0,1 mg/dm^3, III category - 1 mg/dm^3.

GENERAL CHARACTERISTICS

The pure vanadium is greyish-blue malleable metal with a specific gravity of 5.688 and a melting point of 1735°C. Vanadium exhibits high reactivity and forms a number of complex inorganic and organic compounds, but the most characteristic is a chemical bond V-O.

The average content of vanadium in the Earth's crust is 5.10^{-2}%. He is distracted element occurs in the composition of titanoiummagnetitic and sedimentary iron ores and oxidized ores containing copper, lead and zinc. Natural concentrates of vanadium are coal and oil.

Upon heating vanadium forms oxides with varying degrees of oxidation: VO - insoluble in water but soluble in acids, naturally rapidly passes into the trivalent vanadium; V_2O_3 - soluble in acids, slowly oxidises under the influence of the air; VO_2 - exhibits amphoteric properties, easily oxidized to pentoxide; V_2O_5 - most wide spread vanadium compound, moderately soluble in water, well dissolves in bases forming salts: ortho-, para- and metavanadates. By number of compounds formed, vanadium concede only to carbon.

USE

Vanadium is used in metallurgy for the preparation of a variety of steels and alloys with other metals. V_2O_5 is a raw material for the preparation of metal vanadium and vanadium alloys of aluminum, titanium, and iron. From it are prepared, and vanadium catalysts necessary for the production of sulfuric acid. As chemical reagents used various salts of vanadium.

Metavanadat is used in the production of aniline paints, colorful ceramics and glass. Furthermore, the vanadium pentoxide and its salts have been used for catalysis in the production of phthalic and maleic anhydride, vinyl acetate, cyclonexilamine and various oxidation reactions. Ferovanadium be used to obtain high-quality steel.

PROFESSIOINAL AND UNPROFESSIONAL EXPOSURE

Concentration of dust in vanadiumcontant slag at crushing reaches tens mg/m^3. The process of receiving and processing of vanadium pentoxide accompanied by the release into the air of the working environment of condensation and disintegrating aerosols whose total amount fluctuated from 0.03 to 1,0-2,0 mg/m^3.

In the preparation of pure reagents (vanadium salts and vanadium pentoxide), their concentration in the air reaches 2,0-6,0 mg/m^3. Production of ferovanadium accompanied by intensive formation of aerosols with a complex composition. In general concentration of metal aerosols 10-30 mg/m^3 content of vanadium in the air at melting fluctuated from 0.05 to 3,0-4,0 mg/m^3.

When cleaning steam generators of TEPP, fueled by fuel oil, the concentration of vanadium in the breathing zone of workers fluctuates widely and reach tens mg/m^3.

The main source of vanadium in the environment are central fueled by oil, fuel oil and coal. Remains of burning oil contains more vanadium than coal. In oil refining vanadium entirely goes into mazut oil, where its content reaches 1,400 g/t mazut. When burning fuel oil 90% of vanadium be disposed of with smoke.

Source of pollution of air, water and soil with vanadium are metallurgical and chemical plants where they receive vanadium compounds or using them to obtain alloy steels. In the area of metallurgical plants concentration of vanadium pentoxide range from 0.98 to 2 μg/m^3.

Wastewater from producing vanadium plants contain up to 700 mg/dm^3 vanadium and after treatment - 0,34 mg/m^3.

The natural content of vanadium in fresh produce is about 2 μg /%. Most rich in vanadium are beans - 203 μg%, cabbage and carrots - 111-112 μg% and apples - 21 μg%.

METABOLISM AND MECHANISM OF ACTION

Vanadium is essential ultramicroelement in mammals, but the evidence that is the same for people still are not enough. The amount of vanadium in the body is harmless to a certain level, above which the observed manifestations of toxicity.

Body content of vanadium is 100-200 μg no tendency for accumulation. The main source of vanadium is food, daily admission intake is around 10-30 μg. The level of absorption of vanadium contained in food is not known.

The importance of the respiratory route of administration of vanadium in the body increases in pollution of environment and production conditions. It is believed that the absorption of vanadium in the lungs is faster and higher than intestinal and reaches 25 to 100 percent of inhaled and retained amount of vanadium. Absorption through the skin has no practical significance.

Absorbed vanadium is evenly distributed in the

body. Briefly retained in bone, liver, kidney, muscle, spleen, endocrine and sexual glands. Excreted primarily in the urine, particularly with increased exposure.

In the biological control of the exposure can be used the following tests: urine vanadium, vanadium in the blood (serum), plasma, vanadium in hair and in faces.

PREVENTION

To optimize working conditions is particularly important introduction of continuous and centralized production processes. To reduce air pollution using scrubbers and electrostatic precipitators, and the processes of crushing, grinding and roasting of vanadium slag takes place in pressurized equipment with effective local ventilation and remote control. The presence of a powerful local ventilation is especially important in the production of vanadium metal and the vanadium chlorides.

To protect the inhalation is recommended to use respirators. To protect the body and arms are recommended suits of special suitable materials and rubber gloves.

In prophylactic medical examination must participate therapist, ORL specialist and radiologist. Individuals diagnosed with pneumoconiosis, diffuse pneumosclerosis, anemia, liver damage is vocational rehabilitation. The same recommendation is valid for workers with allergic diseases of the airways and bronchi.

MERCURY AND ITS COMPOUNDS (Hg)

HYGIENE STANDARDS

Air of the working environment:

- Mercury - vapour of metal in the elementary state - mean-shift MAC 0,05 mg/m^3;
- Inorganic and aryl compounds - mean-shift MAC - 0,1 mg/m^3;
- Organic and alkyd compounds - mean-shift MAC - 0,01 mg/m^3,
- Short-term maximum MAC - 0,03 mg/m^3.

Air settlements: Mercury and metal compounds - daily average MAC - 0,0003 mg/m^3.

Surface water: I category - 0,0002 mg/dm^3, II category - 0,001 mg/dm^3, III category - 0,001 mg/dm^3.

Food: From 0,005 mg/kg for milk and infant food, milk-based to 0,1 mg/kg for guts and dried vegetables; maximum of 0,4 mg/kg for mussels and 0,6 mg/kg of freshwater fish (0,7 mg/kg - sea fish).

GENERAL CHARACTERISTICS

Mercury is a silvery liquid metal with a molecular weight of 200.59 and density of 13,546 g/cm^3 at 20°C. At temperatures below -39°C becomes white and malleability. At normal temperature, was evaporated. The maximum possible concentration at 20°C was 15,2 mg/m^3. Mercury vapours are 7 times heavier than air. Its solubility in water is low, increases with the increase of the oxygen content in it. Dissolve in hydrochloric acid, hot concentrated sulfuric or nitric acid and so-called. "aqua regia". Mercury dissolves number of metals (gold, silver, zinc, lead, tin, etc.) to form amalgams. In normal conditions it is not oxidized by atmospheric oxygen.

Mercury is distracted element. Natural concentrates of mercury are sulfide ores. Small amounts are found in native condition. The average mercury content in the Earth crust is 8.10^{-6}%. The total amount of mercury in seawater is 206 mil. tons, and the above-ground biomass - 0.5 mil. t. The total amount of mercury in the air is 300-350 t, as the concentration over the land is in an order higher than that over the ocean. It is believed that the reason for this is mainly agricultural work. With 1 m^3 of rainwater falling on the ground 200 μg mercury, per 1 year the total exceeds 100 000 t, which is 15-20 times more than the global annual production.

Mercury intensively included in the so-called water migration, characterized by a high absorption coefficient of brown algae (200.0). This coefficient for terrestrial plants is 7.58. Stably fixed in the soil by forming complexes with humic acid. The half-life of the soil was 250 years. From land to ocean waters annually in the form of soluble organic compounds pass 2.6 thousand tons of mercury; from the soil to the air - 1000 t; in the biological cycle includes 40 thousand tons.

The sources of mercury in the environment are anthropogenic and natural. Natural are divided into: global (Earth's mantle, ocean, underground and surface water, biosphere), regional (mercury deposits - ore zones and regions) and local (ore fields). In environmental and working environment mercury is present in a wide range of physico-chemical conditions and compounds, each of which has specific chemical and toxic properties. They are classified as follows:

1. Inorganic mercury forms:

a) the vapour of mercury in elemental state,

b) inorganic compounds of mercury (II) and mercury (I), which in the body is converted to Hg^{2+}

2. Organic mercury compounds:

a) alkyl mercury compounds: methylmercury, ethyl mercury and propyl mercury

b) alloxy alkyl mercury compounds

c) aryl mercury compounds as phenyl mercury and others.

Vapour of mercury in elemental state are the most common contaminants in the work environment and alkyl mercury compounds (methylmercury) - in the environment. They are the most toxic compounds.

Technogenically dispersed mercury compounds (soluble salts, organic compounds) are distinguished by pronounced geochemical mobility as compared

with natural (mainly sulfides, hardly soluble and weakly volatile), and therefore are environmentally hazardous.

USE

The mercury is used in various industries: preparation of chlorine and caustic soda (mercury cathode electrolysis); synthesis of inorganic and organic mercury compounds (including organomercury pesticides); in electrical engineering and instrumentation; the extraction of precious metals (gold) from ore; for steel alloy materials; heat transfer; catalyst in the chemical industry; to obtain amalgams and amalgamation (incl. in the dental practice); as an anti-rot wood; in laboratory and medical practice; production of lamps - quartz and fluorescent lamps, X-ray tubes and explosives.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Mercury is a classic example of how at occupational exposure critical effect and critical organ in the same substance may change depending on the chemical form, route of administration, duration and object of impact. In short-term exposure to high concentrations of mercury vapor critical organ are lungs. In long term (chronic) exposure to mercury vapor critical organ is the brain.

Critical organ of inorganic salts and aryl derivatives of mercury are kidney. Critical organ of alkyl mercury short-chain compounds (methylmercury, ethyl mercury) is the brain and at pregnant women - the brain of the child.

Mercury is absorbed by the building materials and structures in working and living areas, wooden items of equipment, furniture, clothing and more. The quantity of absorbed mercury depends on the time of impact, the amount of mercury in the air, the properties of the absorbent material and the temperature. Contaminated with mercury elements of the interior at desorption turn into a secondary source of mercury vapor in the room.

Mercury has a pronounced ability to bioconcentration (i.e. food chain), which explains the cases of poisoning by organic mercury compounds from fish as food.

METABOLISM AND MECHANISM OF ACTION

Mercury is nonessential cumulative, a toxic for body element. Body content of mercury in adult is about 13 mg.

Vapour of mercury in elemental state (as a contaminant in the work environment most common) enter through the airways. The absorption in the lungs is high (80%), which includes about 80% retention and 100% absorption. Absorbed mercury quickly passes into the bloodstream. After accidental ingestion of metallic mercury absorption in the gastrointestinal tract is negligible - 0,01%. Absorption of inorganic mercury compounds in the gastrointestinal tract is estimated at about 7%, depending on the solubility of the absorbed compound.

Alkyl mercury short-chain compounds (methylmercury) are more volatile and dangerous if inhaled by aryl mercury. Pulmonary their absorption is about 80%.

A significant part of the mercury in the diet (the main source of population) in the form of methylmercury, especially in fish. Provide the toxicity of mercury, WHO recommended provisional tolerable weekly submission of mercury 300 μg, at it methyl mercury maximum of 200 μg. Practically all of the received in the gastrointestinal tract methylmercury is absorbed - 95%. The skin can pass toxic acting amounts of alkyl mercury.

Resorption of aryl mercury compounds after inhalation and skin contact is weak. The absorption in the gastrointestinal tract is almost complete.

In the division between organs mercury show affinity for scaly epithelium of the skin, hair, sweat, and salivary glands, thyroid gland, gastrointestinal tract, liver, pancreas, kidney, testes and prostate.

The retention time of the accumulated mercury fluctuated widely for different organs. It is the longest in the brain, kidneys and testes.

Main excretory organs for mercury are the kidneys (by tubular excretion in the urine) and liver (by bile secretion in faeces). With less importance are the secretions of the gastrointestinal tract, sweat, excretion of hair and nails.

Mercury content in blood and urine are informative toxicokinetic tests during nearby exposure, but did not clarify the problem of mercury content in the brain. Exposure assessment of an individual by mercury in urine is possible only at several analyzing 24-hour urine samples for several days at prior exposure over one year.

PREVENTION

The main events for the prevention of chronic mercury poisoning are:

- Eliminating possible sources of mercury in the working environment; proper storage of mercury and its salts; changes in technology aimed at the exclusion and replacement with some less toxic or non-toxic substances;
- Location of sources of pollution of the working environment by mercury vapor;
- Protection of structures, equipment and work furniture;
- Compliance with the rules for safe work and personal hygiene when work with mercury;
- Efficient processing of clothing;
- Preliminary and ongoing instructions on safe work for all persons in contact with the mer-

cury.

Preliminary and periodic medical examinations involving therapist, neurologist, dentist and at indications a psychiatrist and gynecologist, are required. If signs of mercurialism persons temporarily removed from contact with the mercury, with subsequent treatment in ambylatory or clinical setting.

When operating in contact with mercury is recommended that workers receive than diet B more: Vitamin C - 100 mg, vitamin E - 20 mg, vitamin B complex - 1 dragee every other day, vitamin B_{12} - 3 µg, folic acid - 300 µg, enriched of pectin fruit drinks and fruit purees, mousses, 1 l mineral water Kavarna or Gorna Banya, Targovishte, Ivanyane, Belovo Izvorishte, Varshetz, Varna.

CADMIUM (Cd)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC cadmium and inorganic compounds (such as cadmium) - 0,05 mg/m^3. Specific effects: embryotoxic and teratogenic for animals.

Mean-shift MAC for cadmium stearate - 0,1 mg/m^3.

Air settlements: Average annual MAC - 0,00001 mg/m^3, average daily MAC - 0,00002 mg/m^3.

Drinking water: Cadmium in mg/dm^3 not more than 0.01.

Surface water: I category - 0,005 mg/dm^3, II category - 0,01 mg/dm^3, III category - 0,02 mg/dm^3.

Food: From 0,01 mg/kg in milk and infant food, milk-based; to 0,5-1,0 mg/kg in the guts of farm animals (liver, kidney, etc.).

Soil: 5,0 mg/kg.

GENERAL DESCRIPTION

Cadmium is greyish-white, silver, soft and malleable metal having an atomic weight 112.40, melting point 321°C, a boiling point of 767°C, a density of about 8.63 to 8.69 depending on the processing. Insoluble in water, slightly dissolved in hydrochloric and sulfuric acid. At ambient temperature its surface layer is oxidized to cadmium oxide.

Of the compounds of cadmium industrial importance are: cadmium oxide, cadmium sulfate, nitrate, carbonate, sulfide, sulfoselenid and salts with halogens (chloride, bromide, iodide). Recently, introduced in production organic compounds of cadmium with fatty acids (caprylic, stearic, lauric) and intermetallic compounds of tellurium and selenium.

Cadmium is an distracted element and does not form separate ore deposits. In nature, cadmium and compounds thereof are found in sulphidic polymetalic ores, most often zinc, in an amount of 0.1 to 5%. The ratio of cadmium/zinc fluctuated from 1:350 to 1:100. Rich in zinc ores contain more cadmium. The average cadmium content in the Earth crust is 1,3.10^{-5}%. In small amounts are found in all objects of the environment, mainly in soils with alkaline pH, sea waters, bottom sediments. It tends to bioconcentration in plankton.

USE

The most widely used cadmium is in electroplating and production of the alloys. Cadmium coatings are more resistant than zinc in aggressive environments. Therefore, electrical and chemical industry equipment are covered with cadmium or cadmium-zinc alloy. Cadmium plating to airline and shipping details.

Cadmium is a component of many alloys: fast melt, special, with precious metals and for production of bearings. Alloy of copper and 1% cadmium (cadmium bronze) used for telegraph and telephone cables. An alloy of cadmium (20%) with lead and tin is used for typography plates. Copper-zirconium-cadmium alloy is used for high-voltage lines. In the jewelry industry used double (gold and cadmium), triple (gold, silver and cadmium) and quadruple (gold, silver, copper and cadmium) alloys. Cadmium of high purity is used for the preparation of alloys with semiconductor properties (cadmium arsenide, antimonid, tellurite) for opto-electronics industry.

Cadmium is used as a neutron absorber in nuclear reactors. Enter into the composition of the catalysts for the chemical industry. Cadmium sulphide (red and yellow paint) is used in the paint industry for decorating porcelain and in painting. Cadmium chloride is used in the manufacture of paints, cadmium oxalate - in ceramic, cadmium carbonate - in battery production, cadmium stearate - stabilizer in the production of plastics, cadmium iodide and bromide - in photography.

Cadmium is needed in the glass industry for the production of colored glass. It comes in numerous fungicides as zineb, maneb and some superphosphate fertilizer. Its content in superphosphates ranges from 50 to 170 mg/kg. Enriching the soil with cadmium is precisely on account of mineral fertilizers.

Nickel-cadmium batteries are used in satellites, spacecraft and automatic meteostations.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Upon manufacture of cadmium in all industries where it or its compounds are used, to detect cadmium aerosols in the air of the working environment. In the hydrometallurgical method of manufacture of cadmium in zinc plant, the cadmium content in the air of the working environment ranges from 0.01 to 0,062 mg/m^3. In modern technologies of manufacture in all stages of the technological process, cad-

mium in the air of the working environment is under MAC. In termovakuum cadmium plating amount of cadmium aerosols up to 0,9 mg/m^3. Unprofessional exposure is formed by the amount of cadmium entering the body with food, air pollution settlements of motor vehicles and tobacco. The cadmium content of tobacco is about 200 μg%. The amount of cadmium in the body increases with age up to 50 years. At this age it reaches 9-40 mg, varying depending on the geographical area and smoking - at smokers it is twice as high. The main sources of environmental pollution by cadmium are technogenic: enterprises of non ferrous metallurgy and mechanical engineering, electrical, chemical, chemical- pharmaceutical industry, in agriculture - plant protection agents and mineral fertilizers, in the cities - mainly road transport. It is, however, particularly to note that in areas with intensive anthropogenic pollution cadmium content in humus soil horizon of soil is 3-5 times the background and in the green aboveground plant parts amount of cadmium 20-30 times exceeds the background - bioconccentration.

METABOLISM AND MECHANISM OF ACTION

Cadmium is nonessential highly toxic accumulated metal. To prevent toxic effects on workers and the population must take into account all possible sources and routes of administration of cadmium in the body.

Food is the main source of cadmium for population. Crabs, fish and animals (mainly in the kidneys and liver) accumulate relatively large amounts of cadmium. Daily amount of cadmium in the diet ranges from 10 to 85 μg, but in some countries, especially where marine fish are a major food, reported higher values. FAO WHO proposed allowable amount of cadmium intake - 70 μg, but generally acceptable - only 20-50 μg. The absorption in gastrointestinal tract is about 5% (from 1 to 12%). Deficiency of calcium, iron, and protein in the diet increase the absorption of cadmium.

Airway is a major occupational exposure. Deposition of particles having a diameter below 5 μm is about 25%. About 60% of the delayed cadmium is absorbed, ie absorption after inhalation is approximately 15%. It reaches 50% in persistent smokers. Coarsely dispersed particles are propelled by the cilia of epithelial cells into the pharynx and be swallowed.

Skin absorption is found only in contact with organic compounds.

Cadmium is retain briefly in the blood. It accumulates mainly in the kidneys and liver, which in steady state contain 80% of the body weight of metal. In the kidney cadmium is primarily concentrated in the crust whose capacity to accumulate cadmium is limited to about 200-400 μg/g tissue. Above this critical concentration renal dysfunction occurs- firstly proteinuria.

In cells cadmium is associated mainly with metallothionein - a protein containing a plurality of sulfhydryl groups, which bind the metals (cadmium, zinc, iron, copper) and thus protects the sensitive enzyme systems of the cell. Its synthesis in the liver, kidney and intestine was induced by cadmium.

Cadmium is excreted primarily in the urine and less bile. Small amounts are excreted through sweat and hair.

Cadmium is accumulated poison with a half-life of the whole body around 10-30 years, of kidney - 15-40 years, 4-19 years liver and blood - 2.5 months.

Exposure assessment and biological control in professionally exposed individuals using toxicocinetic tests to determine cadmium in urine, blood and hair.

PREVENTION

Radical technological measures against cadmium intoxication is restricting the use of cadmium and its compounds. Since it is not always possible it is important to know that the recommended centralization of the manufacture of cadmium-containing materials within the same industry. It is appropriate cadmium products and compounds are produced in the physical state that prevents or limits to maximum release of aerosols in the air of the working environment. Also recommended: comprehensive mechanization and automation of processes with remote control, introduction of vacuum technology in production processes, equipment with increased hermetisation and efficient aspiration, mechanical loading and pneumatic transport.

Rational architectural and planning solutions are of great importance. Workshops and departments for the production of cadmium and its compounds should be isolated rooms. In mechanical engineering, instrumentation and others sectors such isolation should be provided for termovakuum cadmium plating and galvanic baths.

To protect inhalation should be ensured dust respirators, masks, pnevmohelmet. Through appropriate clothing to protect the skin. It is also recommended the use of creams and shampoos for washing hands, complex-contained.

Preliminary and periodic medical examinations are required. Workers in contact with cadmium is recommended to ensure diet B of the professional safety catering. Additionally provide: vit. C 100 mg/day, vitamin E 20 mg, vitamin B complex 1 dragee every other day vitamin B_{12} - 3 μg, folic acid 300 μg, fruit drinks, purees and mousses, mineral water 1 l of Gorna Banya, Varshets, Varna, Targovishte, Ivanyane

or Belovo.

The role of health education for the prevention of toxicity of cadmium is exceptional. Compliance with the rules of personal hygiene is required. It is prohibited in the workplace persons in professional contact with cadmium to eat, drink and smoke. Given the role of tobacco it is desirable to motivate workers to minimize smoking or completely abandon it.

TIN AND ITS COMPOUNDS (Sn)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC for tin and inorganic compounds, oxides (as tin) - 2,0 mg/m^3; Mean-shift MAC for tin-organic compounds (as tin) - 0,1 mg/m^3.

Food: preserved food and drink in cans - 200,0 mg/kg.

Canned foods and beverages in glass container - 50,0 mg/kg.

Baby foods in metal packaging - 100,0 mg/kg.

Baby food in glass container - 25,0 mg/kg.

Other food products in addition to the above - 25,0 mg/kg.

Drinks without canned in metal packaging - 5,0 mg/kg.

GENERAL DESCRIPTION

The tin is white brilliant soft and plastic metal with a molecular weight of 118.69, density 5,85 g/cm^3 at 20°C, a melting point of 231,9 ° C, a boiling point of 2620°C. In the compounds is of 2$^+$ and 4$^+$ valence.

Tin is a characteristic feature for the upper Earth crust. Its content in the lithosphere is 2,5.10^{-4}%. The concentration of tin in the earth layers is associated with magmatic and hydrothermal processes. Known are 24 natural mineral, with the greatest industrial importance are cassiterite (SnO_2) and statins (Cu_2FeSnS_4). Tin migrates slightly. The dry or moist air having a temperature of to 100°C not oxidize tin (it protects thin, but dense and stable layer of tin dioxide). Insoluble in water is poorly dissolved in dilute hydrochloric and sulfuric acids. With halogens formed di- and tetrahalogenides such SnX_2, SnX_4.

In the air of large cities tin content varies from 3 to 300 ng/m^3, in rural areas is even lower, but anthropogenic polluted areas reaches 0,8 μg/m^3 (in Boston).

Emissions from electric tin concentration reaches 640 μg/m^3. 700 m away from the plant - the source of tin content in the air is about 4,0 μg/m^3. The most intensive polluting tin heating coal and oil.

In the aquatic environment (rivers and lakes) the average concentration of tin 0,5 μg/dm^3, a maximum of 2,5 μg/dm^3. In industrial wastewater are established concentrations of tin to 530 mg/dm^3.

The concentration of tin in soils vary from a few to several hundred mg/kg (data - up to 410 mg/kg).

The content of tin in food products is as follows: potatoes - 0,19-0,65 mg/kg, wheat - 5,6-7,9 mg/kg, peas - to 18,7 mg/kg. Total by food intake in the body enter from 187 μg to 8,8-17,14 mg/day, and if used for food cans - up to 38 mg/day.

USE

Up to 40% of the amount produced in the world tin is used for tinning of sheet metal for cans. The remaining quantity of tin is used for the production of solders and alloys. As a rule solders are alloys of tin, lead and cadmium. Unleaded solders except tin containing silver, antimony, zinc or indium. Antifriction properties is an alloy of tin, antimony and copper - "babit". Alloy of tin and zinc foil packaging known as tin-foil (silver-paper).

Tin dioxide used for producing heat-resistant enamels and glazes. Crystal tin sulfide is a component of paints, imitating gold plating. Niobium stanid is one of the most famous superconducting materials in the world. Tin component of titanium alloys for avio-industry, of zirconium - for nuclear reactors. Used for the production of automotive radiators, air conditioners, heat exchangers and electronics, dentistry (amalgam).

The excellent properties of tin in casting, as well as its malleability, susceptibility for carving, noble gray-white color explain the use of tin in decorative art from ancient times.

Halogenated organic tin (acrylates, acetates, maleates, and their hydroxyl compounds) are potent insecticides. Used to impregnate textiles, wood structures, vessels as protection against biological destruction due to development of fungi, microorganisms, algae, mussels, snails and others. They are processed ponds against excessive multiplication of phytoplankton and other aquatic organisms.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

No evidence of acute inhalation professional poisonings. Acute poisoning mainly from food. Symptoms of acute poisoning affecting the gastrointestinal tract are registered with canned food and drinks. The lowest toxicity dose tin as component (impurity) in food is 250 mg/kg. Especially dangerous are canned foods and beverages in corrosion and migration of tin by packaging. Corrosion of the package depends on the type and quality of food products, the duration and the storage temperature, the pH and the amount of air in the can. Oxidizing agents (nitrates, copper and iron compounds) anthocyanic pigments, methylamine, sulfur dioxide intensified corrosion, and soluble salts of tin, sugars and colloids (gelatin) will suspend. Vegetables, accumulating nitrates are unsuitable for canning in

cans. The storage of preserved food under unsuitable temperature and the temperature increase with 1°C increase the content of tin in foodstuffs with 2 mg/kg per month. The content of tin in canned food products is increasing, and it is stored in already open cans.

METABOLISM AND MECHANISM OF ACTION

There is no consensus about the relationship tin - human organism. Opinions vary on nonessential to essential alleged, even if proven essential ultramikroelement. Its content in the body of the person generally up to about 17 mg.

He joined mainly oral and, in production - of inhalation. Trialkyltin compounds with short chain penetrate the skin. Absorption of tin and inorganic compounds is low (about 5%). Better absorbed organic compounds. Absorbed amount of tin for a short time circulates in the blood, 80% bound to erythrocytes. Most large amounts of tin found in the kidneys, liver and bones. Its content in still-born babies is negligible, indicating that does not penetrate the placenta. With age, the amount of tin is increased only in the lung, in other organs and tissues after 10-12 years of age concentration of tin are hardly changed. Excreted rapidly from the body mainly by the kidneys and a small part in bile. The low absorption, poor retention in the tissues and rapid elimination from the body explain the low toxicity of tin and its inorganic compounds. Organic tin compounds undergo biotransformation in the liver with the participation of mixed function oxidases, especially cytochrome P-450.

PREVENTION

Prevention of increased receipt of tin in the body of canned foods requires strict control over the entire chain: compliance with the requirements for agrochemical use of nitrogen fertilizers in agriculture, manufacturing and storage of cans.

In an industrial environment is mandatory effective ventilation. High dust respirators are recommended, in contact with tin hydride and tin chloride - filtering mask. When working with organic compounds requires special measures to protect the skin.

MANGANESE (Mn)

HYGIENE STANDARDS

Air in the work environment: Manganese oxide and inorganic compounds (such as manganese) - mean-shift MAC 0,3 mg/m^3; short maximum MAC 3,0 mg/m^3.

Manganese cyclopentadienyl tricarbonyl and others organic manganese compounds (manganese talat) - additives to gasoline (such as manganese) - mean-shift MAC 0,1 mg/m^3.

Air settlements: Daily average MAC for manganese and its compounds (calculated as manganese dioxide) - 0,01 mg/m^3.

Drinking water: Manganese in mg/dm^3 not more than 0.1.

Surface water: I category - 0,1 mg/dm^3, II category - 0,3 mg/dm^3, III category - 0,8 mg/dm^3.

GENERAL CHARACTERISTICS

Manganese is a brittle grey metal which on contact with air easily oxidized, especially if it is fragmented state. It has a melting point 1244 ° C, a boiling point of 2095 ° C and a density of 7.44. It dissolves in acids. Is reacted with a halogen, sulfur, phosphorus, carbon, silicon. When melting manganese his vapours in high-temperature furnace zone associated with oxygen from the air to form brown smoke, mainly from MnO and Mn_3O_4. Element variable valence (from 2^+ to 7^+), manganese oxides formed a large number of which most known as air pollutants are: manganese oxide (MnO), trimanganese tetroxide (Mn_3O_4), dimanganese trioxide (Mn_2O_3) and manganese dioxide (MnO_2). The average content of manganese in the Earth crust is 0.1% spread is distracted state. It is found in ore deposits most often in combination with iron. Vigorously migrates in all objects of the environment. The most flexible in acidic waters. Concentrated in soils and bottom sediments forming iron-manganese concretions.

USE

Manganese and its compounds are widely used in steel production for ferro-manganese, mechanical engineering, chemical industry, glazes, enamels, paints, batteries (dry items), welding electrodes, in the textile industry and in agriculture in the composition of pesticides and microfertilizers, antidetonator for production of environmentally clean (unleaded) gasoline.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

All the literature of chronic occupational manganese poisoning are associated with the operation of its oxygen compounds (oxides) in the mining and processing of manganese ores; casting of high quality steels and alloys; production of dry galvanic elements (batteries); electrodes and fluxes for doing arc and oxygen welding. In these high-temperature processes the disperse phase of the aerosol condensation contains substantially pure oxides of 2- and 3-valent manganese. In the composition of the welding aerosols manganese content reaches 30% of trivalent chromium - 10%, nickel - 6%. The greatest solubility differs manganese, which explains the prevalence his resorptive toxic effect.

Manganese is an essential trace element with high biological activity. Found in the composition

of many proteins, DNA, heparin and more than 100 vital enzyme systems. Receipt of manganese in large quantities can cause both acute and chronic poisoning, because manganese is polythrop cell poison, damaging many organs and systems. Most likely this is due to the complex interplay of biological and toxic effects of the metal, reflecting the complex relationship between manganese dependent enzyme systems and homeostasis of trace elements.

The main source of non-professional exposure to manganese for population is food. Of ingested through food and drinking water in manganese at gastrointestinal tract is absorbed only 3%, probably as divalent manganese. The level of absorption depends on the reciprocal of the level of manganese in the diet and is controlled by homeostatic mechanisms. The amount of iron in the diet and in the body affects the absorption of manganese. In elevated doses manganese and iron are antagonists. Iron deficiency leads to increased transport of active manganese in the duodenum and small intestine. The level of calcium and phosphorus also affect the absorption of manganese.

METABOLISM AND MECHANISM OF ACTION

It is believed that the normal, usual diet contains enough manganese to prevent lack condition.

A major toxicological problem is occupational exposure, particularly in inhalation admission of highly-dispersion manganese aerosols. Secondly, it should be borne in mind ingesting rough- dispersed particles from the nasopharyngeal region. Dermal enter only organic compounds of manganese.

The metal accumulates primarily in the liver. When exposed in workers were identified elevated levels of manganese in the lungs, liver and kidneys. Manganese may accumulate in the hair (especially in the dark hair) and other pigmented areas of the body (retina, pigmented conjunctiva, dark skin, other areas rich in melanin incl. brain formations).

The metal ions are emitted almost completely (98%) of the bile in faeces probably related to bile acids. Only about 0.1-1.3% of the daily receipt of manganese is excreted through the urine. In exposed workers hair can be considered as a way to eliminate manganese of the body.

A specific feature of manganese is its neurotoxicity (manganese parkinsonism).

There are currently no generally accepted toxicokinetic indicator of exposure to manganese. The determination of metal in blood, serum, urine and feces so far not demonstrated its capacity as exposition test to assess the level of exposure. Increased excretion after provocation did not correlate with the degree of toxicity. On the other hand significant correlations between the specialized service in contact with manganese and manganese content in blood and urine also not been established. Weak is the correlation between manganese in blood and in urine.

PREVENTION

Main in the prevention of manganese poisoning is the protection of respiratory organs: effective ventilation and the mandatory use of PPE - dust masks and respirators, goggles, and at welding in closed spaces - gas masks of insulating type.

Preliminary and periodic medical examinations are required featuring neurologist, therapist, dermatologist and ORL specialist.

Recommended diet B of safety professional nutrition.

COPPER (Cu)

HYGIENE STANDARDS

Air of the working environment: Copper - metal vapor (such as copper) - mean-shift MAC 0,1 mg/m^3, irritating; copper - oxides and inorganic compounds (such as copper) - mean-shift MAC 1,0 mg/m^3; copper oxychloride - mean-shift MAC 0,5 mg/m^3.

Air settlements: Copper - Daily average MAC 0,01 mg/m^3;

Copper oxide - Daily average MAC 0,002 mg/m^3.

Drinking water: Copper is not more than 0,2 mg/dm^3. Surface water: I category - 0,05 mg/dm^3, II category - 0,1 mg/dm^3, III category - 0,5 mg/dm^3.

Food products: from 0,4 mg/kg for milk, to 50,0 mg/kg of dried vegetables, cocoa powder, 60,0 mg/kg of offal (liver, spleen, kidney, etc.) and 100, 0 mg/kg for tea.

GENERAL DESCRIPTION

Copper is pink or reddish metal with a high plasticity (ductility), warmth conductivity. Temperature of its melting point is 1083° C, a boiling point of 2543° C and a density of 8.92 to 8.94. Dissolved in 0.3% solution of hydrochloric acid in gastric juice. The chemical activity is not very high. In the compounds is the first or second valence. When heated above 185° C is carried out surface oxidation by air. With wet chlorine forming copper chloride, easily interact and with other halogens. Salts of monovalent copper (I) are practically insoluble in water, while those of copper (II) have a high solubility. Forms numerous complex compounds.

The average copper content in the Earth crust is about 4,7.10^{-3}%. Total world reserves of copper ores amounted to 465 mil. t. Among minerals predominate sulfides, sulfates, phosphates, chlorides, native copper, carbonates and oxides. Native copper occurs in combination with malachite $(CuOH)_2CO_3$, with azurite and other minerals.

The average copper concentrations in the waters

of rivers and lakes is about 7 μg/dm³, at sea - 0,9 μg/dm³. An important role in the migration of copper in the hydrosphere have plankton - zoo- and phytobenthos. Certain types of plankton concentrate copper to 90,000 times.

The copper content in the soil is on average about 15-20 mg/kg. More than 90% of the copper in the soil in the form of organic complex compounds. Migration capability of copper depends on the acid-base status and redox reactions of the environment.

The total copper content in phytomass on the planet is 25 mil. t. Copper is one of the catalysts necessary for the existence of plants, which contain copper in the range of 8,4-24 mg/kg dry matter.

The copper content in the air ranges from 10 to 100 μg/m³.

Bio-indicators of environmental pollution with copper compounds can serve birds that accumulate copper in feathers. Well copper concentrate and can serve as bio-indicators of pollution marine blue-green algae and mussels.

USE

Copper is used in refrigeration, electrical engineering, powder metallurgy and communications. A significant part of the produced copper put in the production of alloys (brass, bronze, etc.), which are used as structural, antifriction, corrosion-resistant, warmth- and electrical conductors in mechanical engineering, vessel industry, aerospace and automotive industry, device and others. Copper salts are used in the manufacture of synthetic fibers, glass and enamel, in electroplating, the preservation of wood in ore dressing and production of mineral paints. Many copper compounds are used as pesticides in pure form (copper sulfate) or in the composition of complex products (cuprozin, cuprozan etc.).

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Main sources of environment pollution by copper are the enterprises of ferrous metallurgy, transport, copper-containing fertilizers and pesticides, plating processes and the burning of oil and coal in various industries. The annual volume of technogenic contamination of copper is distributed in the following way: in the air disposed of 56 thousand tons, of copper, solid waste - 77 thousand tons, of pesticides - 94 thousand tons, of chemical plants - 155 thousand tons. Copper contaminated soil changes its structure - decrease its porosity, water permeability is violated, deteriorates water-air regime of the soil.

Although exposure to copper is common in industry, are not reported chronic toxic effects characteristic of metal, other than local irritation.

There are no known health effects associated with environmental pollution with copper. Many metals, environmental pollutants (molybdenum, cadmium, mercury), influence copper metabolism and can cause a relative deficiency of copper in the body.

METABOLISM AND MECHANISM OF ACTION

Copper is an essential element in a complex role in the body's functions. Toxicity it is relatively low.

The human body contains 100-150 mg of copper; about 10% in the liver and about 50% in the muscles and the bones.

Daily admission of copper intake is about 2-3 mg and fully meets the needs of the body. The absorption in the gastrointestinal tract is an average 57% in children is higher. Depends on the content and the chemical form of the copper in the diet, the rate of gastrointestinal passage, the presence of antagonists such as zinc, mercury, silver, cadmium, molybdenum and the ingredients of the food which reduces the absorption of copper (sulfate, phytates, ascorbic acid, fiber). It is realized mainly in the stomach, duodenum and upper small intestine. The absorption of copper from the gastrointestinal tract is regulated homeostatic. In this process involved metallothionein, which performs the following functions: carrier of copper; freeboard form in the cells and mucosal block, which was saturated at high concentrations and prevent excessive resorption. High doses are absorbed in progressively lesser degree, depending on the saturation of the body with copper.

The data on the absorption of copper by inhalation are scarce.

Absorption in skin contact is possible for some organic compounds of copper.

After the release of metallothionein copper passes in plasma in a free state or linked to amino acids. In the portal circulation, it is transported to the liver, primarily related to albumin. In blood copper is distributed equally between red blood cells and plasma.

The liver is the central organ in homeostasis of copper in, that it be temporary deposited.

Excreted through the bile. Small amounts are excreted in sweat, urine, hair and nails. Copper crosses the placenta and is secreted in milk.

The half-life of the whole body is four weeks.

The copper content in serum (plasma), urine, blood, in hair, nails and can be used for evaluation of exposure.

PREVENTION

To prevent the effects of local irritation on UAP workers recommended after work to make alkaline and oil inhalations.

NICKEL AND ITS COMPOUNDS (Ni)

Carcinogen for humans (Group 1) Nickel metal (Ni); Soluble salts: nickel sulphate ($NiSO_4$), nickel chloride, ($NiCl_2$), nickel nitrate [$Ni(NO_3)_2$], nickel carbonyl (Ni tetracarbonyl) - $Ni(CO)_4$.

HYGIENE STANDARDS:

Air working environment: Mean-shift MAC: 0,05 mg/m^3 for nickel metal soluble compounds, nickel sulphide, nickelchromphosphat (such as nickel); 0,007 mg/m^3 - nickel carbonyl.

Air settlements: Daily average MAC: 0,0002 mg/m^3 nickel - soluble salts; 0,001 mg/m^3 - metallic nickel and nickel oxide.

Surface water: I category - 0,005 mg/dm^3, II category - 0,2 mg/dm^3, III category - 0,5 mg/dm^3.

Food products: from 0,1 mg/kg (milk) to 3 mg/kg (pulses) and 8 mg/kg (cocoa powder and tea).

GENERAL DESCRIPTION

The nickel is resistant to water, the nickel carbonyl - insoluble in water, other compounds are soluble in water in varying degrees (most soluble - nickel nitrate).

Nickel is a silvery white metal with a characteristic luster, very malleable and ductile. It occurs in two modifications: alpha- and beta. The transition from alpha to beta-modification occurs at heating to 250-300°C. Both versions have different crystal grid. The treated metal nickel (sheets, wires, etc.) is a beta-nickel.

Nickel is ferromagnetic, at a temperature of 358°C, loses ferromagnetic properties. Valence is inconsistent (in simple compounds is divalent). Characterized by an average chemical reactivity. It is resistant to atmospheric gases, water, halogens, sulfur and others. Slightly soluble in dilute acids, resistant to strong alkalis. By reacting with acids forming salts of divalent nickel.

The soluble salts of nickel are different in color and characteristic crystals. Nickel carbonyl is a colorless easy flowing liquid with a characteristic sharp odor.

Natural nickel component of the Earth's core, is a mixture of five stable isotopes. The nickel content in the Earth crust is 8.10^{-3}%. It occurs as sulphide copper-nickel, oxidized silicate and arsenic ores. 53 minerals formed. In anthropogenic clean areas nickel content of the soil is constant. Between migration into groundwater, surface water and plants and imports into the soil at the account of the destruction of soil minerals, decomposition of dead plant and animal organisms, fertilize the soil with fertilizers and precipitation from the air, there is a natural equilibrium.

Atmospheric air, water, soil and plant food contaminated by nickel mainly from enterprises (mining or processing nickel) and TEP (using fuel oil or coal). In heavily anthropogenic contaminated by TEP regions are formed artificial biogeochemical provinces, as oil and coal are natural concentrates of nickel.

In acidic and neutral waters nickel vigorously migrates. Concentrated in bottom sediments of contaminated ponds forming hard soluble organometallic complexes.

The nickel content in the plant products is higher than in animal products. It depends on the geochemical characteristics of the region, the intensity of fertilization with superphosphats, use of nickel contents fungicides and anthropogenic pollution. In cereals and pulses content of nickel reaches 0,089-1,09 mg/kg raw grain, in fruits and vegetables - from 0,1 to 2,0 mg/kg fresh weight.

USE

Nickel is used for the preparation of alloys with other metals: iron, chromium, copper and the like. Copper-nickel alloys are characterized by high plastics and corrosion resistance. Ferromagnetic alloys are especially valuable for production of elements sensitive to changes in the magnetic field. Alloys of nickel with cobalt are used for making feroacustical apparatus. By nichrome (nickel and chromium) and feronichrom (iron, chromium and nickel) is made reactive technology, gas turbines and construction of nuclear reactors.

Alloys of nickel with molybdenum and nickel with molybdenum and chromium are used for making chemical apparatus operating in hard agressive environment. Significant amounts of nickel are used for the production of batteries and anti-corrosion coatings: details of the chemical equipment, medical instruments, apparatuses, dishes, gauges and more. Nickel and its salts are catalysts in the chemical industry.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Main source of nickel for the body of persons without professional contact is food. The total amount used with diet is about 250 µg/day.

The absorption of nickel in the gastrointestinal tract is low - around 5%. It is assumed that a homeostatic regulated.

Substance	Molecular weight	Melting point	Boiling point	Density g/cm^3
Nickel metal	58,71	1453°C	Around 3000°C	8,9 (20°C)
Nickel sulphate	154,78	–	–	3,652 (25°C)
Nickel chloride	129,62	–	–	3,508 (22°C)
Nickel nitrate	182,72	–	–	–
Nickel carbonyl	170,71	–	liquid hard	1,361 (0°C) 1,78 (-55°C)

In production conditions nickel joined mainly by inhalation. Inhaled amounts are absorbed better than ingested orally and directly affect by the solubility of the compound. Along with general toxic action dominate the complaints and symptoms of irritation and necrotizing effect on the mucosa of the upper respiratory tract. The most frequently observed chronic sinusitis with anosmia (84.1%), acute sinusitis (5.6%), cysts and cystyc like formations (10.3%). Under the influence of nickel sulphide and oxides develop nickel pneumoconiosis. Serious problems by carcinogenic activity of nickel and its compounds (nickel carbonyl). Latency period - beginning of professional contact - occurrence of lung- or paranasal sinuses cancer is 13 to 31 years.

Sensitizing activity of nickel has been proven both in professional contact and in living conditions. Improvements in technology limited professional contact and reduce the spread of professional nickel dermatitis. Recently nickel dermatitis is more common among atypical professions (surgeons, dentists, shop assistants, cashiers, waitresses, hairdressers). Due to the widespread use of nickel-plated objects in life, sensitization and allergic dermatitis and eczema in population increase. Dermatitis have been described in contact with nickel-plated frames of glasses, rings, watches, buckles, clasps and safety pins, nickel coins, tableware, disinfectants and detergents containing nickel compounds. There is a trend towards worsening the problem, since the use of nickel products is increasing steadily - by an average of 10% per annum.

METABOLISM AND MECHANISM OF ACTION

Nickel enters the body by inhalation and oral route. In studies of the conditions and mechanism of the occurrence of skin sensitivity was shown that the sensitization of the organism is associated with penetration of nickel through the skin.

In blood, the metal is distributed between leukocytes and serum. Found in three forms: - Ni (II) complex with albumin; - Ni - metaloprotein complex and Ni - α1 - glycoprotein complex; - ultrafilterable Ni (II) complexes with amino acids or polypeptides.

Body nickel content is about 10 mg, divided in decreasing concentration in bone, lung, colon, small intestine, skin and others. With age, increasing the amount of nickel only in the lung.

Absorbed nickel is excreted primarily in the urine (90%). In the sweat concentration is 20-fold higher than in urine. Biliary excretion is no-significant. Except through sweat glands, secreted by the mammary glands and thus falls in milk.

Professional and non-professional exposure is evaluated by determination of nickel in blood and urine.

PREVENTION

Prevention of chronic professional intoxication with nickel carbonyl and in electrolysis workshops based on the maximum hermetisation of processes and equipment, automation and mechanization of nickel-plated.

In preliminary medical examination is recommended to conduct a skin test. Periodic medical examinations involving dermatologist, ORL specialist and oncologist are required.

In chronic allergic skin diseases and bronchial asthma workers were removed from contact with nickel, chromium and cobalt. Cross-allergenicity is possible.

Also provided strict controls on air quality of the working environment and use of PPE. If you need to operate at high concentrations to use filtering or insulating type masks, waterproof suits and gloves.

LEAD (Pb)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 0,05 mg/m^3; Specific effects: Gonad-toxicity, probably carcinogenic to humans - 2 group. Mean-shift MAC for working adolescents (15-18 years) - 0,005 mg/m^3.

Air settlements: Average annual MAC for lead and its compounds (except TEL), calculated as lead - 0,001 mg/m^3, average daily MAC - 0,001 mg/m^3.

Drinking water: Lead not more than 0,05 mg/dm^3.

Surface water: I category - 0,02 mg/dm^3, II category - 0,05 mg/dm^3, III category - 0,2 mg/dm^3.

Soil: From 20 to 80 mg/kg, depending on the pH of the soil.

Food: From 0,1 mg/kg in margarine, vegetable and animal fats to 10,0 mg/kg in mussels, crustaceans and tea.

The lead content must be determined in the following foods: milk, canned meat, sea fish, flour, spinach, fruit juices and nectars, sugar, candy, chocolate, cocoa powder, infant food of milk, vegetable, fruit and meat-based and soft drinks.

GENERAL DESCRIPTION

Lead is greyish-blue metal with atomic mass 207.2, high density (11,35 g/cm^3 at 20°C), melting temperature 327.4 ° C, a boiling point of 1740 ° C, practically insoluble in dilute acids, soluble in nitric acid, acetic acid and hot concentrated sulfuric acid. The solubility of lead (TEL) in soft water in the presence of O_2 and CO_2 has a great hygienic importance.

Upon heating the lead is oxidized to form oxides, are associated with halogens, sulfur, and tellurium.

By organic lead compounds greatest relevance are tetraethyl-lead (TEL) and tetramethyl. They are insoluble in water. Dissolve well in organic solvents and fats.

Have moderate volatility.

The average content of lead in the earth's crust is 1,6.10^{-3}%. Extraction of lead-zinc ores or containing mainly copper, zinc and iron. Formed around 80 natural minerals: sulfates, carbonates, phosphates - galenit (PbS), anglesite ($PbSO_4$), cerusit ($PbCO_3$).

Lead vigorously migrates into the environment, more active in acidic environments. Through its ability to bioconcentration included in biocircle. Accumulates in great quantities in clay soils, humus and bottom sediments.

USE

Globally, 55% of extracted lead is used for the production of batteries, lead pipes, acid-resistant mechanization for the chemical industry, protection against X-rays and other ionizing radiation. About 22% is used in the form of alloys and 23% as chemical compounds: paints for glass and ceramic industry, stabilizers in the manufacture of polymers and rubber, alkylating organic compounds, antidetonation graft to gasoline and the like.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

In the air of the working environment lead is present as vapor and aerosols. Particularly dangerous to the health of workers are condensing, fine disperse aerosols that are formed during casting or high-temperature processing of the metal. Risk occupations and industries are: founders and metallurgists in casting and refining of lead; miners of lead mines; plumbers; fitters in water and canalization systems (WCS); fitters; shipbuilders; printers; police - street regulators; welders; electric welders; installers of special equipment for the chemical industry; workers from the production of plastics, rubber and caoutchouc; workers of gas stations; decorators of glass and ceramics; workers from the production of batteries; workers for repair of bridges and others in direct contact with lead.

At concentrations of lead in the air of the working environment about 0,01 mg/m^3 content in the blood of exposed workers ranges from 6 to 140 mg% (average 32-41 mg%), which is more than 2 times higher than the control level in unexposed individuals.

Source of lead can be other activities, professions or hobbies for individuals: applied artists potters, masters of unique ceramic and glassware; manufacturers of lead shot and fishing weights; lovers of electronic equipment performing consistently lead solder; collectors of pottery, swept to prepare "healthy" food in them.

Given the long history (centuries) of lead as a companion of human in life and production, currently lead is ubiquitous environmental pollutant. Local sources of pollution with lead are lead-produced and lead-manufacturing enterprises. Large populations are threatened by non-professional exposure to lead through leaded paint, emissions from road transport and drinking water.

Emissions from non-ferrous metallurgy containing more than 0,2 mg/m^3 of lead, pollute the air of open space far above hygiene standards. Within a radius of up to 4 km. ground lead manufacturing enterprises the lead content in the soil reaches 80-3500 mg/kg, at natural background levels, ranging on average from 2.6 to 43 mg/kg.

The impact of road transport as a powerful source of contamination of soils and growing crops on them settled in wide strip 150-200 m on both sides of highways. The most intensive pollution (from 3 to 6 times above background) at a distance of 60 meters from the roadway. A significant proportion (50%) have surface contamination by dust containing large quantities of lead compounds.

Apart from the non-ferrous metallurgy and transport soil contaminated by lead and by micro- fertilizers containing up to 1% lead.

It has been found that lead migrates from the material of the water pipes in quantities from 0,09 to 0,58 mg/dm^3, depending on the pH, water temperature, the amount of lead-containing stabilizer (for pipes made of polyvinylchloride), material thickness and diameter of the tubes. Migration continues from 1 month to 2.5 years, the amount of lead in the water level can reach hundreds of times over the hygiene standards.

Natural lead content in food products is negligible - from 1.1 to 10,7 µg%. In areas with intensive anthropogenic pollution levels of lead can reach from 0,558 mg/kg in wheat to 3,67 mg/kg in some vegetables at the rate of 0,5 mg/kg.

Source of lead can also be drugs, theatrical make-up and some cosmetics.

METABOLISM AND MECHANISM OF ACTION

For human lead is a nonessential trace element, typical xenobiotic at high toxicity and pronounced tendency to material accumulation. Particularly sensitive to lead are children and pregnant women. As a rule, the signs of chronic lead poisoning in children were detected at lower concentrations of lead in blood **(Fig. 1)**. Priority damaging the central and peripheral nervous system, blood cells, metabolism of vitamin D and calcium, reproductive function. The injuries may be irreversible. Lead penetrates placental, which explains the high risk to the fetus. The imperfections of the blood-brain barrier in children under 3 years of age makes them the most vulnerable population groups. Very often in childhood signs of chronic lead poisoning are limited to unusual hyperactivity and lightly mental retardation.

In the body, inorganic lead and its compounds fall through inhalation and ingestion. Inhaled way of penetration is very important in professional contact with lead. Digestive pathway is essential for the whole population. Only organic lead compounds (eg. tetraethyl lead) penetrate through the skin.

The daily amount of lead incoming by diet is mostly about 100-200 μg, but in some regions it

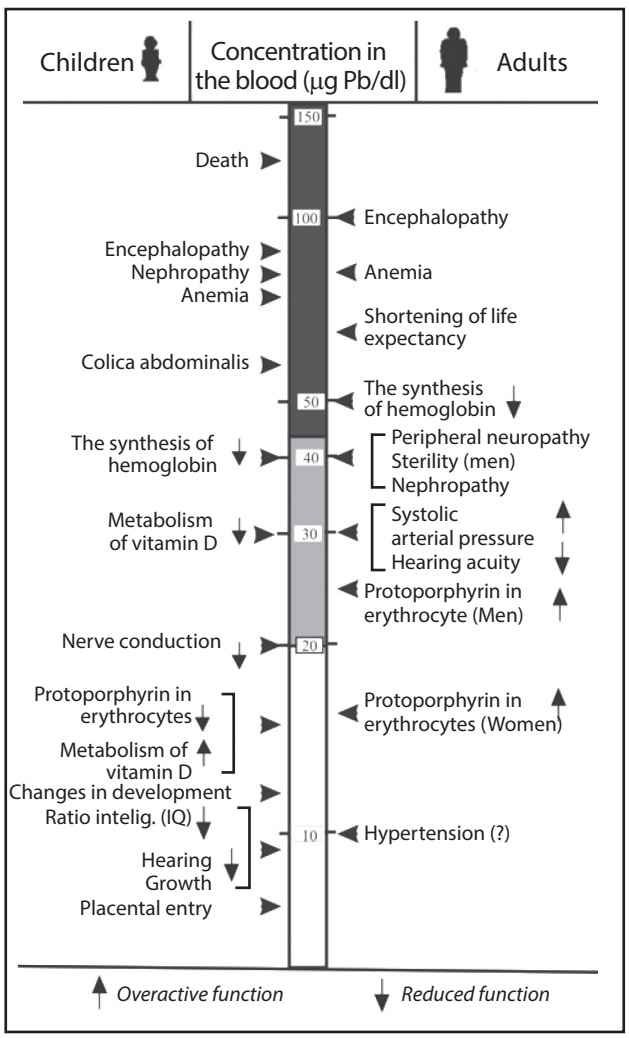


Fig. 1. Influence of inorganic lead on children and adults - the lowest level of observed adverse changes

reaches 500 μg/day. According to WHO recommendations for daily amount for adults is to 400 μg/day. Given the high sensitivity of children US Agency for Control of Food and Drug Administration (FDA) provides for measures to reduce aggregate imports of lead in children 1 to 5 years to 100 μg/day.

Incoming by inhalation and oral - inorganic tract lead is not metabolized. Metabolic transformation is characteristic of the organic lead compounds.

The absorption of lead in gastro intestinal tract (GIT) is about 10-15% of ingested quantity. For pregnant women and children resorption is increased to 50%. Features in the feeding significantly affecting the absorption of lead. It is increased by malnutrition (low caloric and low protein), calcium, iron and phosphorus deficiency.

Up to 35% of inhaled lead particle of size 0,1-1,0 μm are deposited and held in the lungs. Pulmonary absorption is about 30% and depends on the size of the particles, the solubility of the compound, the individual physiological characteristics and the presence of pathological changes in the organism. Rising to 50 percent by inhalation of exhaust gases or soluble lead compounds, smokers and people with chronic diseases of the respiratory organs. In bones and teeth concentrate 95% of the total content of lead in the body - a typical material accumulation. Only 2% is lead in blood and it is distributed so - almost 99% of the circulating lead is associated with the erythrocytes and only about 1% remains in the plasma. It is experimentally found that after a single treatment period of half-life from the blood in adult individuals is about 25 days of soft tissues - about 40 days, from the bones - more than 25 years. Accumulated bone lead is a potential endogenous source - physiological conditions (pregnancy, lactation), stress (alcohol 'hit') or chronic illness activate inert lead in the bones and the level in the blood rises.

Lead is excreted primarily in the urine (75-80%), to 15% of the bile secretions and faeces of the GIT, less than 8 percent with sweat and horny formations - hair and nails. By mammary glands falls in milk in small but proportionate content of blood lead levels.

The exposure assessment is done by conventional toxicokinetic tests: lead in blood, urine, hair and teeth.

PREVENTION

When operating in contact with lead in order to limit the exposure is recommended:

- maximum mechanization of production processes,
- encapsulation of places with intensive dust and spraying water on powdery materials
- wet cleaning of the premises
- a device of hermetic cabins for workers at the casting of lead,
- improving the design of furnaces for high-temperature processing of lead,
- introduction of a continuous process of refining lead and closed production cycles
- increasing the volume of ventilation to 15-20 m/s and more.

Preliminary and periodic medical examinations are required. Workers in contact with lead are provided: the diet B as a safety professional nutrition, vitamin C 100 mg, vitamin E 20 mg, vitamin B complex 1 dragee every other day, vitamin B_{12} - 3 μg, folic acid 300 μg, enriched with pectin fruit drinks and

fruit purees, mousses, 1 l mineral water from Kavarna or Gorna Banya, Targovishte, Ivanyane, Belovo, Varshetz, Varna.

SELENIUM (Se)

HYGIENE STANDARDS

Air of the working environment: Selenium and compounds (such as selenium) - Mean-shift MAC - 0,2 mg/m^3, teratogen for animals; Hydrogen selenium (such as selenium) - Mean-shift MAC 0,03 mg/m^3.

Air settlements: Selenium dioxide - Daily average MAC - 0,00005 mg/m^3, maximum single MAC - 0,0001 mg/m^3.

Surface water: I-III category - 0,01 mg/dm^3.

GENERAL CHARACTERISTICS

Selenium exists in crystalline and amorphous forms. It represents grey-dark with brown hue substance. Under normal temperature most resistant hexagonal crystal grey or metallic selenium. The temperature of the melt was 217°C, a boiling point of 685°C, a density of 4,79 g/cm^3. Does not dissolve in water. Under ordinary conditions it is resistant to the action of air, oxygen, water, hydrochloric acid and dilute sulfuric. Well dissolved in nitric acid and "aqua regia". With fluorine, chlorine and bromine formed haloides with hydrogen at 350-440°C - hydrogen selenium. At high temperature, dissolved in organic substances.

The content of selenium in the earth's crust averages 6.10^{-5}%. Pure selenium minerals and native selenium are very rare. More often there are small selenium- sulphide hydrothermal deposits. There are 38 minerals: selenide (compounds with selenium metals: claustalit PbSe, timanit HgSe, naumanit AgSe etc.); selenites (salts of selenious acid) and seleniates (salts of selenic acid).

In slightly alkaline selenium vigorously migrates and is able to form selenium biogeochemical provinces or biosphere subregions. One of the factors determining the absorption of selenium from the plant is the amount of water-soluble selenium in the soil.

USE

Selenium is mainly used for the production of rectifiers and photopiles. It synthesized various selenides with semiconductor properties that are applied in the manufacture of thermo elements, photoresistors and cathod-luminofores. Selenium is used in the glass industry as a colorant, in the vulcanization of rubber and in metallurgy to form a fine grain structure of the steel.

Selenium is used as a catalyst in the process of hydrogenation. In organic synthesis is used selenium dioxide as the oxidant and the starting material for the preparation of organic and selenites compounds which are used as extractants, antioxidants and drugs.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Occupational exposure to selenium with signs of damage to the health of workers is observed in the copper production; melting of aluminum, coated with selenium and in the manufacture of steel.

Chronic toxic effects in increased exposure of the selenium from the environment are unspecific.

The content of selenium in the air near the copper enterprises and the territory of the factory reaches 0,0004-0,0005 mg/m^3. Much more intense polluting the air with selenium in volcanic activity (in volcanic gas content of selenium is a thousand times more).

In surface water content of selenium rarely exceed 1 μg/dm^3. Maximum values have been established in the mineral waters (acidic and hydro carbonic) - 6 to 8 μg/dm^3.

High ability to accumulate selenium is characteristic of soil bacteria, actinomycetes and fungi. In the selenium content of 0,4 mg/kg in soil in edible and poisonous mushrooms concentration of selenium ranges from 2 to 12 mg/kg dry matter.

The content of selenium in plant products depend on the geochemical characteristics of the region, in environmental pollution with selenium. In legumes contain 0,24-0,73 mg/kg dry seeds. In wheat, rye, barley, oats and peas content of selenium varies around 0,257-0,304 mg/kg dry mass. Significantly less amount of selenium contained in potatoes, corn, cabbage, onions, carrots, cucumbers and apples - from 0,004 to 0,142 mg/kg dry mass. The meat of bovine animals contain 0,929 mg/kg selenium eggs - 0,022 mg/kg, dairy products - 0,100-0,298 mg/kg dry matter.

METABOLISM AND MECHANISM OF ACTION

Selenium is an essential trace element that is toxic to mammals and man when he entered the increased amounts of diet or inhaled air. Toxic doses are 100-300 times higher than those needed for life. Human body contains from 3 to 15 mg selenium. Daily admission to the diet varies from 60 to 300 μg, depending on the characteristics of the area, the composition and preparation of food. The absorption in the gastrointestinal tract is up to 90% (50-100%), influences significantly from the content of methionine in the diet - this amino acid inhibits the absorption of selenium-containing amino acids.

Newly admited selenium is found mainly in plasma associated with the α_2 and β_1 - globulin fraction and the free amino acids (cysteine, methionine, leucine).

After tissue distribution maximum levels found in the kidneys and liver; lower - in the retina, spleen, lung, myocardium, skeletal muscle and brain. Selenium accumulates in hair. At occupational exposure

elevated levels are found in the lung.

In tissue selenium undergoes biotransformation: reduction, methylation and connection or direct involvement in proteins (Se-cysteine instead of cysteine, Se-methionine instead of methionine).

Selenium cross the placental barrier and is secreted in milk. Emitted mainly in the urine, but also in sweat, feces, hair and exhaled air.

In biogeochemical provinces livestock (cows, sheep, pigs, dogs, horses) have so-called "alkaline" disease - chronic selenium toxicity. In the same areas in medical examinations of the population are established changes in the enamel of the teeth, subicteric skin, chronic inflammation of the joints, rash, nail diseases, anemia, gastrointestinal diseases and nervous disorders.

A characteristic sign of increased exposure in chronic selenium toxicity is the smell of garlic in expire air, due to the generated in biometilation dimetilselenid.

PREVENTION

Technological events are the most important and radical measures for the prevention of chronic intoxications in production: hermetisation of equipment; mechanization of operations for loading the reactor with technical selenium; isolation of operations related to the separation of selenium in the air of the working environment in premisses.

Preliminary and periodic medical examinations are required. Pregnant women vocational rehabilitation immediately - without contact with selenium. At work should use personal protection: rubber gloves when working with a solution of selenium thiosulfate; dust respirators and masks in packaging technical selenium; if necessary, gas masks and skin protection.

THALLIUM AND ITS COMPOUNDS (Ti)

HYGIENE STANDARDS

Air of the working environment: Thallium and its compounds (such as thallium) - Mean-shift MAC - 0,05 mg/m^3.

GENERAL DESCRIPTION

Thallium is a soft light gray metal with a molecular weight of 204.37, a meltin point of 303,5°C, a boiling point of 1472°C, a density of 11,82 g/dm^3 at 20°C. Crystallizes in two modifications. Its surface is oxidized by air at normal room temperature. Insoluble in water and bases. Easily react with mineral acid (nitric, sulfuric), but not with hydrochloric acid due to the formation on the surface coating of thallium chloride.

Slightly soluble halides, sulfides, chromates of monovalent thallium it closer to monovalent silver, copper, gold and divalent lead and mercury. Trivalent thallium is readily reduced to the monovalent, form complex compounds which are superior in stability at those of bismuth, antimony, iron, chromium and aluminum. With metals forming alloys.

The content of thallium in the earth's crust is from 0.43% to 1.0.10^{-4}. Occur in the composition of rare minerals, manganese, copper, zinc and arsenic ores. The content of thallium in soil is 1.0.10^{-5}% in seawater - 0,01 µg/dm^3, freshwater 0,01-14,0 µg/dm^3, and in the air - from 0.04 to 0.48 ng/m^3. In areas of minefield thallium content in groundwater reaches 2,7 mg/dm^3.

USE

Thallium and its compounds are used to manufacture semiconductors, electronics and electrical engineering; for doping germanium, silicon and cadmium compounds; for the preparation of photoresistors and photoelements with high sensitivity; in atomic engineering for different scintillation counters; for activation of luminescent crystals; in optics, instrumentation and mechanical engineering. Thallium salts are used as octane antidetonators, catalysts to produce luminous paints, artificial semi-precious stones, pearls and diamonds, in the composition of depilatory creams and pastes, and rodenticides.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

In the production of thallium - metal and its oxides, the concentration of thallium in the premises reaches 0,18 mg/m^3, in casting metal - 13-17,4 mg/m^3. The production of salt, content of thallium in the air of the working environment is about 0,136-0,354 mg/m^3, in the synthesis of mono- crystals - 0,004-0,007 mg/m^3.

The most significant sources of environmental contamination with thallium are thermal power plants of oil, coal and fuel oil and enterprises of non ferrous metallurgy, mining copper, lead and zinc.

Thallium can get into the drinking water and food in highly anthropogenic of contaminated areas and of excessive fertilization with potassium fertilizers.

METABOLISM AND MECHANISM OF ACTION

Thallium is a nonessential ultramicroelement characterized by properties similar to those of lead: material accumulation and toxicity. In the body penetrates in all possible physiological pathways: enteral, inhalation of aerosols and through the skin.

Under normal conditions with food and drinking water daily take 1.6 to 2,0 µg, by inhalation - 0,05 µg. The thallium content in hair was 4,8-15,8 ng/g, in the urine - 0,13-1,69 µg/dm^3, in nails - 0,72-4,93 ng/g. Medium in body contained 100 µg thallium.

Thallium is rapidly absorbed through the all mucous membranes - enteral and UAP, as well as through the skin. Peaks in the blood after 12 hours and soon disappears. Distribute the internal or-

gans, especially kidneys, salivary glands, testes, liver, spleen, prostate, brain. The next redistribution in the body is deposited in bones and hair.

Because of the similarity with potassium possible biological interaction between two elements: thallium can replace potassium and compete with him in the construction of biological membranes. The infusion of potassium increases the clearance of thallium and extraction of the tissues.

Thallium is excreted in urine and faeces. Excretion is long, can reach 1 year. Excretion through salivary glands is substantial, up to 15 times higher than by the kidneys.

Depending on the individual sensitivity the lethal dose for humans ranges from 0.5 to 3 g, minimum lethal dose is 12 mg/kg body weight.

PREVENTION

In all manufacturing processes where possible contamination of the premises with thallium are required mechanization and automation of manufacturing processes, hermetisation of equipment and effective ventilation.

It recommended the introduction of closed cycles excluding manual operations. Machining and polishing crystal should be carried out only under local ventilation.

Particular attention should be paid to the disposal of solid waste that are extremely dangerous for the environment and the population. Working with thallium after the shift must take a shower, and if necessary to organize sanitization of the body and clothing. Frequent change of clothing is obligatory, while repair and emergency works it is used only once. In addition of moisture- and dustproof suits, are provided: respirators, goggles, rubber gloves. Preliminary and periodic medical examinations are required. Recommended monthly survey of thallium in urine.

TELLURIUM (Te)

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 0,1 mg/m^3

Air of settlements: Daily average MAC - 0,0005 mg/m^3 (tellurium dioxide).

GENERAL CHARACTERISTICS

Tellurium is a silver-gray brittle metal with a pronounced sparkle, molecular weight of 127.60, density 6,64 g/cm^3 at 20°C. When heated burns with a blue flame and forms TeO_2. React with halogens, hydrogen formed telluro-hydrogen - colorless, poisonous gas with a sharp odor. Insoluble in water and organic solvents. Dissolved in concentrated nitric acid, in "aqua regia" (a mixture of concentrated nitric and sulfuric acid), low in hydrochloric acid and alkalis. It exists in two allotropic states: hexagonal crystals and dust. With metals form tellurides.

Tellurium is one of the rarest distracted elements. It is represented by eight stable isotopes. The average content in the earth's crust is about 1.10^{-7}%. 40 mineral forms: tellurides and their oxidation products. Typical impurity to pyrite or in a polymetallic, copper- zinc and lead- zinc ores.

USE

Telluride and its alloys are used for the production of semiconductors. Pure tellurium is alloying additive to lead, steel and cast iron; component of alloys with a high resistance; used in the vulcanization of rubber; coloring glass and ceramics industry in the production of reddish glass and ceramics and in the manufacture of latex mixtures.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Professional exposed to tellurium and its compounds (mainly TeO_2) are workers of workshops for electrolysis and obtaining pure metals from ore with impurities of tellurium.

When workers in ferrous metallurgy (steel) exposed to tellurium in concentrations of 0.01 to 0,1 mg/m^3 shall be established: the smell of garlic in expire air and sweat, metallic taste in mouth, anorexia, rhinitis, somnolence, these content in urine up to 0,06 mg/l.

METABOLISM AND MECHANISM OF ACTION

Tellurium is a nonessential ultramicroelement per human. From the known tellurium - metal, tellurides (compounds of metals), tellurites and telurates (salts of telluric and telurorouse acid), at a weak toxicity has tellurium metal. Most toxic are water-soluble tellurites. Tellurium and its compounds enter the body mainly through inhalation and in limited quantity by digestive. Intestinal absorption is low due to poor solubility. Only organic compounds penetrate intact skin. Tellurium accumulates in all parts of the body, most in the kidneys, liver, brain and testes. Internal organs and mucous membranes of the mouth and the stomach or upper respiratory tract, specifically stained dark green to black with earthy nuance. This staining was due to the reduction of the compounds to tellurium metal. Restored tellurium is methylated to dimethyl tellurium, which is the reason for the specific smell of garlic. Excretion from the body of tellurium is very slow, performed mainly by the kidneys. Small amounts are excreted via the bile and stool (dimethyl tellurium and tellurium metal); by lung and sweat (garlic breath from the mouth and the smell of garlic from the body); by salivary glands (metallic taste in the mouth).

The mechanism of toxicity is not fully understood. It is believed that the observed sleepiness and other

Air of the working environment

Chemical	Empirical formula	Mean shift MAC	Short maximum MAC
1. Phosphoric anhydride	P_2O_5	1,0 mg/m^3	-
2. Phosphorus pentachloride	PCl_5	1,0 mg/m^3	-
3. Phosphorus trichloride	PCl_3	1,5 mg/m^3	3,0 mg/m^3
4. Phosphine	PH_3	0,1 mg/m^3	0,2 mg/m^3

Air settlements

Pollutants	Daily average MAC	Maximum single MAC
1. Phosphoric anhydride	0,05 mg/m^3	0,15 mg/m^3
2. Phosphine	0,001 mg/m^3	0,01 mg/m^3

Drinking water

Phosphates in mg/dm^3 not more than 0.5

Surface water	I category	II category	III category
1. Phosphates (PO_4)	0,2 mg/dm^3	1,0 mg/dm^3	2,0 mg/dm^3
2. Phosphorus - the total content as PO_4	0,4 mg/dm^3	2,0 mg/dm^3	3,0 mg/dm^3

neurological symptoms are due to a reduction of the cortical excitability. It is also known to cause growth failure, hair loss, paralysis.

PREVENTION

Workers in contact with tellurium subject to preliminary and periodic medical examinations.

Protection of workers is ensured by effective ventilation in the working premises. In the presence of tellurium aerosols need to wear respirators. Workers in contact and tellur-hydrogen should use filter-type masks. To provide clothing and rubber gloves. It is imperative to observe the rules of personal hygiene.

PHOSPHORUS COMPOUNDS (P)

HYGIENE STANDARDS **(Table)**

GENERAL CHARACTERISTICS

Phosphorus freely in nature does not occur. Was detected in the form of phosphates in the composition of the minerals: chlorapatite, fluorapatite, vivianite or phospharite. Its content in the earth's crust is about 0.12%.

Phosphorus exists in three allotropic states: white phosphorus (highly toxic), red phosphorus - low toxic due to insolubility in biological fluids and black phosphorus.

White phosphorous is a colorless soft crystalline form, lighting in the darkness and quickly yellowing under the influence of light. The melting point of white phosphorus is 44,1°C, boiling temperature - 275°C, flash point 34°C, density - 1.83. Vapour of phosphorus is 4.3 times heavier than air. At ambient temperature is low volatile, in the air form a white fog, containing phosphorus and its oxides. Slightly soluble in water. When access of light and air very easily oxidized and self-igniting. Therefore, it is stored in the dark and under water. Reacts violently with sulfur, halogens and metals.

The red phosphorus is powder with dark red to brown. Melting point - 600°C, a flashpoint of 240°C, a density of 2.2. Red phosphorus is less active than white phosphorus, at normal temperature with water vapor and oxygen react slowly.

Phosphorus is one of the main elements that make up living organisms. As an essential component of protoplasm, nervous tissue and bone, the phosphorus is present in significant amounts in molecules with great biological importance: nucleotides and phospholipids. In nature, phosphorus is the main factor limiting the growth of autotrophic organisms both in the water and on land. It is assumed (for the nitrogen has been shown) that phosphorus is the main regulator of all other biogeochemical cycles. The amount of nitrate in the water and oxygen in the atmosphere depends on the state of the cycle of phosphorus.

Human activity has significantly increased amounts of natural phosphorus. Annually in wastewater of urban sewerage fall 2 kg of phosphorus for each resident of the settlement used to maintain personal hygiene modern shampoos and detergents. The majority of the phosphorus used as fertilizer remains in the soil, because of the high content of calcium, aluminum and iron, which forms a phosphorous compounds. Part of the excess falls in freshwater pools, playing a major role in the occurrence of eutrophication (aging and complete disappearance of aquatic fauna due to accumulation of minerals, primarily phosphorus and nitrogen, and intense growth of aquatic plants).

USE

White phosphorus is used for obtaining red phosphorus, phosphorus compounds and alloys; pesticide is used for rodent control; as fume formative and incendiary means. Specially purified phosphorus is used in the electronics and semiconductor industry.

Red phosphorus is used in the manufacture of matches, agriculture and metallurgy.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Acute poisoning with phosphorus are described exclusively by ingestion of white phosphorus, although possible and acute poisoning with phosphorus steam.

Chronic poisoning of phosphorus vapours are described in concentrations of white phosphorus in the air of the working environment 0,2-1,2 mg/m^3 in the manufacture of matches, also at concentrations 0,1-0,16 mg/m^3 (but at the same time vapour of phosphoric anhydride, phosphine and hydrogen fluoride).

Being on the skin white phosphorus ignites, develops high temperature, causing slow healing burns. Local adverse effect on the site of combustion provide the forming phosphoric acid.

In metallurgy, metal processing, containing impurities of phosphorus, with concentrated acids liberates highly toxic gas phosphine (PH_3).

Accidental or suicidal ingestion of phosphorus compounds (mostly pesticides) causes unprofessional poisoning or death.

METABOLISM AND MECHANISM OF ACTION

White phosphorus enters the body through the respiratory, mucosa of the gastrointestinal tract after ingestion and through damaged skin from burning. Upon receipt of phosphorus in the body at large quantities it is possible to detect in the blood in elemental state. When phosphorus poisoning, occur disturbances in the metabolism of proteins and carbohydrates, in the deposition of glycogen in the liver. And increases fat deposition in the liver.

The excretion of inorganic phosphorus is carried out mainly by the kidneys.

PREVENTION

Manufacturing processes in obtaining and use of white phosphorus must be hermetisated and the premises equipped with efficient ventilation. At work use respirators or filter masks. Special attention is paid to personal hygiene, carefully washing hands, face and body shower. Compulsory oral hygiene and teeth. Persons with carious teeth are not allowed to work in contact with phosphorus. Strictly forbids eating and smoking in workplaces. Limited to manual manipulation of phosphorus (in proceedings provides mechanical loading with phosphorus). It is recommended that workers who performed several professions frequently change jobs in contact with phosphorus. Preliminary and periodic medical examinations are required to X-ray of jaws and others bones by indications. Recommended diet D of the safety professional nutrition.

CHROMIUM AND ITS COMPOUNDS (Cr)

Chromium oxide (III) - Cr_2O_3,

Chromium trioxide (VI) - CrO_3 - proven carcinogen for people - 1 group,

Sodium chromate - $Na_2 CrO_4$,

Potassium chromate - K_2CrO_4,

Sodium dichromate - $Na_2Cr_2O_7.H_2O$,

Potassium dichromate - $K_2Cr_2O_7$,

Chromium trichloride - $CrCl_3$.

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC 0,5 mg/m^3 for chromium - dust from metal, ores and concentrates; 0,05 mg/m^3 for chromium compounds.

Drinking Water: Chromium (VI) - not more than 0,05 mg/dm^3.

Air settlements: Daily average MAC for hexavalent chromium calculated as CrO_3 - 0,0015 mg/m^3; Maximum single - 0,0015 mg/m^3.

Surface water: I category: trivalent chromium - 0,1 mg/dm^3, hexavalent chromium - 0,02 mg/dm^3; category II: trivalent chromium - 0,5 mg/dm^3, hexavalent chromium - 0,05 mg/dm^3; category III: trivalent chromium - 1,0 mg/dm^3, hexavalent chromium - 0,1 mg/dm^3.

Food products: from 0,1 mg/kg (in milk, fruit) to 0,5 mg/kg (in the intestines of animals and birds).

GENERAL CHARACTERISTICS

Chromium is gray hard melting metal with molecular weight 51.99, density 7,19 g/cm^3 (20°C), melting point 1890°C. Chromium and chromium oxide are insoluble in water; chromium trioxide, chromates and bihromatite are soluble. Pure chromium metal is plastic. Chemical is less active. Under ordinary conditions it is resistant to oxygen. Formed compounds with fluorine and salts with oxygen-containing acids. In the compounds is from 2+, 3+ or 6+ valency. Derivatives of bivalent chromium are very strong restorers; of trivalent chromium - can be restorers or oxidizing agents. The most resistant are the compounds of trivalent chromium.

USE

Wide use of chromium due to its thermal stability, hardness and corrosion resistance. Best known is as an alloying addition to steel, stainless steel, heat- and acid resistant. Chrome-nickel alloy (nichrome, nimonik) are used for the manufacture of reactive equipment, gas turbine and nuclear reactors. Significant amounts of chromium are used for decorative corrosion - chrome-plating. Chrome powder is used in metal ceramics and production of welding electrodes. Trivalent chromium alloy is used for the jewelry industry and ruby laser technique. Chromium oxide is a raw material to produce chrome, paint for glass and ceramics, chrome catalysts. Obtained from

chromium trioxide.

Chromates is used as chemical reagents in the textile industry, leather industry (tanning solutions), wood (wood preservatives) and agriculture (decontamination of seeds).

Bichromates are widely used in metal, leather, textile, ceramic industry, for the production of paints and varnishes and in pyrotechnics.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Lithosphere chrome is 0.055%. In the free state does not occur. The basic form is a natural chromium oxide. Chromium forms a 20 minerals, mainly compounds of oxygen and iron: chromite, kemerit etc.

In 0,5-2 km radius of chromworking enterprise ambient air contains chromium 10 to 30 times more than sanitary norm.

The natural content of chromium in open reservoirs fluctuate widely, up to 0,112 mg/dm^3. In seawater chromium content is about 0,05 μg/dm^3, in the oceans - 0,28 μg/dm^3. In drinking water, the average chromium content is about 0,0023 μg/dm^3.

In unpolluted soils natural chromium content is about 1,8.10-3% depending on the type of soil. Concentrated in the topsoil horizon.

Chromium was detected in all the plants in concentrations of up to about 5 mg/kg. Up to 95% of the total was concentrated in roots.

Chromium is found in all foods of plant or animal origin. Presented in the form of inorganic compounds or as coordination complex with nicotinic acid and amino acids so-called glucose-tolerant factor (GTF), with pronounced influence on carbohydrate metabolism.

Acute inhalation intoxication in production are described in concentrations of 20 mg/m^3. Concentrations in the range of 4-5 mg/m^3 cause perforations of the septum nasi in more than half exposed persons for a period of six months to three years. In all workers observed rhinitis and pharyngitis, and for some individuals it is possible to detect an enlarged liver and kidney dysfunctions

METABOLISM AND MECHANISM OF ACTION

Chromium enters the body mainly by the food in an amount of 25 to 282 μg/day, usually 50-150 μg/day. Deficiency in the diet of trivalent chromium, which is an essential trace element for the body, is a topical issue for some countries (recommended introduction of chromium in the amount of 50 to 200 μg/day to overcome the deficit in food).

In the human body contains 6-12 mg chromium. With age the amount is reduced.

The greatest amount of chrome settled in her hair, liver, lung, kidney, endocrine glands and intestines. Tissues of newborns contain more chromium than those of adults. The chromium content in the heart, lung, aorta and spleen decline in the first 10 years of life, in the liver and kidneys had been held during the second decade. Lungs is the only organ with increasing concentration over time.

The chromium content is significantly reduced in ischemic heart disease, particularly myocardial infarction, and also in the duodenal ulcer disease and liver disease.

The content of chromium in hair, nails, blood and lymph nodes of persons in professional contact with chromium is significantly higher.

Trivalent chromium is an essential trace element, hexavalent chromium is highly toxic. Intracellular its conversion to trivalent chromium is the basis of the critical toxic effects.

Chromium enters the body by inhalation and digestive tract and through the skin. The absorption of chromium in the lung (mainly soluble compounds of hexavalent chromium) can be significant and fast. Chromium accumulates in the lung into insoluble compounds.

The absorption of chromium in the gastrointestinal tract is about 1% on average, for the trivalent chromium ranges from 0.1 to 1.2%, for hexavalent chromium - 2%, for GTF - up to 10%. Cell membranes are readily permeable to the chromium (VI), but not to the chromium (III). In mitochondria and nuclei of chromium (VI) is reduced to chromium (III) and remains in this form in the tissues and in the connected state.

Chromium, probably as GTF passes transplacental and in the same form secreted in milk.

Urine is the main route of excretion of chromium. Faeces contain mainly unabsorbed from food metal.

In biological control using the following toxicokinetic tests: urine chromium, chromium in blood, serum (plasma) and chromium in hair.

PREVENTION

The most important and radical measures for the prevention of chronic professional intoxications are technological and technical: replacement of toxic hexavalent compounds with low toxic; full hermetisated equipment; maximum mechanization and automation; effective local ventilation.

Entering on work subject of preliminary medical examination. In periodic medical examinations involved and oncologists. Persons with pneumosclerotic changes in the lung and/or recurrent skin lesions are vocational rehabilitation.

It is necessary to ensure strict monitoring of compliance with individual protection measures: respirators or masks of insulating type; rubber gloves; suits with hoods, and for galvanotechnicians and handling solutions - waterproof clothing; protective

ointments, creams and pastes for hands of the type "biological gloves". To clean the skin of the hands and decontamination of chromium, recommended special protective ointments containing EDTA and ascorbic acid. Handwashing is appropriate to use the 5% solution of sodium thiosulfate or 10% sodium bisulfite.

Importance is the prevention of skin trauma, and inflammatory diseases of the upper respiratory tract.

ZINC (Zn)

HYGIENE STANDARDS

Air of the working environment: Zinc oxide (such as zinc) - Mean-shift MAC - 0,5 mg/m^3, maximum short MAC - 10,0 mg/m^3.

Air settlements: Daily average MAC - 0,05 mg/m^3.

Drinking water: Zinc not more than 5,0 mg/dm^3.

Surface water: I category - 1,0 mg/dm^3; category II - 5,0 mg/dm^3; category III - 10,0 mg/dm^3.

Soil: from 20 to 370 mg/kg depending on the pH of the soil.

Food: from 5,0 mg/kg for milk and infant food milk-based to 200,0 mg/kg of mussels, crustaceans and others.

GENERAL CHARACTERISTICS

Zinc is a silvery blue shiny metal, stronger reducer, able to shift much of divalent metal from its salts. In compounds always of the second valency. The temperature of melting is 419,5°C, the boiling temperature of 906°C and a density of 7.133 at 20°C.

Zinc - metal readily soluble in dilute acids. In water well soluble following salts of zinc: chlorides, bromides, iodides, nitrates, sulphates and acetates. Low soluble are zinc sulfide, carbonate, fluoride, phosphate, arsenate, arsenite, chromate, oxalate, silicate, cyanide, ferrocyanide.

Zinc is widely distributed in nature element, its content in the Earth's crust is 1,5.10^{-3}%. It enters into the composition of a large number of minerals. Pure zinc ores in nature as a rule hardly meet. For obtaining zinc, polymetalic ores are processed, wherein the initially obtained concentrates comprising: zinc - 48-58%, lead - 1-2%, copper - 2%, iron - 5-10% and sulfur up to 30%. The zinc content in the air of cities around 0,040-0,047 μg/m^3.

Zinc content of the soil depends on the nature of the sediments, organic matter content, the texture of the soil and its active reaction (pH). Ranged from 15 to 80 or more mg/kg, in the areas of ore deposits reached n.10^{-2} - 1%.

In sea waters the amount of zinc fluctuated from 2 to 50 μg/dm^3. In surface freshwater pools zinc content varies more widely: from 0.6 to 120, and in some regions of Siberia to 577 μg / dm3, average 1,3 μg/dm^3. Significantly higher concentrations of zinc are found in the waters of the regions of ore deposits - up to 20-35 mg/dm^3.

The zinc content in the groundwater outside the ore areas averaged about 10 μg/dm^3, rarely reaches 50 μg/dm^3, while the waters draining the deposits amount of zinc is 1000 times more.

Zinc is essential bio-element and is contained in all the tissues of plants and animals. The average content in the body of reptiles and amphibians is about 1-3 mg%, and in the body of mammals and birds - 23 mg%.

Ability to accumulate zinc have forage grasses (alfalfa), natural grass vegetation and some poisonous plants. In the food content of zinc ranges from 0,47 mg/kg in egg white to 58,2 mg/kg in meat. From plant products as much zinc is in the parsley, carrots, potatoes, beets, cabbage; of cereals most zinc contained in the rye; the maximum amount of zinc found in pomegranates.

USE

The most important is zinc as galvanoplasty (58.8% of all plating) to protect iron from corrosion. Zinc is used in industry for the preparation of alloys: with copper (brass); copper and lead (bronze) and copper and nickel (melchior). Expanding the use of its anti-friction alloys containing 5-10% aluminum and 5-10% copper. Zinc sulfide activated with silver and aluminum have fluorescent properties and mixed with cadmium sulfide is used for making television tubes and displays (kinescope). Zinc parts obtained by smelting under pressure is widely used in aerospace, automotive, and others industries. Zinc is used in stabilizing devices that correct deviations of satellites in space and its compounds (zinc-silver oxide batteries) are a source of energy in spacecraft.

Anhydrous zinc chloride is used as dehydrating agent in reactions in organic chemistry, in the production of paints, viscose and as flux in hot dip galvanizing, tin plating and soldering.

Zinc antimonid used in devices for converting electrical energy into heat, spot cooling detectors and as a component of electronics and instrumentation in space ships.

Zinc selenide is used in the semiconductor and fluorescent technique for producing photoresistors, photoelements, photosensitive layers and transparent ceramic.

Zinc oxide is a raw material in the production of powder, glass, ceramics, matches, celluloid, paints, dental face cement, cosmetics, rubber.

Zinc salts are used in agriculture as fertilizers (sulphates, nitrates and chlorides) and polimicrofertilizers that enhance the growth of root system of plants and improve their protein metabolism, accelerate redox reactions, stimulate the synthesis of chloro-

phyll, vitamins C and vitamin P.

Zinc is used as a stimulator for fish roe and larvae of carp. Zinc compounds are widely applied in medical practice: zinc sulfate as antiseptic at conjunctivitis, laryngitis, urethritis and vaginitis; zinc oxide in the composition of ointments and pastes for external use.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Zinc is one of the most common anthropogenic pollutants. His technophylity (the ratio of annual production to the average content in the Earth's crust) is 10^{8-9}. Zinc dig from the earth in an amount exceeding five times the natural removal of the surface by the waters, as land (soil) annually adds 3.1 mln. tons of zinc.

The main source of environmental pollution are enterprises of non ferrous metallurgy. Composition of their waste is determined by the composition of processed ores, if are copper-zinc, per ton of copper accounts for two tons of zinc. From the air by precipitation annually on 1 km^2 area is allocated an average of 72 kg zinc.

Wastewater from metallurgical enterprises, concentrating factories, electroplating factories, chemical plants and enterprises for artificial silk, contain zinc compounds from 0,25 to 4,4 mg/dm^3 (such as zinc).

The soil is contaminated with zinc through atmospheric precipitation, in treatment with zinc phosphide against rodents, at fertilization with zinc fertilizers. Increased zinc content is found near TEP due to its high content in oil and coal.

The content of zinc aerosols in the air of the working environment in the metallurgical and electroplating workshops ranged from 0.3 to 5-7 mg/m^3.

Zinc refers to the elements tend to bioaccumulation. In grass the zinc content is up to 13 times more than in the soil. Lichens, which actively absorb atmospheric precipitation with dissolved therein elements, accumulate zinc 84 to 153 mg/kg dry matter and can serve as indicators of environmental pollution with zinc.

Under normal conditions (soils non contaminated with zinc) the intensity of absorption of zinc is high, soil-plant coefficient of zinc is from 3 to 30 (sometimes reaches 50 or more). High content of zinc in soil reduces this ratio, which proves the existence of a physiological barrier in plants of zinc absorption.

According ability to migrate aquatic zinc refers to easily removable elements. In neutral and acidic water easily migrates, in the alkali - form precipitates of hydroxides and salts. In minor contamination of the rivers, the most sensitive objects, convenient for biomonitoring are freshwater invertebrates. In significant pollution, to toxic levels, monitoring should include a hygienic analysis of water and bottom sediments.

METABOLISM AND MECHANISM OF ACTION

Zinc is an essential element. It is relatively non-toxic to a wide range of tolerance (from normal to toxic levels). Some authors consider that besides acute inhalation poisoning, occurring as so-called "Foundry fever" or chemical pneumonitis, and acute oral toxicity after ingestion of a high dose of zinc with function disorder of the gastrointestinal tract, chronic poisoning with zinc by the working or environment does not exist.

The main source of zinc for the body is food. The daily need of zinc reached 5 mg. In the adult body contains 2-3 g of zinc. The amount of zinc in various organs and tissues varies - about 3 mg% on average. The levels of zinc in the skin, hair and nails depends on nutritional status.

Absorption of zinc after ingestion also varies widely - from 5 to over 90%. It depends on the status of the body and the composition of food: amino acids in proteins bind zinc and amplify the oral absorption; calcium, phytates and fiber reducing them. In the process of distribution between organs and tissues, does not indicate a trend towards accumulation of zinc. Excreted primarily through the intestines. Relatively small amounts are excreted through urine. Substantial amount of zinc can be eliminated from the body through sweat. Under normal conditions (adults with daily average receiving of food), the half-life time of the whole body is about 1 year.

Information about zinc metabolism in the body is obtained by determining the concentrations in the plasma (serum), urine, hair and sweat.

PREVENTION

Main direction to improve working conditions in metallurgical plants is the improvement of processes and equipment, mechanization and automation of manufacturing operations. Moreover, the complex remedial measures in the production of zinc include: - complete hermetisation of equipment; - establishing an effective ventilation system; - mechanization of cleaning processes in scrubbers and electro filters; - use of vacuum pumps for submission of raw materials and others.

To protect the UAP recommended special respirator, including filtration and ion-exchange padding with an efficiency of 99% protection and operation time up to 72 hours.

Preliminary and periodic medical examinations are conducted with the participation of a therapist, ORL, ophthalmology and radiology. Recommended regular sanitation the oral cavity.

7.3 ALKALIS AND ACIDS

7.3.1. ALKALIS

GENERAL CHARACTERISTICS

HYGIENE STANDARDS

Air of the working environment

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Potassium hydroxide	2,0 mg/m^3	–	Caustic effect
2. Calcium hydroxide	5,0 mg/m^3	–	Irritation
3. Sodium hydroxide	2,0 mg/m^3	–	

Potassium base is white opaque highly hygroscopic substance. Outdoor passes in potassium carbonate (potash). Calcium hydroxide (slaked lime) is an aqueous solution of CaO (quicklime), which is a white amorphous powder with a melting point of 2580°C, a boiling point of 2850°C, density 3.16. And slaked quick and lime absorb carbon dioxide from the air. Sodium hydroxide is a white opaque solid, highly hygroscopic, having a melting point of 320°C, a boiling point of 1378°C, density 2.13. A strong base, in store outdoors gradually turns into sodium carbonate.

USE

Potassium hydroxide are used to produce liquid soaps, potassium salts, printing ink, in the textile industry and printing industry.

Sodium hydroxide is used in the manufacture of fibers, soaps, paper and textile industry, descaling and cleaning of oils and oil fractions.

The calcium base used in the production of cement, construction, metallurgy, food and leather industry, glass industry, agriculture and ecological objectives - wastewater treatment.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Alkaline aerosols are a common factor in the work environment given the widespread use of mainly potassium and sodium hydroxide in various sectors and industries. Basics itself have strong local caustic (burning) effect on skin and mucous membranes. Dissolved proteins and penetrate into the depth of the skin. After skin burn of base remain permanent scarring. In constant contact with hot alkaline solutions stratum corneum of the skin softens, swells and gradually falls of. This condition is known in the literature as "hands of laundress".

Especially dangerous is falling solution (drops, spray) of bases in the eye. Lesions rapidly spreading and not only affect the cornea but penetrate deeply into the tissues of the eye and can cause severe pathology including blindness.

The powder of calcium oxide acts on UAP not only as a mechanical irritant, causing discomfort and sneezing. On wet skin, especially the mucous membranes occurs and caustic action.

METABOLISM AND MECHANISM OF ACTION

Alkalis are irritating and caustic effect on the skin and mucous membranes. They gel tissue in contact, dissolving proteins which form albuminates. Penetrate deeply and cause painful and difficult healing burns.

These solutions operate depending on concentration and temperature.

Especially dangerous and very serious consequences are burns with bases at esophageal accidental engulfment and accidents.

PREVENTION

To prevent of basics is imperative to ensure workers with special work clothing, rubber gloves, boots and goggles or a transparent helmets. The use of hydrophobic ointments and creams to protect the exposed areas of skin.

7.3.2. ACIDS

Nitric acid - HNO_3

Orthophosphoric acid - H_3PO_4

Acetic acid - CH_3COOH

Picric acid - $C_6N_3O_6OH$

Sulfuric acid - H_2SO_4

TCA - CCl_3COOH

Hydrogen chloride and hydrochloric acid - HCl

GENERAL CHARACTERISTICS

Concentrated inorganic acids containing sulfuric acid are chemical mixtures carcinogenic to humans (Group 1).

Nitric acid is a colorless liquid with a boiling point of 83,4°C and a density of 1.502. Vapour of nitric acid were 2.2 times heavier than air. A strong acid and an oxidizing agent - oxidizes all metals except gold, platinum,

HYGIENE STANDARDS

Air of the working environment

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Nitric acid	5,0 mg/m^3	–	Irritation
2. Acetic acid	25,0 mg/m^3	37,0 mg/m^3	
3. Picric acid (2,4,6-Trinitrophenol)	0,1 mg/m^3	–	Allergen, irritation
4. Sulfuric acid	1,0 mg/m^3	–	Irritation
5. Trichloroacetic acid solution	7,0 mg/m^3	–	
6. Hydrogen chloride and hydrochloric acid	8,0 mg/m^3	15,0 mg/m^3	Irritation

Air settlements

Pollutants	Daily average MAC	Maximum single MAC
1. Nitric acid	0,15 mg/m^3	0,40 mg/m^3
2. Acetic acid	0,06 mg/m^3	0,20 mg/m^3
3. Sulfuric acid (in the molecule of the acid)	0,10 mg/m^3	0,30 mg/m^3
4. Sulfuric acid (a hydrogen ion)	0,002 mg/m^3	–
5. Hydrochloric acid (a molecule HCl)	0,20 mg/m^3	0,30 mg/m^3

rhodium and iridium. Straw, sawdust and other porous organic materials in contact with nitric acid and spontaneously in flame and emit nitrogen dioxide. Alcohol and turpentine in contact with nitric acid to detonate. Under the influence of light and heat yellowing as a result of formation of nitrogen dioxide. Dissolve in water in any proportion.

Acetic acid is a liquid having a boiling point 118.1°C and a density of 1.04 at 20°C. Mix with water in all proportions.

Picric acid is a solid - yellow crystals in the form of sheets or prisms with a strong bitter taste. Upon gentle heating to evaporate without being decomposed. Dissolved in water - 1.22% at 20°C. Very well dissolved in alcohol. Explodes at detonation, but much dangerous its salts, exploding on heating, friction or shock. Temperature of outbreak 300-310°C.

Sulfuric acid in its pure form is oleiferous liquid, transparent, colorless, slightly volatile, highly hygroscopic and odorless. Boiling point 330°C, density 1,834. At ambient temperature (about 18-20°C) no smoke, when heated to 50°C and more separated sulfur trioxide vapor that with humid air form aerosols - white haze. With the water is mixed in any proportion, releasing a considerable amount of heat. Concentrated sulfuric acid is a strong oxidizer. Technical sulfuric acid contains impurities of arsenic and in contact with metals form highly toxic arsenovodorod (arsine).

Trichloroacetic acid is a white or slightly yellowish, hygroscopic powder with a melting point of 59.2 ° C, boiling point 197.5 ° C and a density of 1.617 at 46 ° C. Dissolve well in water, ether and alcohol. The solution is a strong acid.

Hydrochloric acid is an aqueous solution of hydrogen chloride. At a concentration of 38% density is 1.19. Dissolve most metals except gold, silver, platinum, tantalum, niobium and the like. Technical hydrochloric acid contains impurities including arsenic.

Orthophosphoric acid is a colorless easy melting crystals with a melting point of 42,35 ° C and a density of 1,834 at 18 ° C. Well dissolved in water, with hot water mixed in all ratios.

USE

Nitric acid is used in the manufacture of fertilizers, explosives, film tape, rayon, nitrocellulose lacquers, nitrates, acids, aniline dyes; in printing; in non ferrous metallurgy, an oxidizer for rocket fuel.

Sulphuric acid is used to produce a variety of salts and acids, fertilizers, explosives, in organic synthesis for hydration, sulfuration, alkylation; in petrochemical, metal, textile and leather industries.

Hydrochloric acid is used for preparing chlorides of metals, organic products - chloropropane, dyes, alcohol, glucose, sucrose, gelatin and glues, for the tanning and coloring of leather, the saponification of fats, coloring fabrics, purification of metals, in electroplating and oil production.

Orthophosphoric acid is used in the production of phosphate fertilizers, salts of phosphorus, gental and special types of cement (for protection against ionizing radiation), to remove limescale, for anti-corrosion coatings, for impregnating fabrics with fire resistant mixtures, in the production of photographic film and photoreactive, in electroplating, and as a catalyst.

A large number of organic acids are used in the textile industry, production of synthetic rubber, indigo, plasticizers, drugs, pesticides and in food industry.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

It is very difficult to list all the professions from all sectors. Wherever used acid is a risk of acute poisoning with acid spill, and almost constant (chronic) acting concentrations typical for production.

All acids have a local effect on skin and mucous

membranes. The effects range from annoying to severely burning action, later development of difficult healing inflammatory lesions of the skin and mucous membranes.

METABOLISM AND MECHANISM OF ACTION

Due to its solubility in water and coming electrolytic dissociation organic and inorganic acids damage the skin and mucous membranes in direct contact. The dimensions and the extent of the damage depends on the acid concentration and the time of contact with the tissue. Inhalation of vapor mixtures of acids (acid mist) damage to the lining of the UAP depends on the properties of aerosols in the composition of fog.

Aerosols of sulfuric acid are more toxic of sulfur dioxide.

Nitric acid causes severe skin burns. Diseased tissue colored yellow because of upcoming xanthoproteinic reaction. The diluted nitric acid solution may cause eczema.

Under the influence of vapour on chromium, sulfuric and phosphoric acid occurs erosion of dental enamel. For prolonged chronic exposure are possible manifestations of general toxic action.

PREVENTION

Protect skin, eyes and mucous membranes of UAP through masks filter type, goggles, transparent shields or masks, special clothing of anti acid-treated polymer fabrics, gloves, aprons and boots made of acid-resistant materials.

Mechanization of operations transport, transfusion and charging with acids. Full hermetisation of the technological equipment.

For people in contact with acids required preliminary and periodical medical examinations involving ORL specialist, dermatologist and dentist.

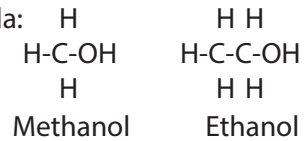
M. Koleva

7.4 ALCOHOL

METHYL AND ETHYL ALCOHOL

Empirical formula: CH_3OH (methanol);
C_2H_5OH (ethanol)

Structural formula:



Methanol Ethanol

HYGIENE STANDARDS

Air of the working environment: Methyl alcohol - mean - shift MAC - 50,0 mg/m^3; Ethyl alcohol - mean - shift MAC - 1000 mg/m^3.

Air settlements: Methanol - daily average MAC - 0,5 mg/m^3; maximum single MAC - 1,0 mg/m^3;

Ethanol - daily average MAC - 5,0 mg / m3, maximum single MAC - 5,0 mg/m^3.

GENERAL CHARACTERISTICS

Monovalent (monohydric) alcohols are hydrocarbons containing a hydroxyl group. They have a characteristic pungent odor. Mixed with water in any proportion (totally soluble in water). They are volatile. The lower limit of ignition of methanol in the mixture with air is 3.5%.

USE

Alcohols are used as solvents, paints and pesticides; varnishes and lacquers; in the pharmaceutical industry as extracting agent and as reagents in organic synthesis. One quarter of the produced methanol is used as solvent, the remainder is distributed mainly for dilution (denatured) ethanol and the production of formaldehyde. Much of the work ethyl alcohol used in the manufacture of alcoholic beverages.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Occupational exposure to methanol and ethanol is typical for operators and workers in the chemical industry, distillery, pharmaceuticals (mainly phytochemistry) and furniture industry.

METABOLISM AND MECHANISM OF ACTION

In production conditions the main road of penetration in the body is inhaled. Possible and absorption through the skin. The absorption is prolonged, and the accumulation is continuously throughout the period of exposure because of the high solubility of the vapour of methanol and ethanol in water. Tissue distribution is dependent on their water content.

The methanol is oxidized to formaldehyde and formic acid, which are highly toxic. The oxidation is

slower than that of ethanol. Connects with glucuronic acid.

Ethyl alcohol is oxidized to acetaldehyde and acetic acid.

The alcohols are eliminated from the body as CO_2 in the composition of exhaled air and as metabolic products in the urine composition (mainly formic acid in methanol).

PREVENTION

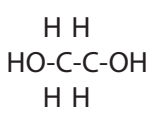
At high concentrations of methanol in the working environment is recommended to use a gas mask filter type. It is desirable in all technologies methanol can be replaced with ethanol or other suitable substituents. Along with raising health awareness and the information of workers about toxic properties of methanol, necessarily labeled containers containing methanol. Pipelines for methanol are indicated with signal paint and warnings. It is also necessary to ensure maximum hermetisation of technological equipment.

It is believed that acute inhalation poisoning with ethanol in production without further oral ingestion is no possible.

ETHYLENE GLYCOL (Glycols; Diols)

Empirical formula: $C_2H_4(OH)_2$

Structural formula:



HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 60 mg/m^3, maximum single - 125 mg/m^3.

GENERAL CHARACTERISTICS

Ethylene glycol (EG) is a colorless, nearly odorless liquid with a sweet taste. M.m. 62.07, a boiling point of 197,2°C, completely soluble in water, well was dissolved in ethyl alcohol, acetone, glycerin, lower in ether, chloroform and benzene. Almost incombustible, without corrosive activity and less volatile.

USE

Synthesized in 1859. Discovered as a substitute for glycerin, EG attracted the attention of military experts and its use increased sharply in some sectors of the defense industry in I-World War. It is widely used in the airline and road transport, and in various industries such as: basic component of antifreeze, brake fluids and anti-refrigerating liquids. Currently, 65% of the production of EG used for the preparation of antifreeze. Substitute for glycerin production of frost resistant dynamite, electrolyte in the production of capacitors and radiodetayli, a constituent of fibre glass building materials resin-based, solvent in the tobacco, textile, leather, pharmaceutical and cosmetics industry, raw material in the production of polyester fiber, lubricant in production of high density synthetic fibers, based on caproate, a plasticizer in the production of plastics and cellophane.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

EG does not create excessive concentrations in the working environment due to its low vapor pressure at ordinary temperature. Risk of acute and chronic poisoning inhaled gone. Barely penetrate the skin and poses no risk of dermal absorption in direct contact. EG represents a danger to the body when ingested per os. Endangered are large number of workers because of its wide use, but the most common acute fatal poisoning register with drivers and fitters. Takes as mistake, because it resembles alcoholic drink.

Concentrations in ambient air as a rule does not create, as has a short half-life and is not volatile.

MECHANISM OF ACTION AND METABOLISM

Oxidation of EG by alcohol dehydrogenase to glycoaldehyde is a critical step in the metabolism which leads to oxalic acid - cause of acute renal failure, metabolic acidosis and fatal outcome in most cases. About 60 cm^3 a lethal dose of man. Chief pathway leads to the formation of CO_2 by glycoaldehyde, glycolic acid and glyoxal. There is also so-called minor path, which leads to oxalic acid with limit level of excretion to 16% at low doses EG.

Under the same conditions the amount of CO_2 exhaled reaches 84%. Pure EG is much less toxic than the metabolites that are formed in the body. This fact has been used successfully to prevent the fatal outcome ingestion of large doses of EG. If blocking alcohol dehydrogenase by introducing another substrate (specifically - ethyl alcohol) elimination of EG unchanged in the urine increases and the patient overcomes renal failure and metabolic acidosis.

PREVENTION

In the presence of high concentrations in the air of the working environment is recommended to use a respirator or gas mask filter type.

If used as a solvent, a plasticizer, an electrolyte, especially under heat and the formation of aerosol - local aspiration ventilation.

If used as antifreeze and in the composition of other non-freezing liquids must be followed accepted rules for safe handling: informing workers for the safe transportation, labeling packages with the appropriate labels.

Workers in contact with ethylene glycol subject to periodic check-ups.

M. Koleva

7.5 AROMATIC HYDROCARBONS

Benzene

Alkylbenzenes: toluene, xylene, ethyl benzene, styrene and isopropylbenzene.

The aromatic hydrocarbons are compounds with one or more benzene rings. They include: Benzene and so-called alkylbenzenes, namely: Toluene (methylbenzene), xylene (o-, m- and p-isomers of dimethylbenzene), ethyl benzene, isopropylbenzene (cumene) and styrene (vinyl-benzene).

HYGIENE STANDARDS

Air of the working environment:

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Benzene	3,25 mg/m^3		Carcinogenicity to humans (group 1).
2. Toluene	150 mg/m^3	300 mg/m^3	
3. Xylene	221 mg/m^3	442 mg/m^3	Irritation
4. Ethylbenzene	435 mg/m^3	545 mg/m^3	
5. Isopropylbenzene (Cumene)	100 mg/m^3	250 mg/m^3	Skin-resorptive effect
6. Styrene	85 mg/m^3	215 mg/m^3	Possibly carcinogenic to humans (group 2B)

Air settlements:

Pollutants	Daily average MAC	Maximum single MAC
1. Benzene	0,1 mg/m^3	1,5 mg/m^3
2. Toluene	0,6 mg/m^3	0,6 mg/m^3
3. Xylene	0,2 mg/m^3	0,2 mg/m^3
4. Ethylbenzene	0,02 mg/m^3	0,02 mg/m
5. Isopropylbenzene	0,014 mg/m^3	0,014 mg/m^3
6. Styrene	0,003 mg/m^3	0,003 mg/m^3

Surface water:

Indicator	I category	II category	III category
1. Benzene	0,5 mg/dm^3	0,5 mg/dm^3	1,0 mg/dm^3
2. Styrene	0,1 mg/dm^3	0,2 mg/dm^3	0,5 mg/dm^3

GENERAL CHARACTERISTICS

Benzene is a colorless liquid with a specific smell. Mix without restrictions with non-polar solvents: hydrocarbons, turpentine, ethers; dissolved fat, rubber, resins and the like. Explosive concentrations of benzene vapors vary from 1.5 to 8%. In almost the same range (from 1.27 to 7%) are explosive concentrations of toluene in a mixture with air. The specific sweet smell is common organoleptic feature of most aromatic hydrocarbons. All of them are obtained directly or indirectly mostly of crude oil and less than coal tar.

USE

Despite the exceptional qualities of benzene use as solvent restricts and wherever technologically is possible, be replaced by less toxic solvents because of proven carcinogenic properties. Still applied in organic synthesis to obtain: phenol, styrene, cyclohexane, caprolactam, fibers, plastics, detergents and pharmaceuticals. It is also used for extraction of protein, skiming bone and solid waste. Relatively new field of application of benzene are laser technologies.

Toluene is used for the preparation of caprolactam and TNT (trotyl), in the production of dyes, pharmaceuticals, plastics, resins, varnishes and the like.

Air-gasoline and high-octane automobile gasoline contains aromatic hydrocarbons in limited quantity.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Benzene settled in air of the working environment in enterprises of oil, petrochemical and coke-chemical industry as a constant intermediate product. Aromatic hydrocarbons are volatile and create concentrations similar to hygiene standards in the air of the working environment, and in use of

products containing them. The number of persons exposed, significantly exceeds directly employed in various industries, because of the universal character of aromatic hydrocarbons such as solvents and theirs general use.

Source of water pollution with benzene and aromatic hydrocarbons are all industries that use these compounds, petroleum storage depots, refineries and petrochemical plants, and on navigable rivers - river transport.

Aromatic hydrocarbons (particularly benzene) weakly oxidized under the influence of biological processes in the water and have no impact on the oxygen content in it.

It was found that the underground and aboveground parts of higher plants (roots, shoots, leaves, fruits) have the ability to absorb and metabolize benzene.

METABOLISM AND MECHANISM OF ACTION

The main route of entering of benzene and toluene in the body is inhaled, in proven high respiratory absorption: 50-90% for benzene and 57-72% for toluene. Skin absorption of benzene vapor is negligible. Steam of toluene not penetrate the skin.

Benzene and toluene are distributed unevenly in the tissue, in the greatest amount is found in the adipose tissue, bone marrow and nervous tissue.

The major metabolite of benzene is phenol - 50-90% of the amount of benzene. With the exhaled air is eliminated from 3 to 45% of the total amount of benzene in the body, 89% of the total adsorbed amount of benzene is eliminated within 2-3 days. Exposition test for benzene in the air is content of phenol in urine.

The main quantity of toluene metabolized to benzoic acid (80-100%). Benzoic acid reacts with glycine to form hippuric acid. About 10-20% of the benzoic acid conjugated with glucuronic acid and sulfuric acid to form benzoilglyucoronic acid and sulfates.

With the exhaled air is eliminated from 8 to 31% of the received in body toluene. The remaining quantity is rapidly excreted in the urine (half-life period of 2-3 hours) as benzoic and hippuric acid.

PREVENTION

The main preventive measure is the ban benzene to be included in the formulation of solvent.

Healing working conditions is achieved through maximum hermetisation of process equipment.

Preliminary and periodic medical examinations in contact with benzene are required. Pregnant women vocational rehabilitation immediately. Avoid contact of adolescents (before age 18) with benzene.

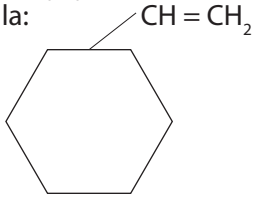
Appropriate organization of professional protective nutrition (diet D), giving extra vitamins: vitamin. C - 100 mg, vit. B_{12} - 3 μg, folic acid - 300 μg, and the alternation of mineral water 1 liter two months of Mihalkovo, Shipkovo 1 month of Gorna Banya, Targovishte, Ivanyane, Belovo, Varshetz or Varna.

STYREN[1]

Possibly carcinogenic to humans (group 2B)

Empirical formula: C_8H_8

Structural formula:



GENERAL CHARACTERISTICS

Styrene (vinyl-benzene) is a colorless liquid with a sweet (at low concentrations) and unpleasant (at high concentrations) smell. Melting point 30.6°C, the boiling temperature of 145.2°C, insoluble in water and is soluble in ethanol, acetone, benzene, ether. Reacts violently with strong oxidants. Highly flammable. Flash temperature 30°C. Temperature of self-ignition 530°C. Generate electrostatic charges when pouring, shaking and blows. Above 65°C spontaneously polymerize with the danger of fire and explosion. With air form explosive mixtures.

USE

It is widely used in industry. Significant quantities are consumed in polymerization, and for the preparation of polystyrene, styrene-acrylonitrile resin, styrene-butadiene rubber, polyester resins. Polystyrene and its co-polymer akrilnitrilbutadien styrene are used for the production of packaging for food products.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Risk of occupational exposure exist in the production of styrene and polystyrene and use dissolved in styrene unsaturated polyester resins. Exposure varies greatly and depends on the task being performed. High concentrations in the air of the working environment is created for cleaning and maintenance of the facilities.

The biggest polluter of the environment with styrene is the petrochemical industry. Significant amounts are released in breach of distillation columns, leakage and spillage from containers during storage and transportation. Emission sources are polymerization processes, the burning of polymers, exhaust gas mixtures from motor vehicles. Tobacco smoke contains 18-48 micrograms styrene/cigarette.

The use of styrene as a solvent in life is a potential source of emissions.

[1] A. Mihaylova

METABOLISM AND MECHANISM OF ACTION

It has been found that 50-90% of the inhaled styrene metabolized. The biotransformation is carried out by oxidation of the double bond in the cytochrome P-450 and P-448. The formed styrene oxides (styrene-7-8-oxide and styrene-3-4-oxide-2) included in the coupling reaction with glutathione and using the transferases form conjugates. The major metabolites in humans are mandelic acid and fenilglioxsalic and in smaller quantities hippuric and 4-vinylphenol. Styrene oxides have short half-life. Studies in recent years show that the toxicity of styrene is mainly related to the effects of styrene oxides.

Styrene has a narcotic effect. There is a strong irritant effect on skin and mucous membranes. Damaging haemopoieses and the liver. Deposits in adipose tissue. Violates the permeability of the blood brain barrier.

Expressed narcotic phenomena occur less frequently, as strong irritation of styrene forcing workers to evacuate the workplace. Irritative effect occurs in concentrations over 408 mg/m^3.

PREVENTION

It is not allowed women in fertile age have contact with styrene. Performed compulsory check-ups with a focus on hematopoiesis, liver, respiratory and nervous systems. To pay attention to personal hygiene of workers. At elevated concentrations in the air of the working environment, accidents and fires, workers to provide: clothing from coated fabrics, airtight goggles or face shield, mask filter, latex gloves and footwear - boots with leather face and a rubber sole.

A. Mihaylova

7.6 GASES

Gases, vapors and smoke may cause lung damage in five main mechanisms:

1. Replacement of oxygen from inhaled air with subsequent asphyxia

2. Absorption into the systemic circulation with obstruction of transport and utilization of oxygen (chemical asphyxia)

3. Exercise of systemic toxicity without affecting the supply of oxygen

4. Direct irritative effects or damage to the respiratory tract, such as the site of the damage depends on the solubility of gases. Gases with the highest solubility are absorbed by the mucous membranes of the nose, upper respiratory tract and conjunctiva (eg. Ammonia), and the more sparingly soluble exert harmful effects especially on the lower respiratory tract (eg. Nitric oxides)

5. Inducing allergic effects directly damaging the lung or disrupting gas exchange **(Table. 1)**.

NITROGEN OXIDES

NO
NO_2
N_2O_4
N_2O_3
N_2O_5

HYGIENE STANDARDS

Air working environment

Nitrogen dioxide -
mean-shift MAC - 4.0 mg/m^3
Nitrogen oxide -
mean-shift MAC - 20.0 mg/m^3

Atmospheric air of settlements

Nitrogen dioxide: Annual average
MAC - 0.05 mg/m^3
Daily average MAC - 0.10 mg/m^3
Maximum single MAC - 0.20 mg/m^3
Nitric oxide:
Average annual MAC - 0.06 mg/m^3
Maximum single MAC - 0.60 mg/m^3

GENERAL CHARACTERISTICS

Nitrogen oxides are intermittent gas mixture that includes nitric oxide (NO), nitrogen dioxide (NO_2), dinitrogen tetroxide (N_2O_4), dinitrogen trioxide (N_2O_3) and dinitrogen pentoxide) N_2O_5), and vapor of nitrous, hyponitrous and nitric acid, that of air are reduced by the organic substances and pass into nitrogen oxides. Relatively constant components of the mixture are nitrogen oxide, nitrogen dioxide, and dinitrogen tetroxide.

In production conditions the most common is nitrogen dioxide, which is a reddish-brown gas with a

Tab. 1. Mechanisms of toxic effect on lung from gases (by Charles E.Becker, 1997)

Substance (gas)	Source
Simple asphyxiants	
Carbon dioxide	Fires
Methane	Underground mining of ore and coal
Nitrogen	Work for hyperbaric conditions
Chemical asphyxiants	
Carbon monoxide	Fires
Cyanide	Gold Mining; Galvanization; Fires
Hydrogen sulfide	Oil refining; Decay of organic matter
Systemic toxicants	
Arsin gas	Microelectronics
Phosphine	Microelectronics
Paraquat	Agriculture
Metal aerosols pyrogenic action (causing "Fondry fever")	Melting of metals; Production of alloys
Irritants direct irritation	
Chlorine	Production of bases, disinfectants and detergents
Ammonia	Production of mineral fertilizers; refrigeration manufacture
Nitrogen oxides	Melting of metals; Exhaust gases
Sulphur dioxide	Production of paper; Refining
Hydrogen fluoride	Microelectronics
Allergens	
Isocyanates	Production of polyurethane
Aerosols of platinum compounds	Production of catalysts; electroplating
Aerosols of solders and flux	Soldering

strong irritating odor and is heavier than air. Temperature (T°) of melting 11.2°C, T ° of boiling point 20.7°C.

In case of contact with metals, chlorinated hydrocarbons and ammonia cause explosions. Does not burn, but it increases the combustibility of other substances. It manifests as a strong oxidizing agent. Especially dangerous is at temperatures above 21°C.

Nitric oxide is a colorless volatile gas with a sharp sweet smell. Melting point 163.7°C, T° of boiling 151, solubility in water 73.40 cm/dm³. At high concentrations stained brown in the air. With water forms corrosive vapours. In contact with ozone and chlorine compounds is explosive. It is explosive when heated, and fire - by improper storage.

USE

They are used in the production of celluloid, photoplates, sugar refining. Nitrogen dioxide is used as a nitrating agent and a catalyst in the production of sulfuric acid.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Nitrogen oxides are found in the production of nitric, sulfuric, chromic and picric acid, nitrates, nitrogen fertilizers, explosives (nitrocellulose, nitrotoluene, nitroglycerin), toluene. Are formed in oxygen- and electric welding, when blasting in mines and others. Released in large quantities when pouring nitric acid and in contact with metals and organic substances. High concentrations of the order of 300-3800 mg/m³ create bins in the fermentation of the cereal seeds.

Yearly over 53 mln. tons of nitrogen oxides released into the atmosphere. Sources of emissions are vehicles (internal combustion engines), the use of solid and liquid fuels for heating in TEPP and household, production of explosives and nitric acid, as well as tobacco smoke. The content of nitrogen monoxide in tobacco smoke ranges from 98-135 mg/m³, and nitrogen dioxide - from 150-226 mg/m³.

METABOLISM AND MECHANISM OF ACTION

Nitrogen dioxide metabolized to nitrites and nitrates, which are excreted in the urine.

Nitrogen oxides have a local effect - irritation of the mucous membranes of the respiratory tract and eyes and general resorbtive, predominantly affecting the nervous system and blood. They are methemoglobin generating, consequently arise hypoxia and anoxaemia. When connecting 70% of hemoglobin into methemoglobin death occurs. Exhibit and vasodilatory activity (nitrite effect).

Their irritative action provokes the release of large amounts of histamine, which favors the development of pulmonary edema.

PREVENTION

Total technical measures include automation, hermetisation and effective ventilation. Workers must be provided clothing from coated fabrics, latex gloves and rubber/PVC boots, while accidents, ex-

plosions and massive chemical contamination - gas masks with filter for acid gases.

Preliminary and periodic medical examinations with focus on blood, respiratory and nervous systems.

AMMONIA (NH_3)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 18.0 mg/m^3.

Atmospheric air of settlements:

Daily average MAC - 0.04 mg/m^3,

Maximum single MAC - 0.20 mg/m^3.

GENERAL CHARACTERISTICS

Ammonia (ammonium anhydrite, ammonia aqua) is a colorless, volatile gas with a strong irritating odor. Melting point 77.75°C, Boiling T° 33.35°C, a density of 0.771 g/l (0°C) and 0.59 g/l (25°C). Soluble in water and organic solvents. The aqueous solution is highly alkaline and acids react violently. Causing corrosion of metals. With air form explosive mixtures.

USE

It is widely used in the production of nitric acid, nitrogen fertilizers, urea, caustic soda, cleaners for the household. Feedstock for obtaining explosives and artificial fibers. It is used in metallurgy, rubber, paper, textile, leather and food industries, as well as for purification of water.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Increased emissions are registered in the synthesis of ammonia, coking and dry distillation of coal, the production of fertilizers and explosives.

In the air is released in the decomposition of organic substances from animal excrement, canals and wastewater, fertilization of soil and volcanic eruptions. Large quantities are released during accidents in production and transportation, as well as fires.

Opportunities for contamination of surface water exist in the use of fertilizer and manure in areas with a strong livestock, the decomposition of organic materials and natural accumulation of air.

METABOLISM AND MECHANISM OF ACTION

Ammonia metabolized to glutamine and urea in the liver. The released nitrogen of glutamine is involved in protein synthesis. It is believed that glutamine is the first metabolic product.

There are irritating and corrosive to the eyes, skin and upper respiratory tract. It damages the cell directly, as manifested alkaline corrosive and causes burns on all mucous membranes.

PREVENTION

Includes hermetisation and automation of manufacturing processes, replacing ammonia with other compounds (in refrigeration production), use of personal protective equipment.

In cesspool cleaning (leather industry), tanks and more, workers must use clothing from coated fabrics, gas masks filter ammonia, long latex gloves and rubber or PVC boots.

In preliminary and preventive medical examinations focus be in terms of the respiratory system, nervous system, skin and blood.

ARSINE (AsH_3)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 0.05 mg/m^3.

Atmospheric air of settlements:

Daily average MAC - 0.002 mg/m^3.

GENERAL CHARACTERISTICS

Arsine, arsenic trihydride is a colorless gas with a characteristic odor, heavier than air. Melting point 113.5 ° C, T ° Boiling - 62.5 ° C. When light, moisture and high temperatures (300 ° C) decomposes. By reacting with the chlorides and nitric acid can cause explosions. With air also form explosive mixtures. Combustion releases arsenic trioxide.

USE

Used for preparation of arsenic as an absorbent in the production of semiconductors.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Arsine is a factor in the work environment in the production of semiconductor chips in microelectronics. Small amounts of gas are released from the treatment with acids arsenic contented rocks and burning of fossil fuels. Risk of exposure exists in the processes of electro plating and engraving. High concentrations are created when working in confined spaces - cleaning of tanks, reservoirs, vats and accidental release of stored arsine.

METABOLISM AND MECHANISM OF ACTION

In the biotransformation of arsine form elemental arsenic or arsenic dihidrid. Trivalent arsenic at chronic exposure may be a carcinogen.

Arsine was dissolved in plasma. In red blood cells forms insoluble complexes. Coming hemolysis is multifactorial. Inhibits catalase and blocks the sulfhydryl groups of enzymes in erythrocytes. Provoke oxidative stress similar to that observed in G6PhDh deficiency. Developing renal impairment may be due to direct involvement of the renal tubules, secondary hypoxia reaching to anemia or precipitation of hemoglobin-haptoglobinic complex in tubular lumen.

PREVENTION

In gassed premises includes individual protection clothing from coated fabrics, fuel gas masks

with oxygen bottles, gloves (rubber) and boots (rubber or PVC). Preliminary and periodic medical examinations to be oriented to the nervous and digestive systems, liver, skin and blood. Recommended diets rich in protein and lipotropic factors with restriction of fats and increase the content of thiamine, refreshing drinks and more fluids.

CARBON MONOXIDE (CO)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 40,0 mg/m^3

Short-term maximum MAC - 200,0 mg/m^3

Atmospheric air of settlements:

Daily average MAC - 10,0 mg/m^3

Maximum single MAC - 6.0 mg/m^3

GENERAL CHARACTERISTICS

It is a gas, colorless and odorless, lighter than air. Burns with a blue flame. and is oxidized to carbon dioxide. Ignition temperature 610°C. In contact with strong oxidizers risk of fires and explosions. With air form explosive mixtures.

USE

Carbon monoxide is a feedstock for many organic syntheses. It is used for producing metal from them oxides, carbonyl chloride, carbonyl sulfide, aromatic aldehydes, formamide, formic acid, hexahydroxibenzen, methyl alcohol, synthetic gasoline, and the like.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Carbon monoxide is the most common industrial gas. Formed in all processes where there are conditions of incomplete combustion of substances containing carbon.

As a result of human activity into the atmosphere annually spend 350-600 10^{6}t CO. Domestic sources of CO are incomplete combustion of fuel, failures in pipelines and gas equipment, fires and smoke. Carbon monoxide is a component of various gas mixtures (gases) as a blast furnace, coke, water, gas generators, explosive and others. The concentration of CO under ground after blowing reach 350 mg/m^3.

In the chemical industry emitters of CO are the production of ammonia, catalytic cracking, formalin, the synthesis of hydrocarbons, phosgene, methyl alcohol, methane, formic acid. CO is formed in the production and processing of synthetic fibers. Metallurgical enterprises ejected into the atmosphere daily 320-400 t CO.

More than half of CO entering the atmosphere separated from road transport. CO content of the unburnt gases emitted from the engines is about 12%, taking into account that their composition includes about 200 chemicals. In combination with these CO exhibits a pronounced toxic effect as compared to the isolated action.

METABOLISM AND MECHANISM OF ACTION

In the body, CO is associated with compounds containing in the molecule a divalent iron (myoglobin, cytochromperoxidase, cytochromoxidase, catalase) and mainly by hemoglobin. Separated in the urine as a complex compound with iron.

The affinity of hemoglobin to CO is 240 times higher than to oxygen. Linking CO with hemoglobin, formation carboxihemoglobin and lowering the oxygen capacity of the blood is the main mechanism of action of the noxa. The formation rate of carboxyhemoglobin depends on the concentration and duration of exposure, locomotor activity (which increases the amount of air inhaled per unit time) and the ambient temperature, as well as the health status of the exposures. The formation of carboxyhemoglobin is a reversible process, but the time for half-elimination (T1/2) is sufficiently long - from 2 to 6.5 hours and depends on the initial levels of COHb. This may lead to accumulation of COHb and increase its content in the blood, when exposed to relatively low concentrations of CO.

The carbon monoxide blocks cellular enzymes, and it causes tissue hypoxia. Hypoxemia and hypoxia determine violations in metabolic processes, such as hyperglycemia, glucosuria, accumulation of lactic acid, lowering the alkaline reserve, disruption of protein synthesis, development of acidosis.

PREVENTION

Special attention should be paid to ventilation, hermetisation and personal protective equipment. Fire and explosions to provide protective clothing, complete with front apron, gas masks and filter, cold-protective gloves and shoes. Medical surveillance of workers to be targeted at the nervous system and blood. To organize periodic monitoring of the content of carboxyhemoglobin.

HYDROGEN SULFIDE (H_2S)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 14 mg/m^3,

Short-term maximum MAC - 21 mg/m^3

Atmospheric air of settlements:

Daily average MAC - 0.003 mg/m^3,

Maximum single MAC - 0.005 mg/m^3.

GENERAL CHARACTERISTICS

Hydrogen sulfide (channel gas) is a colorless gas with a characteristic odor, melting point - 122°C, boiling temperature - 46.3°C, a density of 1.26 (20°C), solubility in water 0.26/100 g (0 ° C), vapor pressure 127.3

mm Hg (0 ° C) and 297.5 mm Hg (20 ° C). Reacts violently with strong oxidizing agents (risk of fire and explosions). With air form explosive mixtures. Decomposes during combustion to form sulfur dioxide.

USE

Hydrogen sulfide was used to produce sulfur and sulfuric acids, thiol compounds, sulfides, as a reducing agent in analytical chemistry. It is used in the manufacture of gunpowder, matches, in leather, sugar, paper and textile industries.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Large quantities of hydrogen sulphide are released during the extraction and exploration of natural gas and crude oil. The sulfur content of crude oil is 0-5%, in the earth gas reaches 42%, in coal - 80 g/kg. Sources hydrogen sulfide are and geothermal springs. High concentrations were measured in plants using geothermal waters.

Hydrogen sulfide is removed by some industrial processes, as a result of the reaction of elemental sulfur or sulfur containing compounds with organic substances at high temperatures.

Hydrogen sulfide is an intermediate product in the production of carbon-disulphide, artificial silk, leather tanning, sugar refining, production of coke of sulfur-containing coal and others. Separated into septic tanks, wells and canals in the anaerobic decomposition of organic matter.

METABOLISM AND MECHANISM OF ACTION

Hydrogen sulfide belongs to irritating gases. Odor is felt at concentrations of 1-2 mg/m^3, and irritation has at concentrations above 6 mg/m^3. Metabolized by oxidation to thiosulphate and sulfates. Like cyanide inhibits cytochrome -C-oxidase and other enzymes containing heavy metals in a molecule. Causes intracellular hypoxia and asphyxia. Especially sensitive organ for acute effects are brain - causes coma and death. In chronic effects are typical eye (conjunctivitis and keratitis) and lung (chronic bronchitis) disabilities.

PREVENTION

Systematic control of concentrations of hydrogen sulfide in the air of the working environment (getting used to the smell occurs rapidly). It is recommended that periodic changing of jobs by those who are not contaminated with hydrogen sulphide. Use personal protective equipment: clothing made of coated acid resistant fabrics, breathing mask, rubber gloves and boots. Preliminary and periodic medical examinations to be oriented to the nervous, respiratory and cardiovascular systems, and eyes. Dietary modes include lipotropic (methionine, lecithin, etc.) and biologically active substances, and vitamins E, B_6 and nicotinic acid (PP).

SULFUROXIDES (SO_2) (SO_3)

HYGIENE STANDARDS

Air of the working environment:

Sulfur dioxide mean-shift MAC - 5.0 mg/m^3,
Maximum Short MAC - 10.0 mg/m^3.

Atmospheric air of settlements:

Sulphur dioxide
Average hourly rate - 0.350 mg/m^3,
Average daily rate - 0.125 mg/m^3,
Standard to protect natural ecosystems - 0.020 mg/m^3.

GENERAL CHARACTERISTICS

Sulphur dioxide (SO_2) - a colorless gas with a characteristic odor, while in cooling - a colorless liquid. Melting point - 75.5 ° C, boiling temperature - 10.1 ° C, solubility in water 22.8 g/100 g (0 ° C). Heavier than air. With water forms sulfurous acid, in the presence of strong oxidizing sulfur passes into sulfur trioxide, respectively in sulfuric acid. When moisture is highly corrosive.

Sulfur trioxide (SO_3) - separated mainly by vapour from fuming sulfuric acid. In humid environments quickly becoming aerosols of sulfuric acid.

USE

The sulfur dioxide is a feedstock for the preparation of sulfuric acid. Used in the vulcanization of rubber, production of cellulose and ultramarine, bleaching of fabrics (wool and silk) and cellulose, disinfection of premises, preservative food.

The sulfur trioxide is used in the manufacture of sulfuric acid, organic and inorganic substances, batteries, cleaning of metals.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Sulfur dioxide in combination with SO_3 is one of the most widespread pollutants in the working environment in metallurgy (processing of zinc, copper, lead and others ores), in obtaining sulfuric acid, burning liquid and solid (sulfur-containing) fuels in thermal power plants, foundries and forge shops, boiler rooms, production of superphosphates.

High concentrations of sulfur oxides are created in the mines with blasting, the individual amounts depend on the used explosives and mining development.

Sources of emissions of sulfur oxides are:

- liquid and solid fuels used in industry and households;
- Industrial plants;
- Motor vehicles.

METABOLISM AND MECHANISM OF ACTION

The biotransformation of sulfur dioxide occurs with the formation of sulfuric acid that was separated as sulfates. Part of the sulfur metabolites bind to

form S-cysteine, S-sulfoglutation and S-sulfonates. In metabolism may include L-acetylcystein group and to form alkyl- and aryl-S - N-acetylcistein derivatives - mercapturic acids and their derivatives.

Sulphur dioxide causes local damage with irritant and corrosive nature. Facilitates the formation of methemoglobin. Blocking thiol enzymes and potentiate hypoxia.

Sulfur trioxide causes severe burns to the skin.

PREVENTION

Compulsory technical measures are the introduction of effective ventilation and hermetisation of the production process. For reduction of risk prior to detonating the mines must be carried out spraying with water downhole, as well as the removal of the excavated ore, filling the blast holes with wet clay in order to reduce the risk of incomplete combustion, the sampling air for concentrations of CO and NOx no earlier than 15 minutes and no later than 30 minutes after the bombing.

Workers to be provided with personal protective equipment, such as at high risk (explosions, massive chemical pollution) to use the clothing of acid resistant, coated fabric, mask with filter for acid gases, rubber gloves and acid resistant shoes. Preliminary and periodic medical examinations to be oriented to the respiratory system and skin.

HYDROGEN CYANIDE (HCN)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 1.0 mg/m^3,

Maximum short MAC - 5.0 mg/m^3.

Atmospheric air of settlements:

Daily average MAC - 0.01 mg /m^3,

Maximum single MAC - 0.01 mg/m^3.

USE

It is used for synthesis of rubber, synthetic fibers, methylmethacrylate, sodium cyanide, ferrocyanide, for the preparation of plastics, plexiglass, pharmaceuticals and the like. It is used as an insecticide, rodenticide and fumigant.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

In the air of the working environment is released during the production of hydrogen cyanide, benzene, toluene, xylene, thiocyanate, cyanides, in electroplating processes, heating the polymer compositions.

The biggest emitters are producing methyl methacrylate, acrylonitrile and hydrogen cyanide. Present in wastewater from ore-dressing factories, mines, metal plating shops, metallurgical and metalworking plants, gas generators stations, coke-chemical plants and others. The content of hydrogen cyanide in the smoke is 0.002 g/10 g of tobacco.

METABOLISM AND MECHANISM OF ACTION

Hydrogen cyanide (cyanides) is one of the fastest acting known fatal poisons. Cyanides is not essential to the human body, but their presence in food, air, water, the workplace makes them significant, not only at high concentrations, especially in smokers **(Table. 2)**.

It is assumed that in the body is transformed to low toxic thiocyanates (rhodanides), released by the kidneys.

Cytochrom-C-oxidase inhibited by the mitochondrial respiratory chain. Disturbing oxidative processes in the cell, resulting in severe hypoxia develop.

Cytochrom-C-oxidase in the brain is the most sensitive to cyanide, which determines the dysfunction of the central nervous system for major in acute

Tab. 2. Content of thiocyanate and cyanide in the blood of smokers and nonsmokers (by Charles E. Becker, 1997)

Substance / Group	Level	Quantity in µg/ml	Type sample
Cyanide			
No smokers	Average value	0.02	Whole blood
Smokers	Average value	0.04	Whole blood
Acute poisoning		>0.1	Whole blood
Fatal poisoning		>1.0	Whole blood
Thiocyanate			
No smokers		1-4	Plasma
Smokers		3-12	Plasma

GENERAL CHARACTERISTICS

Hydrogen cyanide (formonitril, Prussian acid, cyanic acid) is a colorless gas or liquid with a characteristic odor. The gas is lighter than air. Polymerize in the presence of water as emits heat and increases the pressure (risk of fires and explosions). The fluid is combustible. Ignition temperature 538°C. With air form explosive mixtures.

lethal effects. Chronic effects of hydrogen cyanide in occupational exposure are controversial (smokers, receipt of cyanides by food). Described are: inhibition of thyroid function (hypothyroidism), as a result formed rhodanides; atrophy of the optic nerve; astheno-vegetative syndrome; changes in EKG and clinical laboratory blood values (reduced blood sugar, increased lactate and Hb), skin lesions in contact

with sodium or potassium cyanide. Are possible birth defects (especially in smokers) - low weight in newborns.

PREVENTION

Hermetisation of process.

Workers use special work clothes from coated fabrics, dust mask, gloves and boots. Disposal of waste and cleaning of polluted areas to use breathing apparatus with oxygen bottle. Periodic medical examinations (conducted annually) be oriented to the nervous system, liver and skin. It is recommended that a diet rich in lipotropic (methionine, lecithin etc.) and biologically active substances, vitamins E, B_6, B_1 and C.

CHLORINE (Cl_2)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 3.0 mg/m^3.

Atmospheric air of settlements:

Daily average MAC - 0.03 mg/m^3,

Maximum single MAC - 0.1 mg/m^3.

Drinking water: 0.3-0.4 mg/dm^3 residual chlorine in a 30-minute contact with water.

GENERAL CHARACTERISTICS

Chlorine is a yellow-green gas with a pungent odor, pressure liquefies. Melting point - 101.3°C, boiling temperature - 34.05°C. Heavier than air. Dissolve in water and organic solvents. Reacts violently with organic compounds and metal dust it can cause fires and explosions. Form hydrogen chloride on contact with air. Corrodes plastic, rubber, textiles and metals in the presence of water.

USE

It is used for the preparation of 1,2-dichloroethane, vinyl chloride, hydrochloric acid, chloriated lime, chloride, pesticides, plastics and bleaching agents. It is used in metallurgy, paper, textile, chemical- pharmaceutical and rubber industries, for the disinfection of drinking water and disposal of wastewater.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Risks from exposure to chlorine exists in the production of 1,2-dichloroethane and vinyl chloride. Chlorine is transported primarily in pipelines and tanks. Solid contaminants are possible breakout in the pipelines. Known quantities are released during loading and unloading of tanks. Emission source are and the process of electrolysis of chlorine compounds.

METABOLISM AND MECHANISM OF ACTION

Chlorine occurs irritating and corrosive action.

Upon contact with the mucous membranes form hydrogen chloride and with tissues - hydrochloric acid.

In a massive emergency (accidents, explosions) causes chemical burns to the lungs, blocking the respiratory center, causing reflex death.

Chlorine molecule is a strong oxidizing agent and reacts with many functional groups in the cell.

In chronic exposure are described damage to the respiratory system (chronic obstructive and restrictive bronchitis); skin; ocular mucosa and cornea; rhinitis and frequent development of anosmia.

PREVENTION

Production processes to be automated and used hermetically equipment. To exercise constant control of concentrations of chlorine in the air of the working environment. Workers to provide personal protective equipment. At elevated risk of using acid-resistant, fire resistant clothing, boots of PVC or rubber, latex gloves, respirator with oxygen bottle or an entire gas masks with filter for acid gases. Medical surveillance of workers to be targeted at the respiratory system, skin, eyes and blood. Prophylactic nutrition include lipotropic and biologically active substances, with the addition of vitamins (E, B_1 and C), refreshing drinks and more fluids.

HYDROGEN FLUORIDE (HF)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 2.5 mg/m^3

Atmospheric air of settlements:

Daily average MAC - 0,001 mg/m^3,

Maximum single MAC - 0.01 mg/m^3.

GENERAL CHARACTERISTICS

Hydrogen fluoride is a colorless gas. Melting point - 87,2 ° C, T ° boiling 19,9 ° C, density - 0.9885 (13 ° C). The aqueous solution is a strong acid. Reacts with metals to form hydrogen in the presence of water or moisture. In contact with air are obtained corrosive fumes, heavier than air. Destroys the glass.

USE

It is used in the production of aluminum, superphosphate chlorofluorocarbons hydrocarbons, lubricants, hydrogen peroxide, heat-resistant plastics, insecticides and fungicides; as the solvent of the alcohols and aldehydes, a catalyst in polymerization processes and alkylation; in engraving and polishing of glass.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Professional risk exists in the production of aluminum and phosphate fertilizer, production and use of pesticides, micro - silicon chips, as well as engraving and polishing of glass. At high levels of exposure are monitored by local disabled of mucous membrane irritation to skin burns.

Emission sources are aluminum and superphosphate production. Natural source of fluoride are volcanoes.

METABOLISM AND MECHANISM OF ACTION

Hydrogen fluoride is irritating and corrosive effect on the skin and mucous membranes.

It occurs as a direct cellular poison. Violates calcium metabolism and enzymatic mechanisms. With calcium forms insoluble precipitates and lowers its plasma level.

In chronic exposure occur trophic disorders of the mucous membranes - inflammation of the nasal mucosa, bleeding, perforation of septum nasi, anosmia; laryngitis, bronchitis, pneumonia; conjunctivitis and keratitis. Disabilities: tooth enamel - tan pigmentation, caries; digestive system - frequent gastritis; skin - dermatitis. Deposite fluoride in bones and teeth, which is released slowly by the kidneys.

PREVENTION

Recommended replacement of HF with less toxic compounds, its use in closed systems in effective ventilation. Workers must be provided special work clothes fabric of neoprene and gloves at increased risk (accidents, explosions, massive pollution) - mask brand B and goggles from organic glass. Preventive medical examinations to be oriented to the respiratory system, eyes and skin. In works related to the separation of hydrogen fluoride vapor women are not allowed, regardless of their age.

Recommended enrichment of dietary regimen of vitamins C, A and D and involving calcium supplements.

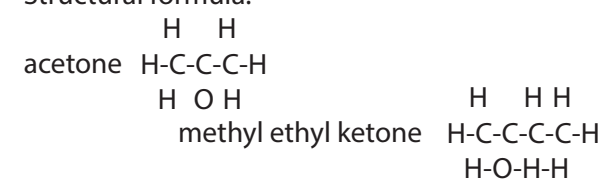
M.Koleva

7.7 KETONES

ACETONE AND METHYLETHYLKETONE:

Empirical formula: acetone - CH_3COCH_3,
methylethylketone - $CH_3COCH_2CH_3$

Structural formula:



HYGIENE STANDARDS

Air of the working environment:

Acetone: Mean-shift MAC - 600 mg/m^3,
Maximum single MAC - 1400 mg/m^3.

Methyl ethyl ketone:

Mean-shift MAC - 590 mg/m^3, maximum single MAC - 885 mg/m^3, irritating effect.

Air settlements:

Daily average MAC - 0,35 mg/m^3,
Maximum single MAC - 0,35 mg/m^3.

GENERAL CHARACTERISTICS

Colorless liquids with a distinctive odor, highly volatile, soluble in alcohol and other solvents. The acetone was mixed with water. Methyl-ethylketone with air forms explosive mixtures in concentrations from 1.97 to 10.2 volume %.

USE

It is used as a solvent of nitrocellulose and cellulose acetate synthetic, rubber, rubber, resins; denaturing of alcohol; as raw material for the manufacture of paints, varnishes, acetate rayon, photographic film and celluloid.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

High volatility determines the presence of acetone in the working environment whenever it is used. Concentrations ranged from 200-400 mg/m^3 to tens of thousands mg/m^3.

Acetone is a natural metabolite of man. The content in the blood normally around 0,7-0,8 mg%, and urine - 20-30 mg/24 h. The content of acetone in the exhaled air is about 0,2-0,3 mg/m^3. Under normal conditions, a person exhales 16,1 ± 1,2 mg of acetone for 24 hours. Therefore, acetone is detected in the air of hermetic chambers - the concentration of acetone in the air of the spacecraft "Soyuse-22" was 5,2 to 7,8 mg/m^3, and the orbital station "Skylab-4" - of 6,5 to 18,7 mg/m^3.

In the air, at 100, 300, 500 and 1000 m away from the plant for acetone are found excessive amounts. In air samples from residential premises in 7 of 10 samples acetone is not found, and the remaining 3 maximum concentration is 0,13 mg/m^3.

In the industrial wastewater from acetone production is evidenced in the amount of up to 16 mg/dm^3.

METABOLISM AND MECHANISM OF ACTION

In production conditions ketones (mainly acetone) penetrate primarily by the inhalation route. There is a possibility of penetration of liquid aerosol

through the lining of the gastrointestinal tract and through the skin. Through analysis of the acetone in the exhale air it has been shown that liquid acetone permeates through the skin in the same amount as well as vapor at a concentration of 5000 mg/m^3. Isolated skin has been found that the rate of permeation through the skin of liquid acetone was 0,002 mg/cm^2/h, and a vapor (at a concentration of 20000-30000 mg/m^3) - 0,059 mg/cm^2/h, i.e. over 20 times faster.

At low levels of exposure to 50% of the dose absorbed is oxidized to CO_2 and exhales. The high coefficient of partition blood/air (338.9 for man), determines the slow release of acetone absorbed by the body. The rest ketones are rapidly eliminated.

Except through exhaled air ketones are eliminated through urine (metabolized and unmetabolized) and up to 10% through the skin.

The amount of acetone in the urine used for exposition test.

The content of acetone in blood is about 50% higher than that in the tissues. The distribution in organs and tissues is as follows: brain, liver, pancreas, kidneys, lungs, muscles and heart.

Acetone is narcotic that harms all sections of the central nervous system. With prolonged inhalation accumulates in the body. The slow excretion increases the possibility of chronic poisoning.

The odor threshold varies around 1.1 mg/m^3. Concentrations 500 times above that level mark threshold of specific action proven electroencephalographic by disrupting the formation of a conditioned reflex. At concentrations above recommended MAC register irritating effect on the mucous membranes of the eyes, nose and throat. Concentration 100-120 mg/m^3 do not induce any signs of toxicity in the body of the workers. In operation, for one week at 10 times higher concentrations is established acetone in the blood. If the concentration of acetone in the air reach 5000 mg/m^3, the amount of acetone in the blood exceed 400 mg/l (at a norm of about 10-20 mg/l, maximal 25 mg/l).

Chronic poisoning with acetone is manifested by atrophic catarrhal changes in the lining of the UAP, anemia and left shift of the leukocyte formula. Changes in protein fractions of blood are also considered characteristic of chronic effect of acetone.

Acetone has a local irritant to the skin. Hours after application on intact skin establish redness with transient character after removing the irritating agent. Exposed areas skin is dry due to significantly depressed function of the sebaceous glands.

Methyl-ethylketone also is narcotic. After the first 3-5 min. in the majority exposed under the influence of concentration around 1000 mg/m^3 was observed irritation of the mucous membranes of the eyes, nose and throat. In short inhalation of concentrations 10 000 mg/m^3 feels strong odor, irritation of mucous membranes of the eyes and nose, at 30 000 mg/m^3 irritation becomes unbearable. In the combined action of the acetone and methyl ethyl ketone in a concentration from 2300 to 3000 mg/m^3 was recorded case of acute poisoning.

The effect of methyl-ethylketone on skin is poorly manifested, but in constant contact observed occupational dermatitis. Pre-drying of the skin slows and damping increases skin absorption.

PREVENTION

Individual protection of skin and UAP through appropriate personal protective equipment: working suit, goggles, gloves.

Special attention should be paid to nutrition. To limit fats, especially animal, to take more proteins containing glutamic acid, a large amount of lipotropic substances and methionine, easily digestible carbohydrates, lactic acid foods, vitamins.

It is also recommended to train workers to conduct breathing pauses with gymnastics and stick to increased physical regime.

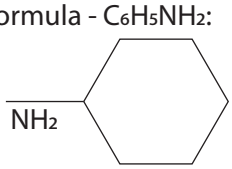
M.Koleva

7.8 NITRO - AND AMINO DERIVATIVES OF BENZENE AND TOLUENE

Nitrobenzene - $C_6H_5NO_2$

Aniline. Empirical formula - $C_6H_5NH_2$:

Structural formula -



o-, m- and p- nitrotoluene - $CH_3C_6H_4NO_2$

o-, m- and p- Toluidines (Aminotoluens) - $CH_3C_6H_4NH_2$

GENERAL CHARACTERISTICS

Nitrobenzene. *Yellow lubricous liquid smelling of bitter almond and sweet-tannic flavor. Molecular*

weight 123.11, boiling point 210,9 ° C. Slightly soluble in water, insoluble in acids and alkalis. Flammable and explosive substance with a lower explosive limit 1.8 volume %.

Aniline. *Lubricous liquid, colorless, but quickly darkening of light in contact with air. There aromatic odor. Boiling point 184,4 ° C, slightly soluble in water (3.4% at 20 ° C and 6.4% at 90 ° C), well dissolved in alcohol, ethers and other organic solvents.*

O-, m- and p- nitrotoluene. *O- and m-nitrotoluene are sallow, lubricous liquids, p-nitrotoluene a crystalline solid. Slightly soluble in water, mixed with alcohol in any proportion.*

O-, m- and p- Toluidines. *O- and m-toluidine are liquids, p-toluidine a crystalline solid. Slightly soluble in water, dissolve well in organic solvents.*

Nitro- and amino derivative of benzene and toluene are characterized by lower volatility and high lipoidosolubility, which is why most frequent poisoning caused by dermal penetration of substances, but rare - occurring by the inhalation route.

Inhalation absorption is high. For some industries, especially in direct contact with liquid product skin resorption plays a major role. Are described and household intoxications through clothes, dyed with aniline.

Because lipoidorsolubility of compounds the greatest amount is found in adipose tissue.

Sources of environmental pollution are mainly enterprises for the production of aniline dyes. Only one such object for 24 h discarded 3600 m^3 effluent containing approximately 17 organic compounds, primarily aromatic nitro derivatives.

Following exposure to nitrobenzene in water it changes color, smell and taste of the water. At a concentration of 4 mg/dm^3 yellowish water at 100 mg/dm^3 becomes almost black with black sludge. The odor threshold for water at 12-24 ° C is 0,6-1 mg/dm^3, and disagreeable flavor occurs at a concentration of 0,03 mg/dm^3.

HYGIENE STANDARDS

Air of the working environment

Chemical	Mea-shift MAC	Maximum single MAC	Specific effects
1. Aniline	2,0 mg/m^3	–	Skin-resorptive effect
2. Nitrobenzene	5,0 mg/m^3	–	Skin-resorptive effect
3. p-nitrotoluene	11,0 mg/m^3	–	Skin-resorptive effect
4. n-toluidine	9,0 mg/m^3	–	Skin-resorptive effect

Air settlements

Pollutants	Daily average MAC	Maximum single MAC
1. Aniline	0,03 mg/m^3	0,05 mg/m^3
2. Nitrobenzene	0,008 mg/m^3	0,008 mg/m^3

USE

Nitrobenzene is the main raw material for the production of aniline: 90% is used for the preparation of aniline, 2% - for the synthesis of other dyes, 7% - as intermediates in organic synthesis, and 1% for other purposes, ex. in the perfume industry, in the manufacture of soaps and more.

The most widely used of aniline in the textile industry for the production of aniline dyes and plastics, in the rubber and pharmaceutical industries, as well as for production of aromatic substances and semi-finished products of naphthalene and aromatic order.

Nitrotoluenes is used for the preparation of toluidines, in the perfume industry and low part as herbicides. Toluidine are used for the production of paints and in organic synthesis. In the composition of many corrosion inhibitors.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

In production conditions nitro- and amine derivative of benzene and toluene was mainly via inhalation and through the skin.

METABOLISM AND MECHANISM OF ACTION

Substantially all of the absorbed aniline metabolized to p-aminophenol, determination of which in urine serves as a exposure test. Major metabolic pathways is aromatic hydroxylation. The secondary roads are: N-hydroxylation, N-sulfoconjugation, N-glucuronidation, binding to glutathione to form aminoglutation and degradation to carbon dioxide.

Is eliminated primarily through the urine as p-aminofenol small part unchanged.

The biotransformation of nitrobenzene (by experimental data) occurs in 2 ways: a) reduction to the aniline followed by hydroxylation of the aniline to the aminophenols; b) direct hydroxylation of nitrobenzene with formation of nitrophenols and partial reduction of nitrophenols to aminophenols. Certain metabolic products are highly toxic. Is eliminated through the urine as 20-30% of the dose appears in the urine as sulfates and glucuronides of p-aminophenol and p-nitrophenol. Urinary excretion is slow - up to 3 weeks.

Nitro- and amino compounds by the mechanism of action and clinical picture of poisoning are very

similar, because in the body to metabolize the same products, but have some peculiarities.

For example, the nitro compounds act more expressed on the nervous system, while the amino compounds are stronger methemoglobine forming; toxicity of nitro compounds is not increased by increasing the number of nitro groups; joining chlorine to the molecule of aniline and nitrobenzene results in a more aggressive action on the blood.

A feature of the toxic effect of the nitro- and amine derivative is its polytropism, but the changes in the blood remains in the clear pathognomic sign of poisoning. Violations of the features of oxyhaemoglobin are associated with the formation of methemoglobin and develops anemia. Chronic poisoning results in increased regenerative capacity of hematopoietic organs, leading to reticulocytosis. Emerging hypoxia and hypoxemia cause dysfunction of all organs, primarily in the CNS. CCC disorders are due to the direct effect on the heart muscle, nerve centers, blood vessels and by the emerging hypoxia.

Nitro- and amino compounds damage the liver. In acute poisoning observed expressed pathology, while chronic poisoning are most often observed changes in the metabolism of pigments, due to injury of liver and hemolysis of erythrocytes. Clinical signs of damage to the gastro-intestinal tract observed. Renal impairment are also characteristic of the toxic effects of amino compounds.

Some of dinitro compounds affect metabolism by violations, that cause in oxidative phosphorylation.

In contact with skin cause dermatitis and eczema, are possible general and local manifestations of allergy.

Some amino compounds induced benign and malignant tumors of the urinary tract and bladder. A classic example is aniline, but also β - naphthylamine, benzidine, α - naphthylamine, o - toluidine.

Amino- and nitro compounds easily penetrate the skin, making this path way most dangerous of entry into the body in real production conditions, where their low volatility could not create high concentrations in the air.

PREVENTION

In contact with aniline and other compounds necessary protection of UAP and skin. Achieved through the introduction of continuous manufacturing processes, hermetisation of equipment, efficient ventilation and purification of emissions, that cause air pollution.

Workers must be provided clothing, underwear, shoes, gloves and respirators. Organized regular replacement of clothing and underwear. Recommended PPE are made from materials nonsorbent harmful substances and easy to clean. When spills and accidental contamination of clothing is recommended worker immediately to change. Especially careful to keep the skin from pollution during the summer. It prohibited in spillage and contamination of the skin to take a hot shower.

Workers are entitled to preliminary and periodic medical examinations. Required control of blood values. Recommended diet D and providing additional vitamins E - 20 mg and Rutascorbin - 1 dragee and 1 l mineral water Gorna banya, Targovishte, Ivanyane, Belovo, Varshetz, Varna.

M.Koleva

7.9 POLYCYCLIC AROMATIC HYDROCARBONS (PAHC)

Anthracene - $C_{12}H_{10}$ Benz (a) pyrene - $C_{20}H_{12}$ Naphthalene - $C_{10}H_8$

HYGIENE STANDARDS

Air of the working environment

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Anthracene	0,1 mg/m^3	–	
2. Benz(a)pyrene	0,00015 mg/m^3	–	Possibly carcinogenic to humans (Group 2A)
3. Naphthalene	50,0 mg/m^3	75,0 mg/m^3	

Air settlements

Pollutants	Daily average MAC	Maximum single MAC
1. 3-, 4-benzpyrene	0,1 μg/100 m^3	–
2. Naphthalene	0,003 mg/m^3	0,003 mg/m^3

Foods:

3-, 4-benzpyrene - 0.03 mg/kg.

GENERAL CHARACTERISTICS

Polycyclic aromatic hydrocarbons (PAHC) are organic compounds containing 3 or more conditioned (glued) benzene rings made of carbon and hydrogen. Are formed in pyrolysis and incomplete combustion or other type of heat treatment under high pressure of organic matter: coke, coal tar and resins, asphalt, oil and petroleum products or contaminated with these solid waste disposal, destroyed by combustion in incinerators. There are hundreds of polycyclic aromatic hydrocarbons. They are a natural component of natural fuels: coal and oil.

At room temperature are solid substances of low volatility. Dissolved in organic solvents. Do not dissolve in water. The majority of PAHC are degraded to simpler compounds under the influence of light.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Percival Pot (surgeon) for the first time in 1775 associated profession with cancer. He described an unusually high incidence of cancer of the scrotum in chimney and explains the origin of the disease to exposure to soot and ashes. Subsequently experimental repeatedly observed carcinogenesis induced by coal tar. Many PAHC have carcinogenic potential, most often it is contained 5 or 6 benzene rings.

Professional risk exists for all workers of enterprises producing or using coke, coal tar, bitumen (asphalt) and others petrochemicals.

PAHC are ubiquitous environmental pollutants. Set aside in the air in forest fires and volcanic eruptions. Found in room air for combustion of coal, wood, oil and petroleum products in fireplaces and stoves on solid or liquid fuel. The air in urban areas is contaminated with PAHC from motor vehicles and city incinerators.

Benz (a) pyrene has the highest potential carcinogen. Coal tar and creosote, which are a complex mixture of liquid and solid hydrocarbons, contain significant amounts of benz (a) pyrene and PAHC.

Cigarette smoke contains a many of PAHC. A cigarette is source of 10-50 ng benz (a) pyrene, chrysene - 18 ng, 40 ng dibenz (a, h) anthracene and 12-140 ng benz (a) anthracene. Cigarette filters retain some, but not all PAHC from the smoke, other inhaled by smokers.

The concentration of the PAHC in food products varies widely. Fried peanuts and coffee, refined vegetable oils, smoked meat, fish and sausages contain PAHC. To 2 μg/kg benz(a)pyrene is prove in smoked fish. Some grains and pulses such as wheat, rye and lentils synthesize PAHC.

METABOLISM AND MECHANISM OF ACTION

PAHC enter the body through inhalation, ingestion and contact with skin. So far there are no accurate data on the metabolism of PAHC in humans. All information on the absorption, distribution and elimination of the PAHC from the body is based on experimental studies with experimental animals. After absorption PAHC penetrate the lymph, circulate blood, metabolized primarily in the liver and kidneys and is excreted via the bile and urine. Due to its lipophilicity accumulate in milk (by the mammary glands) and adipose tissue. Sometimes excretion in faeces (via bile) and urine is effective enough due to the prevalence of enzymes transform PAHC in polar metabolites.

Furthermore, in the liver and kidneys PAHC metabolized in the adrenal glands, testis, lung, skin, sebaceous glands and intestine. Perhaps initially converted into epoxides, which are transformed into dihydrodiolic derivatives and phenols.

Persons with genetically determined high inducibility of the enzyme arilhydroxylazse (oxygenase of mixed function in the composition of the microsomal fraction) form probably the group at high risk of cancer due to contact with PAHC - believes that this is a decisive factor for incidence of lung and larynx cancer.

First epidemiological studies on mortality from lung cancer in workers from coke factory in Japan and England published in 1936. Until nowadays many epidemiological data reveal increased incidence of skin cancer, lung cancer, bladder and gastrointestinal tract when exposed to PAHC workers. In our sanitary legislation coal tars and resins are primarily in chemical mixtures as carcinogenic for humans.

Technical anthracene is significantly more toxic than pure. Inhalation of dust containing anthracene, appears swollen eyelids, irritation of mucous membranes and erythema, which is exacerbated by light - photodermatitis with burning and itching skin. Prolonged contact is observed pigmentation with hornification the surface layers of the skin and telangiectasia. Photodynamic action of technical anthracene was associated with the presence of impurities from carbasole, acridine and others.

The naphthalene affects the nervous system, gastrointestinal tract and kidneys. Typical are the changes in the blood: hemolysis, leukocytosis, anemia. Technical naphthalene is more toxic than pure because of the presence of impurities in thionaphthene, phenols and other.

Contact with skin causes erythema, eczema, appearance of red spots. There are reports of skin illness with redness, diffuse infiltration and pigmen-

tation of nails with general signs of poisoning. Acute dermatitis are frequent in workers in contact with naphthalene.

PREVENTION

Through complex preventive measures needed to limit emissions from coke-chemical industry, ferrous and non-ferrous metallurgy. In work places need to minimizing individual exposure of workers. By appropriate personal protective equipment to completely eliminate direct contact with skin.

It is also necessary to enhance the health education of the population and through effective measures to restrict smoking, especially among pregnant women and nursing mothers.

M.Koleva

7.10 CHLORINATED HYDROCARBONS

Methyl chloride - CH_3Cl (Chloromethane, Freon-40)

Methylene chloride - CH_2Cl_2 (Dichloromethane, Freon-30)

Chloroform - $CHCl_3$ (Trichloromethane, Freon-20)

Carbon tetrachloride - CCl_4 (Perchlorometan, Freon-10)

HYGIENE STANDARDS

Air of the working environment

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Methyl chloride	50,0 mg/m^3	100,0 mg/m^3	
2. Methylene chloride	100,0 mg/m^3	517,0 mg/m^3	Possibly carcinogenic to humans (group 2B)
3. Chloroform	10,0 mg/m^3	–	Carcinogen Group 3
4. Carbon tetrachloride	12,6 mg/m^3	–	

Air settlements

Pollutants	Daily average MAC	Maximum single MAC
1. Methylene chloride	1,5 mg/m^3	3,0 mg/m^3
2. Carbon tetrachloride	1,0 mg/m^3	–

GENERAL CHARACTERISTICS

Methyl chloride. *Colorless gas with a slight sweet smell. Partition coefficient oil/water - 25.3. Explosive - and fire product with a self-ignition temperature of 632°C, region of ignition of vapours in air from 7.6 to 19.0 volume %.*

Methylene chloride. *Colorless transparent liquid with a sweet smell, boiling point 40.1°C and partition coefficient oil/water - 26. Upon contact with an open flame decomposes and forms irritating and toxic substances. Under the influence of light decomposes and emits hydrogen chloride.*

Chloroform. *Transparent colorless liquid with a sharp sweet smell. Boiling point 61.2°C. On light, where free access of air easily decomposed. Upon contact of vapour chloroform with fire form phosgene. Partition coefficient oil/water - 7.3.*

Carbon tetrachloride. *Liquid with a boiling point of 76.8°C. Partition coefficient oil/water - 1000. Non-flammable, but in contact with open flames and high heated objects decomposes to form phosgene. As impurities contains carbon disulfide, hydrogen chloride, hydrogen sulfide, organic sulfides. Slightly soluble in water, soluble in chloroform, alcohol, ether and benzene.*

USE

Methyl chloride is used in the chemical industry as a methylating agent in the production of methylchlorosilanes, siloxanes, methylcellulose, tetramethyl lead; as a solvent in the production of butyl rubber, pesticides, oils and resins; as cooler and mixed with carbon tetrachloride as fire extinguisher.

Methylene chloride is used as solvent in the production of cellulose (particularly triacetyl cellulose), fats, resins, rubber, plastics; as cooler and for extraction of etheral oils.

Chloroform. More than 90% of the produced chloroform is used for the production of freons and fluorine-containing polymers; a small part as a solvent for fats and varnishes, in the pharmaceutical industry, production of alkaloids and rayon, as a fumigant in agriculture.

Tetrachloromethane was widely used as a inflammable solvent for fats and alkaloids, in the manufacture of refrigerants, fire extinguishers, electrical insulators, in dry cleaners and a small part as a means of deratisation.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Possibility of contamination of the working environment by chlorinated hydrocarbons exist wherever work with them in non-hermetic conditions. There are reports of occupational poisoning (mostly of carbon tetrachloride), and household poisonings most often when trying to dry cleaning of clothes.

Wastewater from the production of chlorine containing complex chlorinated hydrocarbons, especially methylene chloride. Such waters are not amenable to biological treatment and a risk of penetration of methylene chloride in open reservoirs. For now, the release of water is only through evaporation after 3-4 days.

Chloroform is formed in drinking water disinfected with chlorine as a result of reaction of free active chlorine with organic compound of natural (humic acids, proteins, amino acids, carbohydrates, etc.) and anthropogenic origin (high molecular compounds).

METABOLISM AND MECHANISM OF ACTION

In production conditions general way of penetration in the body of the chlorinated hydrocarbons is inhaled. Due to the affinity for fat is possible penetration through the skin and mucous membranes, particularly if theirs integrity is impaired.

Quickly absorbed and distributed in organs rich in fat (bone marrow, adipose tissue). Most small amounts are found in the lungs and bones. Metabolized in the liver with the participation of the OMF. One of the possible metabolites of methyl chloride is formaldehyde. The methylene chloride refers to non-reacting substances, in the body are not metabolized practical.

Chloroform and tetrachloromethane are drugs with marked toxic effect (less in chloroform) in relation to their ability to form free radicals.

Absorption of vapour of chloroform at inhalation reaches 60-80%. The content in the blood is the same as the concentration in the brain, in the fat concentration was almost 10 times higher. It is believed that increasing the concentration of chloroform in the blood over 10,4-12,5 mg% can cause death. 30 to 50% of the received amount in the body chloroform metabolizes. There is data that single metabolite is CO_2, an amount that corresponds to 50% of the absorbed dose. About 10% of chloroform absorbed is excreted unchanged until one hour after the exposure. The remainder is eliminated slowly (up to 10 days), especially in persons with obesity.

The biotransformation of carbon tetrachloride is low. About 20% of the absorbed carbon tetrachloride metabolized turning into chloroform, which in turn is oxidized to formic acid and CO_2. The main amount absorbed tetrachloride is eliminated by the lungs unchanged.

Saturated chlorinated hydrocarbons, cycloparaffins and cycloolefins (methyl chloride, methylene chloride, chloroform, carbon tetrachloride) are volatile narcotics. The strength of the narcotic effect of the derivatives of the same hydrocarbon increases with the number of chlorine atoms. Furthermore, chlorinated hydrocarbons increase the sensitivity of the myocardium to adrenaline.

Chronic action, and sometimes single exposure to high concentrations of chlorinated hydrocarbons, causing considerable damage to the internal organs as a result of fatty infiltration of the liver, kidney, heart muscle, pancreas. The most pronounced are these changes under the influence of carbon tetrachloride.

Prominent among chlorinated hydrocarbons is **methyl chloride**, which is relatively weak drug, but is highly toxic to the central nervous system. This is most likely due to the formation of methanol, which accumulates in the body, separated slowly and produce toxic metabolic products (formaldehyde and formic acid). Described cases of severe acute poisoning with methyl chloride (Freon-40) with prolonged loss of consciousness (over 24 hours), clonic and tonic convulsions, and absence of reflexes. Possible residual changes in the psyche, chronic depression and others. Ethanol and other higher alcohols potentiate the toxicity of chlorinated hydrocarbons in a combined action.

The effect on skin of chlorinated hydrocarbons is less pronounced than that of the corresponding saturated hydrocarbons (although they also penetrate through intact skin). Under the influence of chlorinated hydrocarbons is observed irritation of the skin and development of allergic dermatitis.

The methylene chloride was relatively less toxic. Chronic effect present with the symptoms of CNS damage, gastrointestinal tract, liver, and other internal organs, but after an interruption of contact changes are reversible.

There are pronounced cooling effect on the skin due to rapid evaporation (the likelihood of dermal absorption in toxic doses is unlikely).

Chloroform is also a powerful drug with toxic action. Chronic professional poisoning occurs with lesions in the liver.

Tetrachloromethane there is less drug action than chloroform, but is much more toxic. Regardless of the manner of entry into the body causing severe

damage to the liver - centrilobular necrosis and fatty degeneration. Both damaging and other parenchymal organs: kidneys (renal proximal tubules) and lung (alveolar membranes and blood vessels). Alcohol intake, cooling the body and increased concentration of oxygen in the air amplify its toxic effect.

Tetrachloromethane has a severe irritant action to the skin, causing dermatitis and eczema.

Ingestion per os developing severe hepato-renal syndrome, with possible exitus.

PREVENTION

Compliance with the general rules for safe handling of organic solvents is required. Do not ignore the danger of formation of phosgene.

It is necessary to provide a high degree of automation and a good balance between general and local ventilation to remove vapour of chlorinated hydrocarbons or products of their destruction in the place of release. When working with methyl chloride is recommended mixing with strong-smelling substances to quickly detect gaps in hermetisation. At high concentrations and fighting fires it is best to work with insulating gas masks or filter type. An important preventive measure is to avoid direct contact with skin. Mandatory preliminary and periodic medical examinations.

M.Koleva

7.11 CONTAINING FLUORINE CHLORINE HYDROCARBONS

Trichlorofluoromethane (Freon-11) - $CFCl_3$
Dichlorodifluoromethane (Freon-12) - CF_2Cl_2
Difluorochloromethyl (Freon-22) - CHF_2Cl
Difluortetrachloroetan (Freon 112) - CCl_3CF_2Cl
Trichlorotrifluoroethane (Freon-113) - $CF_2ClCFCl_2$
Dichlorotetrafluoroethane (Freon-114) - CF_2ClCF_2Cl
Pentafluorchloroetan (Freon-115) - CF_3CF_2Cl

HYGIENE STANDARDS

Air of the working environment

Chemical	Mean-shift MAC	Maximum single MAC	Specific effects
1. Trichlorofluoromethane (F-11)	5600,0 mg/m^3	–	–
2. Difluorochloromethyl (F-12)	3000,0 mg/m^3	–	–
3. 1,2-difluoro - tetrachloroethane (F-112)	1000,0 mg/m^3	–	–
4. 1,2,2-trifluoro – 1,1,2-trichloroethane (F113)	7600,0 mg/m^3	9500,0 mg/m^3	–

GENERAL CHARACTERISTICS

Chlor-fluoro containing hydrocarbons (CFCH) were synthesized for the first time in 1937 in Germany and soon find application as originally coolers and aerosol propellants. After 1950 and until mid-60s annual production increased by 100,000 tons, resulting in serious pollution of the atmosphere with all the negative consequences. In the early 70s for the first time spoke of the "ozone hole" - thinning the complete disappearance of the ozone layer of the stratosphere (currently "ozone hole" over Antarctica extends over an area of 14 million square kilometers). Two scientific expeditions, in 1986 and 1987 found that this is due to the chlorine atoms emitted from CFCH and other chlorinated hydrocarbons such as methyl chloroform (1,1,1-trichloroethane) and carbon tetrachloride. According signed in 1988 in Montreal Protocol, by the end of the last century the annual production of CFCH had to be reduced to half of the output in 1986 CFCH.

Some are gaseous (Freon-12, Freon-22), other liquid (Freon- 11) substances soluble in organic solvents and lubricating oils, practically insoluble in water. Under ordinary conditions not burn, does not form explosive mixtures with air. Relatively chemically inert, but

USE

Use	Type freon
Dry cleaning, cooling	Freon-113, Freon-11
Refrigerators, air conditioners	Freon-22, Freon-12, Freon-11
Foam fire extinguishing	Freon-11
Aerosol propellants	Freon-12, Freon-11, Freon-14
Production of plastics	Freon-12, Freon-22

in contact with open flames decompose and form difluoro- and fluorochlorophosgene.

Their properties make them sustainable in the lower layers of the troposphere - half-life 40-80 years of Freon-11 (average 50 years) and 75-150 years, an average of 100 years of Freon-12.

METABOLISM AND MECHANISM OF ACTION

As a rule, mixed halogenated hydrocarbons are less toxic than chlorine- or bromine-containing compounds. Represent a high risk that the products of their thermal decomposition containing phosgene, hydrohaloid acids, etc. Often contain impurities sharply increasing toxicity. The introduction of chlorine atoms in the fluorine-containing hydrocarbon chains increases the toxicity of the compounds, which is in connection with the process of dehalogenation (dechlorination) and the formation of free radicals.

CFCH enter the body by inhalation and is rapidly and completely separated then by breath. During inhalation itself is eliminated 95%, and in the next 30-45 min, and the remaining 5% of the respirable dose. Rarely, but there are refrigerants that form in the body of toxic metabolites.

Chlor-fluoro containing hydrocarbons are less toxic compounds. They have a narcotic effect, but at high concentrations, in normal practice of their application rarely reach. No evidence of neurobehavioural abnormalities under the influence of CFCH. They are not strong irritants, though prolonged or frequent action on the skin can cause typical for organic solvents dermatitis.

Increased sensitivity of the cardiac muscle under the influence of CFCH reported for the first time after several cases of sudden death in individuals exposed to very high concentrations of Freon-11 and Freon-12 in the 60s of last century.

According to the National Institute of Cancer Research in the United States Freon-11 showed no carcinogenicity in experiments on mice, as Freon-22 slightly increased incidence of tumors of the salivary glands in male white rats. Twice less, commonly used Freon-31 and Freon-133a, show limited carcinogenic potential in experimental conditions. Freon-22 (one of the more commonly used refrigerants) is genotoxic and weak bacterial mutagen. Numerous CFCH are tested for teratogenicity but different design studies does not allow to adequately interpret the results and make a general conclusion. There is data that Freon-22 has teratogenic in rats, but not in rabbits, which is manifested by microophthalmia and anophthalmia at concentrations of 50,000 ppm (176 899.96 mg/m^3).

REVENTION

Preventive measures are concentrated around the protection of UAP, as well as skin contact with liquid CFCH.

To operate at moderate high concentrations can be used a gas mask filter type. At high concentrations, risk of thermal destruction, accidents, use a mask insulating type. Provides complete hermetisation of technological equipment, protection against contact with hot surfaces, sparks, effective ventilation of rooms.

Workers are entitled to preliminary and periodic medical examinations.

M.Koleva, A. Mihaylova

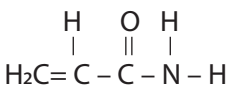
7.12 OTHER ORGANICS

AKRILAMID[1]

Possibly carcinogenic to humans (2B gr.)

Empirical formula, Structural formula

C_3H_5NO



HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 0.03 mg/m^3

Atmospheric air of settlements: not standardized.

GENERAL CHARACTERISTICS

Acrylamide (propenamide, acrylamide monomer) is a colorless crystals with a characteristic odor. Melting point 84,5°C, T° boiling - 87°C at 0.267 kPa, 103°C at 0.667 kPa. It is well dissolved in water, acetone and alcohols. Polymerizes when heated by ultraviolet rays with the danger of fire and explosion. Burning decompose toxic gases and vapors (NOx). Reacts violently with oxidants. Superfine particles form explosive mixtures with air.

[1] A. Mihaylova

USE

Acrylamide is the main raw material for the preparation of polymers and copolymers. The polymers used in the manufacture of building materials, emulsions, varnishes, paints, adhesives, plugging materials in dentistry for hardening compositions with high adhesion. Copolymers and butadiene are used to produce polyurethane. Significant amounts of acrylamide are used in the textile industry and in the manufacture of food packaging.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

The use of closed systems in the production of acrylamide reduces the possibilities to create conditions for occupational exposure. Extreme situations may arise in breach of reactors, in the accumulation of large amounts of dust from acrylamide, shipping and cargo handling activities.

Potential source of emissions is polyacrylamide, used in the purification of water supplies, sewage, industrial wastewater and flotation ore.

Minor amounts are released into the atmosphere in the production of synthetic rubber and organic synthesis.

In the United States has limited the content of monomers in food supplements (1 mg/kg), stored in the acrylamide packaging.

METABOLISM AND MECHANISM OF ACTION

In experimental conditions biotransformation of acrylamide occurs by conjugation. Binding of monomer to glutathione can be carried out in the liver and brain. Biliary excretion of glutathione conjugates was observed in intravenous administration. In urine identified four metabolites: mercapturic acid and three non-containing sulfur compounds. As a major metabolite shows mercapturic acid. About 2% of the introduced in body acrylamide is excreted unchanged in the urine.

Acrylamide occurs locally irritant in contact with skin. It has expressed neurotoxic effects. Damages the peripheral and central nervous system (the mechanism has not yet been elucidated). There is evidence of violation of axon transport, suppress the growth of neural and neuroglia cells, inhibition of mitochondrial respiration, reducing the level of dopamine, noradrenaline and 5-HP (5-hydroxytryptamine). The inclusion of vitamin B_6 to the food ration of experimental animals reduces the toxicity of the monomer.

PREVENTION

Preventive measures include automation and hermetisation of production processes; provide personal protective equipment - face mask, while accidents and explosions - filtering industrial aerosol mask with filter; conducting preliminary and periodic medical examinations with focus on peripheral and central nervous system, liver and kidneys

ACRYLONITRILE

Structural formula: (CH_2 = CHCN);

Empirical formula: C_2H_3CN

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 4,5 mg/m^3.

Specific effects: skin resorption action, possibly carcinogenic to humans, 2A group.

Air settlements: Daily average MAC - 0,03 mg/m^3.

GENERAL DESCRIPTION

Acrylonitrile is a colorless volatile liquid with a slight smell, a melting point of 83 ° C, a boiling point of 77.3 ° C and a density of 0.806 at 25 ° C. Typical is his ability to spontaneously polymerize. Can be stored only after adding polymerization inhibitors. Slightly soluble in water - 7.3%. And is insoluble in all classical organic solvents (acetone, benzene, ether, carbon tetrachloride, ethanol, methanol, ethyl acetate, toluene, xylene, etc.). His vapour is heavier from the air, forming explosive mixtures - limit content in the air - 3-17 vol.% acrylonitrile. Burning forming hydrogen cyanide.

USE

Acrylonitrile is known from 100 years ago, but until World War II almost did not use. By the war sharp increases in the production of nitrile rubber.

In the modern chemical industry is the starting material (monomer) for the synthesis of polyacrylonitrile, which are produced by the known polyacrylonitrile fibers. Used also as an insecticide under the trade name Ventox. Acrylonitrile containing polymers, mainly rubbers - acrylonitrile-butadiene-styrene rubber, acrylonitrile-styrene rubber, are used for the production of pipes and building structural elements, and in the automotive industry. Acrylonitrile is used for the preparation of acrylamide.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Professional acute and chronic poisoning are possible in production given the volatility of the compound. Risk are mainly working in the chemical industry.

The literature describes serious forms of poisoning of unprofessional character in children occurred after rubbing detergent in hair.

METABOLISM AND MECHANISM OF ACTION

In production conditions acrylonitrile penetrates mainly by the inhalation route. Acrylonitrile liquid and vapors penetrate also through the skin.

In the body are absorbed from 20 to 40% of the inhaled quantity. Metabolized in the liver with the

participation of the enzyme rodanase - specific for cyanides. End products of biotransformation are thiocyanates and mercapturic acids.

With breath eliminate 15-20% of the inhaled amount of acrylonitrile. In production conditions complete elimination is complete after 72 hours. As rhodanates were removed 14% of the dose during the first 4-6 hours after entry into the body. By 2-3 days after single exposure is an increased content of sulfhydryl groups in serum.

Acrylonitrile has irritating effect on mucous membranes at high concentrations (acute effect), but these effects are described and in chronic effects. Also described are skin lesions in contact; CNS disorders, and acute poisoning with lethal similar to cyanide poisoning.

For exposition test can serve the determination of acrylonitrile in the blood, the determination of the rhodanates in the urine, the determination of sulfhydryl groups in the serum and mercapturic acids in urine.

PREVENTION

Conduct preliminary and periodic medical examinations necessarily, as strict observance of contraindications for contact with acrylonitrile: organic CNS diseases, mental diseases, endocrine, respiratory system and CHD.

PETROL

Petrol products from the distillation of crude oil (mineral oil) or formed by thermal and/or catalytic cracking of its individual factions. According to their purpose is customary to divide into:

Aviation gasoline - a mixture of high-octane gasoline fractions and hydrocarbon components, to be added octane enhancers and antioxidants.

Automobile gasoline - a mixture of products of direct distillation and cracking process having a boiling point not more than 205 ° C.

Petrol - solvents and extractants - a mixture of low boiling fraction of oil in commonly property - very high volatility.

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC 900,0 mg/m^3 for gasoline - solvent and 300,0 mg/m^3 for gasoline - cracking.

Air settlements: Gasoline (petroleum with low sulfur content, calculated as carbon) - Daily average MAC - 1,5 mg/m^3, maximum single MAC - 5,0 mg/m^3; Gasoline (slate, calculated as carbon) - Daily average MAC - 0,05 mg/m^3, maximum single MAC - 0,05 mg/m^3.

USE

It is extremely broad application except as a fuel and as a solvent of rubber, oils, varnishes (White-alcohol), in the metal industry for degreasing of components and parts, washing engines in transport, extraction of fat, in dry cleaning and more.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Possibility of poisoning exists when used in practice as fuel in garages, at petrol stations, cleaning of tanks, pipelines, in emergency conditions, when using distillation and oil-extraction apparatus, clean clothes, and in any case their use as solvents.

METABOLISM AND MECHANISM OF ACTION

Penetration of gasoline in the body occurs mainly through inhalation in the form of vapour. Absorption of petrol vapour in professional skin contact is negligible. The absorption of liquid fuel through the skin is greater, but in comparison with that of benzene is weaker.

Due to the low solubility of gasoline in the blood even at lethal acute inhalation poisoning amount of gasoline in the blood is negligible - 0.3-0.4% (in experimental animals). Saturation of the blood and central nervous system is very fast. Received the body gasoline is released through the lungs unchanged also very fast. This is crucial for the prevention of acute inhalation poisoning.

Gasoline is highly irritating to mucous membranes (especially respiratory), damages the skin on contact, affects the central nervous system (lipoidodissolvent, narcotic). Described are morphological changes of blood - anemia, basophil pointed red blood cells.

PREVENTION

Prevention of chronic poisoning by inhaled gasoline required to ensure hermetisation of the technological equipment in all manufacturing processes and dynamically track the concentrations of gasoline in the air of the working environment.

Cleaning and repair of containers must be done in advance with purge by air or steam. Workers performing cleaning and repair of tanks should be trained to perform artificial respiration. It is forbidden singly to work in tankers.

In professional contact with gasoline, containing an increased amount of aromatic hydrocarbons recommended periodic monitoring of blood indices.

VINYL CHLORIDE

Carcinogen humans (Group 1)

Empirical formula: C_2H_3Cl;

Structural formula: ($H_2C = CH-Cl$)

HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 2,5 mg/m^3.

GENERAL DESCRIPTION

Vinyl chloride is a colorless gas with melting point -

153,8 °C and molecular mass of 62.5. Slightly explosive in a mixture with air, even at 4-22% of the mixture difficult to ignite. Easy joins a molecule of halogens forming trihalogenetanes. Under the influence of light polymerizes easily. On combustion (950-1466°C) smoke consists of: 27 parts of hydrochloric acid, 581 parts of CO_2, 9 parts of CO, and 0.04 parts of phosgene.

USE

Monomer of polyvinyl chloride, which is widely used in industry and households for production of building materials and products shaping the interior of the house (linoleum, tile flooring, decorative curtains and panels, washable wallpaper, ledges, handrails for stairs and others); water and sewage pipes, artificial leather, toys, food packaging.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Due to the widespread use of vinyl chloride direct professional contacts have over 100 000 workers in the world (in the US alone there are more than 27 000). Over 1/3 of the world population daily contact with products of polyvinylchloride resins.

Sources of vinyl chloride in the environment are plants for the production and processing of polyvinylchloride resin and various polymer materials based on polyvinyl chloride which contain free monomer and during the operation to liberate it.

Depending on the specifics of the technological processes in the production of vinyl chloride and polyvinyl chloride in the air of the working environment of vinyl chloride concentrations can reach values significantly exceeding hygiene standards (during the period 1960-70, the - 22-24 mg/m^3, and before that, and more). When processing a lining made of polyvinyl chloride occurs termodestruction and air in working environment is polluted with significant amounts of vinyl chloride - 28-105 mg/m^3. In the production of artificial leather and leather products are found vinyl chloride concentrations in the range of 2,4-5,6 mg/m^3.

Polymeric construction materials based on polyvinyl chloride for several months at ordinary temperature (18-22 ° C) emit volatile components including vinyl chloride. At high temperature the migration of toxic substances increases.

Vinyl chloride migrated from bottles to storing therein alcoholic beverages in quantity 10-20 mg/dm^3. The rate of migration depends on the duration of storage. It was found that the monomer migrates into water, vinegar, arachis oil and corn oil, orange puree and konjac in concentrations of 0.01 to 33 mg/dm^3.

METABOLISM AND MECHANISM OF ACTION

In production conditions highway penetration of vinyl chloride in the body is inhaled. Skin absorption is controversial.

Inhalation absorbed about 40% of the amount inhaled. At low exposure metabolism is accomplished by alcohol dehydrogenase, and high - with the aid of microsomal enzymes (cytochrome P-450). The first metabolite is chloroethylenes oxide, which is converted into an active intermediate epoxide. For it has been shown to bind to RNA and DNA in vivo, and most likely it is due to carcinogenesis observed in experimental animals and humans.

In intact through the lungs remove about 60% of inhaled vinyl chloride. Remainder metabolized and eliminated in the urine as metabolites whose content does not depend on the concentration of vinyl chloride in the air. Not developed exposition test.

In addition carcinogen (angiosarcoma of the liver) vinyl chloride is irritating to mucous membranes of the respiratory tract and inhibition functions of the central nervous system (depression) at high concentrations (acute poisoning). Chronic effects are detailed and other biological effects such as liver damage - fibrosis, hepatomegaly (and hepatosplenomegaly); vascular damage - Raynaud's syndrome; skin lesions - scleroderma, thinning of the skin; acro-osteolysis; pneumoconiosis; impaired reproduction.

PREVENTION

The reduction of exposure and reduce risk for people in contact with vinyl chloride is achieved through maximum mechanization, automation and remote control of production processes. In case of failure to provide workers with appropriate clothing from coated fabrics, protective eye (maximum sealed goggles covered type) and UAP (gas masks with oxygen bottle or gas mask filter type), latex gloves and waterproof boots.

ETILEN[1]

Empirical formula: C_2H_4

Structural formula:

CH_2

||

CH_2

HYGIENE STANDARDS

Air of the working environment:

Tentative safe level of exposure - 3.0 mg/m^3

Atmospheric air of settlements:

Daily average MAC - 3.0 mg/m^3,

Maximum single MAC - 3.0 mg/m^3.

GENERAL CHARACTERISTICS

Ethylene (ethene) is a colorless gas with a charac-

[1] A. Mihaylova

teristic odor. At elevated pressure and low temperature is liquefied. Temperature (T °) of melting 170 ° C, T ° of boiling 104 ° C. The chilled gas is heavier than air. Does not dissolve in water but is soluble in organic solvents. Reacts violently with strong oxidants. Flammable is to a distance. At high temperature and pressure polymerized. In its rapid evaporation creates a lower temperature.

USE

Used as a feedstock in the production of polyethylene, ethylene oxide, ethyl alcohol, ethanolamine, polyvinyl chloride, thyokol and others. It is used as a drug in surgery. Ethylene is a component of gas mixtures used in welding, lighting and more.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Professional risk exists in the production of ethylene. Main sources of gas removal are leaky flange connections, gaps in the bearing devices, operations sampling for analysis, in the prevention and repair of equipment.

Ethylene is formed in all plant tissues in significant amounts and acts as an endogenous regulator of growth. Is produced by soil microorganisms.

Sources of pollution are producing organic compounds, chemical and pharmaceutical industry, metal processing enterprises, gases emitted from motor vehicles and aircraft.

METABOLISM AND MECHANISM OF ACTION

Ethylene biotransformation occurs in the liver. By microsomal epoxidation to form ethylene oxide, which undergoes hydrolysis to ethylene glycol. Biodegradation of ethylene glycol is carried out by known circuits - conversion to oxalic acid or by the formation of intermediate metabolites (glycolic and hippuric acid), with the end product - oxalic acid. All metabolites are eliminated in the urine.

Ethylene is manifested as asfiktant and narcotic. Asphyxia, as a result of mass entering of ethylene, is proportional to the reduced content of oxygen in the air.

Narcotic effects of ethylene occurs at lower concentrations and does not depend on the lipid level, but on the content of water in tissues (the brain contains 78% water and 12% lipids).

Detected in animal experiment, induction of tumors (sarcomas) upon implantation of the polyethylene, is not associated with the action of the monomers - ethylene.

Ethylene interacts with biosubstratum in account of Vander-Valss connections. In biomedya of body does not undergo changes in its structure. In sufficient partial pressure of water to form a complex - latticed microcrystals hydrates by a fixed configuration. The system is unstable and pressure drop process is reversible.

PREVENTION

It is performed with technical events: automation and hermetisation of the production process and provide personal protective equipment for workers. In case of fire, explosions or mass chemical contamination to benefit from clothing coated textiles, gas masks oxygen bottle, latex gloves and rubber or PVC boots. Periodic medical examinations to be targeted at central, peripheral and autonomic nervous system, cardiovascular system and liver. Workers receive vitamins B_1, B_6, E and C.

ETHYLENE OXIDE

Molecular formula: C_2H_4O; Synonyms: ethylene oxide, 1,2-epoxyethane, oxirane.

HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 2,0 mg/m^3. Possibly carcinogenic to humans (group 2A).

Air settlements: MAC daily average - 0,03 mg/m^3, maximum single MAC - 0,3 mg/m^3.

GENERAL CHARACTERISTICS

Ethylene oxide is a colorless, flammable gas with a smell of ether. At low temperature, is a colorless low-viscosity liquid with a characteristic odor of ether, the temperature of boiling10,7°C and a density of 0.887 at 7°C. It is well dissolved in water, alcohol and ether, has a tendency to polymerization and reaction with many organic and inorganic substances. Lower limit of flammability in air mixture with 3% by volume. Upon heating to 400°C is converted to acetaldehyde, by heating with water to form ethylene glycol.

USE

The ethylene oxide is used for the preparation of ethylene glycol as an intermediate in the manufacture of polyester fibers, polyethylene (film, and various bottles), surfactants, washers and detergents for industrial and domestic purposes. As ethanolamine is used for the preparation of soaps, detergents and chemicals for textile and cellulose industry, such as glycol ethers for the production of surface coatings. It is used as a fumigant and disinfectant (dry sterilization) in agriculture, medical and dental practices, libraries, museums, gassing and disinfection of vehicles, production of milk and dairy products, and spices.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Envisage the technological and technical characteristics of the chemical industry (mechanization, automation and hermetisation of production processes), the most serious risk of occupational exposure exists for workers in the operations associated with loading installations with ethylene oxide and repairs.

Although less than 1% of the total production of ethylene oxide is used for dry sterilization, professionally exposed to ethylene oxide are a large number of persons in all health institutions, responsible for the sterilization of medical instruments.

METABOLISM AND MECHANISM OF ACTION

Ethylene oxide penetrates into the body by inhalation or through the skin. A specific narcotic toxicity that is most likely related to forming metabolic products are: formaldehyde or ethylene glycol. It is possible the formation and trimethylamine. Ethylene oxide is irritating and sensitizing effect. It is an alkylating agent and mutagen, damages male and female reproduction. Described are carcinogenic effects in animal experiments and in workers: mononuclear leukemia, stomach cancer, peritoneal mesothelioma, cerebral glioma.

PREVENTION

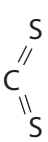
Main prophylactic measure is the hermetisation of the equipment and communications, wherever possible the formation and separation of ethylene oxide. At elevated concentrations in producing exceptional measures to protect the UAP, eyes and skin. Clothing, gloves and boots are waterproof and resistant to ethylene oxide. To protect the UAP used a gas mask filter type.

When dry sterilization is required to comply with all requirements of the instructions. After fumigation the premises must be ventilated mandatory.

CARBON DISULFIDE

Empirical formula: CS_2

Structural formula:



HYGIENE STANDARDS

Air of the working environment: Mean-shift MAC - 10 mg/m^3, proved gonadotrophic and embryotoxic action for animals.

Air settlements: Daily average MAC: 0,005 mg/m^3, maximum single MAC: 0,03 mg/m^3.

GENERAL CHARACTERISTICS

Carbon disulfide is a colorless liquid with a pleasant smell. It has the following properties: a molecular weight of 76.14, a melting point of - 112°C, a boiling point of 46,3°C, a density of 1.26 (at 20°C). Insoluble practical in water, in ether, alcohol and chloroform were mixed in all ratios.

When heated to 100°C sulfide vapour igniting. Combustion releases CO_2 and sulfur dioxide. With the air forms explosive mixtures in concentrations from 1.25 to 50 volume %.

The odor threshold is below MAC, which is a good signal to raise vapour in the working environment and the associated danger.

USE

Carbon disulfide is mainly used for the production of viscose (rayon) and cellophane. It is a good solvent for rubber, phosphorus, sulfur, fats and waxes. It is also used in the vulcanization of rubber as a raw material for the production of rhodanate compounds (thodonides), xanthogenates and potassium thiocarbonate. In agriculture is used as a pesticide.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Vapour of hydrogen sulfide detected in the air of the working environment in the production of rayon in the amount of 1 to 10, and in exceptional circumstances and up to 20 mg/m^3.

Modern viscose plants eject into the air from 1.5 to 40 tons of carbon disulfide per day.

In the area up to 2000 m around the plant detected concentrations in the range of 0,49-0,03 mg/m^3, but sometimes up to 2 mg/m^3.

It is possible contamination of surface water from wastewater plants for artificial leather and rayon.

In the treatment of the soil with a 50% emulsion of carbon disulphide (as a fumigant) creating airborne concentrations from 2,5 to 6,7 mg/m^3. After 24 h of carbon disulfide is not evidenced.

METABOLISM AND MECHANISM OF ACTION

The predominant route of entry into the body in manufacturing conditions is inhaled. Important and dermal route - for 1 h through the skin can be absorbed 17 mg hydrogen sulfide.

Inhalation of hydrogen sulfide vapour initially absorbed 60-80% and 20-30% at the end of inhalation. Equilibrium is reached quickly (within 1 h). The majority of the absorbed amount is associated with proteins, peptides, amino acids, biogenic amines and sugars. Are formed dithiocarbamic, thiocarbamic and xantogenic acid. 10 to 30% of the absorbed carbon disulfide exhaled and less than 1% is excreted unchanged in the urine; 70-90% is subjected to biotransformation and is eliminated from the body as metabolites: thiourea, thiocarbamic acids, sulfates, 2-mercapto-2-thiazoline-5-on. The formation of chelates of thiazolidines with zinc and copper, as well as the binding of CS_2 with SH-groups interfere with the normal function of many enzyme systems (in microsomes, cytochrome P-450). Carbon disulfide violates catecholamines metabolism - an inhibitor of dopamine- betahydroxylase (copper-containing enzyme), which increases dopamine and reduced norepinephrine. Another possible mechanism is the impact on vitamins - established a reduction of vitamin B_6, as a result connecting dithiocarbamic acid with pyridoxamine.

As lipoidosolvent carbon disulfide is narcotic, causing toxic encephalopathy (acute or subacute poisoning) can result in death. Chronic action features in addition to damage to the central nervous system and with pronounced effects on lipid metabolism - dyslipidemias; atherosclerosis; increased incidence of ischemic heart disease and arterial hypertension; retinal microangiopathy; reproduction - mostly spermatogenesis, but also female reproduction.

To assess the degree of exposure is determined: carbon disulfide in exhaled air, carbon disulfide in blood and urinary metabolites (iodine-azide test).

PREVENTION

In addition to sanitary and technical measures to reduce the concentration of carbon disulfide in the air of the working environment through maximum hermetisation, production automation and effective ventilation for workers in contact with carbon disulphide recommended: optimal regime of work and rest, quenching procedures safety professional nutrition to enhance the body's resistance, mandatory preliminary and periodic medical examinations with the participation of a neurologist, obstetric-gynecologist, if necessary and psychiatrist.

PHENOL (carbolic acid, oxybenzene)

Empirical formula: C_6H_5OH

Structural formula:



HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC 7,8 mg/m^3.

Air settlements: Daily average MAC 0,01 mg/m^3, maximum single MAC 0,01 mg/m^3.

Surface water: I category - 0,01 mg/dm^3, II category - 0,05 mg/dm^3, III category - 0,1 mg/dm^3.

GENERAL CHARACTERISTICS

Phenol is one of the most widespread pollutants.

Colourless crystalline solid with a characteristic (specific) odor. Molecular weight 94.11, a melting point of 40,9°C, a boiling point of 188,7°C, a density of 1.0576 (20°C). The crystals of the phenol by the action of oxygen in the air are stained in pink color is due to the quinone - a product of the oxidation. In 100 cm^3 of water at 16°C was dissolved 6,7 g phenol - form is a hydrate. The phenol was dissolved well in chloroform, ether, oils and organic solvents. Technical phenol is a rust-red to black viscous liquid.

USE

The phenol is widely used for the preparation of phenol-formaldehyde and other resins (bakelite, carboline), caprolactam, aromatics, medicinal substances, antiseptic and tanning agents, plastics, dyes and direct for disinfection.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Content of phenol in the air of production facilities and into the air, water and soil varies widely.

In the production of phenol are registered concentrations from 1 to 6 mg/m^3; in impaired hermetisation of equipment increased to 2 times and more. Obtaining phenol by carbonization of coal is accompanied by a concentration in the main workshop of 10-25 mg/m^3, in the workshop of distillation - 50-90 mg/m^3.

The concentration of phenol in ambient air around the ferrous metallurgy off 500, 1000, 2000 and 5000 m is respectively: 0,1-0,4 mg/m^3; 0,2-0,36 mg/m^3; 0,04 mg/m^3 and 0,03 mg/m^3. At this level of air pollution in the belt of about 2500 m away from the source of contamination phenol in soil varies from 0.06 to 19,2 mg/100 g of dry soil. Autotransport also a source of phenol - in the air of the settlements.

High concentrations of phenol settled in wastewater during thermal processing of solid fuel for the production of phenol phtalein, salicylic acid, salol, where regeneration of tires and others.

METABOLISM AND MECHANISM OF ACTION

In production conditions phenol penetrates the body mainly by the inhalation and dermal route. Respiratory absorption is high - 70-80% of the amount inhaled. Vapour of phenol pass directly through the skin. Absorbed phenol is rapidly distributed unevenly in the tissues: kidney, liver, heart, blood, muscles.

The phenol and diphenol metabolites conjugated with sulfuric acid and glucuronic acid.

With breath eliminate traces of unchanged phenol. Small amounts are released as CO_2. The basic amount (over 90% of absorbed) is excreted in urine as free and bound phenolics - in the form of sulphates and glucuronides.

The amount of total phenol in urine is exposition test.

Like other lipoido-solvents, phenol impairs the function of the CNS, more pronounced at higher concentrations in acute impact. Phenol is highly irritating to mucous membranes and skin. Binding to protein and subsequent denaturation and precipitation makes cytotoxic to all cells - causing tissue necrosis, in addition to skin and mucous membranes, also in liver, kidney, heart, urinary tract with appropriate functional disabilities.

Described is carcinogenic effects - skin cancer, in contact with phenol in experimental animals, but has not been confirmed in humans.

PREVENTION

Preliminary and periodic medical examinations are required. All workers receive in advance instruction for individual precautions and safe handling.

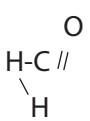
In the presence of phenol vapour in excessive amounts in the air of the working environment it is necessary to use a gas mask filter type and the presence of aerosols - the same mask with filter or respirator. Mandatory take precautions to protect eyes and skin from liquid phenol. Recommended suits of thick, impregnated fabric, gloves and overalls. It prohibited the export of work clothing outside for washing at home. In the medicine cabinet of workshop should have always diluted alcohol (10-40%) for washing the skin after pouring of phenol.

It recommended for workers to organize safety professional nutrition (diet D) and include vit. B_1 and calcium pantothenate.

FORMALDEHYDE

Empirical formula: HCHO

Structural formula:



HYGIENE STANDARDS

Air of the working environment:

Mean-shift MAC - 1,0 mg/m^3,

Maximum single MAC - 2,0 mg/m^3. Irritation, possibly carcinogenic to humans (Group 2A).

Air settlements: Daily average MAC - 0,003 mg/m^3, maximum single MAC 0,035 mg/m^3.

Surface water: I category - 0,5 mg/dm^3; Category II - 0,5 mg/dm^3; Category III - 1 mg/dm^3

GENERAL CHARACTERISTICS

Formaldehyde is a gas with a sharp odor. Synonyms: formic aldehyde, methanal oxomethane. Molecular weight 30.03. Easily soluble in water - 35-40% aqueous solution is called formalin. Gaseous formaldehyde burn and with air and oxygen form explosive mixtures.

USE

It is used as a monomer in the production of artificial (synthetic) resins and polymers on their basis: urea, phenol-formaldehyde and melamine-formaldehyde resins; synthesis of methenamine, drugs and explosives, dyes (indigo, rosemary, auras, acrylic paints); in leather, paper-pulp, textile industry. Excellent preservative properties of formaldehyde are used in photography and storage of plant and anatomical preparations.

PROFESSIONAL AND UNPROFESSIONAL EXPOSURE

Formaldehyde is one of the best known mutagens and this requires strict control in professional contact with the substance.

A constant source of environmental contamination with formaldehyde are enterprises producing or consuming formaldehyde. Contained in exhaust gases of diesel vehicles. It may be formed as a by-product as a result of a complex photochemical reaction, a catalyst which is UVR.

By wastewater possible contamination of groundwater and water of open reservoirs.

On the territory of industrial enterprises pollution of soil can reach 18-72 mg/100 g of dry soil, which can also lead to significant pollution of groundwater. Persistent in the environment at 20 ° C to 5 days.

METABOLISM AND MECHANISM OF ACTION

Formaldehyde is highly irritating and sensitizing effect. Central nervous system depression and especially ocular hills, which most likely is due to its metabolites: methyl alcohol and formic acid. It has mutagenic and carcinogenic effects of chronic exposure. Described are also the development of neurasthenia syndrome, bronchial asthma, neuritis n. opticus, effects on reproduction.

Formaldehyde is an important intermediate metabolite of the body. Formed intracellularly as 5-, 10-metilentetrahydrofolic acid.

Exogenously penetrates into the body by inhalation, ingestion or skin absorption. About 95% of inhaled quantity absorbed and rapidly metabolized to formic acid under the influence of formaldehyde dehydrogenase. The majority of the received formaldehyde in the body is transformed by intermediates (formic acid and methanol) to CO_2, low fraction of formaldehyde is excreted in urine.

Formaldehyde reacts with macromolecules: DNA, RNA and proteins (pathogenetic mechanisms of carcinogenic effect).

For exposition test serve concentrations of formaldehyde in blood and urine.

PREVENTION

To reduce exposure in industrial settings is recommended: hermetisation of technological processes, introduction of continuous processes, work on vacuum apparatus, remote control. If possible replacement of formaldehyde as a preservative with low toxic substances.

For personal protection recommended airtight goggles, waterproof suit, boots and gloves to protect UAP: gas mask filter type, but at high concentrations - insulating type.

Absolute contraindications for work are diseases from asthma and others allergic states and violations of the indicators of external breathing.

D.Tsonevsky, M. Lyapin

7.13 PESTICIDES

In agriculture and forestry **pesticides**[1] used to combat plant pests - bacteria, viruses, acarides, worms, rodents, weeds. To the group of pesticides concern and means for defoliation and destruction of shrubs and trees, for regulating growth. This group includes means for disinsection and deratisation used in households, industry and healthcare.

Pesticides are generally chemical compounds which are artificially synthesized or derived from vegetable products. Recently they have been created and pesticides of biological agents such as bacteria, viruses, fungi and the like. In this connection, the term pesticide includes both chemicals and microorganisms used for pest control.

The oldest pesticide agents were organic substances of natural origin such as tobacco, piretrium, petroleum derivatives. Later began to be used, and inorganic compounds such as the salts of copper, mercury, arsenic - eg. yet in 1860 successfully used so-called Paris green - copper acetoarsenite, against Colorado potato beetle. In 1930, there were 30 pesticides for agriculture - are most commonly used lead arsenate, pyrethrum and nicotine, and in the world today use about 1,400 pesticide active substances, included in about 60 000 commercial preparations. In world production of over 4 mln. tons of pesticides annually, the primary user is the US - 35-40% of the total. In our country are used on average 120-140 pesticide, as an annual average in the country applied to 20-25 000 tons of active substances. Systematic use allows for increased production in agriculture by an average of 60-80%. Conversely, only a few years a greater spread of pests can cause the destruction of hundreds of thousands of acres agriculture cultures.

Widespread application of pesticides, however, lies the potential for disruption of the ecological balance in nature and may have serious - direct and indirect effects on health. This translates into direct or indirect, professional or household intoxication - acute, subacute and chronic poisoning, lowering the reactivity of the body, sensitization, early and late genotoxic effects (teratogenesis, embryo- and gonadotoxicity, mutagenesis and carcinogenesis). In this respect especially dangerous organochlorine compounds, chlorfenoxy herbicides, contaminated with dioxins, some of thiourea derivatives. Also ChlOC and dioxins can for months and years to keep unchanged in the environment and accumulate in the body.

Deficiencies that have chemical agents used in agriculture led to an increased demand for new products, safer for the environment, people and animals. These are biological means for plant protection. Failure to comply with the rules when working with them, however, they can also cause chronic poisoning, hypersensitivity, imbalance of the natural microflora of body, contact dermatitis.

There are several criteria for **the classification of pesticides**:

1. According to its use pesticides are divided into:

acaricides - against ticks;

algaecides - to destroy algae and other vegetation in the basin;

antihelmintics - to struggle with parasitic worms;

antiresistantes - special additives that reduce the resistance of insects to certain substances;

antiseptics - prevention of microorganisms;

attractants - substances that attract insects;

aphicides - to combat aphids;

bactericides - anti-bacteria and bacterial plant diseases;

gametocides - substances causing sterility in weeds;

herbicides - weed control;

desiccants - the destruction of shrubs and low vegetation;

defoliants - to cause early loss and drying the leaves;

rodenticides or zoocides - for rodent control;

insecticides - to combat harmful insects;

larvicides - to destroy the larvae and caterpillars insects; nematocides - against round worms;

ovocides - chemicals, killing eggs of insects; growth regulators - substances affecting the growth and development of plants;

repellents - to repel insects;

fumigants - substances applied in vapour or gaseous (gas, mist, smoke);

fungicides - to combat fungal diseases of plants.

[1] **Pesticides** - lat. **pest** - wracker and **cido**- destroy, kill

In the nature of penetration insecticides (most used pesticides) are divided: contact insecticides - that kill insects on contact with the body; gastrointestinal - that penetrate the body of the insect through the digestive organs; systems - that have the ability to move in the vascular system of plants and insects destroyed after use of plants for food; fumigants - that penetrate the body of the insect through the bodies of breathing (many preparations may come in different ways, but determining what is a main route)

2. Chemical classification. In their chemical composition pesticides are divided into 22 groups **(Table. 1)**. Any substance in the group has its own characteristics and different from other mostly by pesticides and toxic action, but generally they are similar and often with the same mechanism of action.

Table № 1

CHEMICAL CLASSIFICATION OF PESTICIDES

Chemical group

1. Phosphorganic
2. Chlororganic
3. Carbamates and thiocarbamates
4. Dithiocarbamates
5. Triazines
6. Dichlorfenoxyacetic acid derivatives
7. Derivatives of urea
8. Derivatives of amides and anilides
9. Dinitrocompounds
10. Coumarin derivatives
11. Copper preparations
12. Sulphur preparations
13. Benzoilphenylureic
14. Dipyridilic derivatives
15. Pthalimides
16. Sulphates, sulfones and sulfonates
17. Benzimidazoles
18. Synthetic pyrethroids
19. Triazoles
20. Phosphides
21. Methyl bromide
22. Other preparations and mixes

3. Hygienic classification. This classification includes criteria for evaluating pesticides from a health and environmental perspective. Pesticides are divided into 4 groups **(Table. 2 and 3)** depending on their toxic parameters - acute toxicity and early and late neurotoxic and genotoxic damage (most often a limiting criteria is DL_{50} in rat after oral treatment). The criteria also include: percutaneous toxicity, inhalation toxicity, cumulation, sustainability in the environment, mutagenic, carcinogenic and teratogenic action, embryotoxicity, the possibility of acute poisoning, treatment options such as availability of antidotes, irritation of skin and mucous membranes, allergic action. Furthermore, they can be considered: the type of formulation (pellets, liquid, aqueous solution), available emulsifiers, the volatility of the pesticide.

To avoid acute and chronic poisoning with pesticides necessary knowledge of the following positions:

1. Methods of application.
2. Paths of penetration in the body.
3. Toxicological and hygienic characteristics.
4. Preventive measures in transport, storage and use.

Methods for application of pesticides are dependent on certain agrochemical requirements and hygiene rules. The most commonly used: dusting, spraying of solutions, suspensions or emulsions; decontamination in liquids, laying of poisoned baits; fumigation of rooms etc. Spraying with solutions (the most commonly used method) is done, by back-pack sprayers, mechanical tractor sprayers and specially equipped aircraft and helicopters.

Workers are exposed to contact with the active ingredients in the preparation of working solutions during charging of the sprinklers, in the direct treatment of crops, during cleaning of the machines.

Routes of the penetration of pesticides into the body are: through the skin, visible mucous membranes, respiratory system and through the gastrointestinal tract. The methods of application of pesticides determine opportunities for penetration in the body, but believes that a leading path in professional poisoning is skin (possible are both three ways). In some cases, the exposure through the skin is 100-1000 times higher compared with that by the respiratory tract, such as the highest skin permeability have emulsified concentrates, as a result they contain organic solvents and emulsifiers. Easily soluble in lipids PhOC, ChlOC, mercury preparations and nitrophenols quickly penetrate caught on exposed parts of the body, but it can be done and contaminated clothing. Spraying with knapsack sprayers creates the greatest risk of passing the pesticides through the skin.

By respiratory pesticides penetrate the body in all cases of their application, but the highest professional risk when used indoors, when working in sloping terrain, wind or falling into a cloud of pesticide spraying in jet.

Penetration of pesticides through the gastrointestinal tract, most commonly seen in suction mouth of working solutions, nutrition and smoking with unwashed hands, improper use of personal protective equipment, ingestion of concentrated preparations (criminalized cases). Poisonings have been observed

Table № 2. Criteria for classification of pesticides

Criteria	Not registered Extremely dangerous Zero category	May be used only by specialists I category Very dangerous	Work certificate is required Category II Moderately dangerous	Allow for wider use Category III Slightly dangerous
Acute toxicity				
Oral – DL_{50}				
Solid form	To 10 mg/kg	11-30 mg/kg	31-150 mg/kg	Over 150 mg
Liquid form	To 50 mg/kg	51-100 mg/kg	101-500 mg/kg	Over 500 mg/kg
Inhaler CL_{50} (4 h)	To 200 mg/m ^{3}	201-1000 mg/m^3	1001-2500 mg/m^3	Over 2,500 mg/m^3
Irritation of skin and eyes	Highly corrosive	Corrosivity	Irritation	No irritation
Sensitization	Pronounced in humans and guinea pigs	Allergens in humans, non-sensitizing effects in guinea pigs	Supposed effect	No sensitizing effects
Teratogenic effect	Proven abnormalities in people	Proven abnormalities in experimental animals - in 50% of them	Abnormalities in animals with less than 50% of the treated	Lack of teratogenesis
Carcinogenic effect	Proven in humans and in more than one species	Demonstrated in a laboratory animals and suspicious for people	No carcinogenic effect	No carcinogenic effect

Table 3. Criteria for classification of pesticides proposed by the Agency for Environmental Protection (EPA) - USA

Criteria	Class pesticides			
	I	II	II	IV
Oral LD_{50} (for rat)	50 mg/kg	50-500 mg/kg	500-5000 mg/kg	5000 mg/kg
Inhaler CL_{50}	0,2 mg/l	0,2-2 mg/l	2-20 mg/l	20 mg/l
Dermal LD_{50}	200 mg/kg	200-2000 mg/kg	2000-20000 mg/kg	20000 mg/kg
Eye effect	Corrosion; corneal opacity, which did not recover to seven days.	Corneal opacity, which is restored to 7 days.	Corneal not darkens inflammation is restored to 7 days.	No inflammation was observed.
Skin effect	Corrosion.	Strong inflammation at 72 h.	Medium inflammation at 72 h.	Low inflammation at 72 h.
Preventive measures	Fatal after ingestion, inhalation or absorption through the skin. Do not breathe vapors or dust. Do not fall into the eyes, skin or clothing.	Can be fatal after ingestion, or absorption through the skin. Do not breathe vapors or dust. Do not fall into the eyes, skin or clothing.	Damage after ingestion, inhalation or absorption through the skin. Do not breathe vapors or dust. Avoid contact with eyes, skin or clothing.	Low inflammation at 72 h. Not required precautions.

in the consumption of treated food without compliance with the guidelines (quarantine, etc.), or if used for food decontaminated with mercury preparations cereal seed.

Metabolism

The biotransformation of pesticides is carried out by mixed function oxidases (MFO) mainly in liver endoplasmic reticulum. The rate of metabolism affect a number of factors affecting the activity of enzymes involved in the MFO as: age, sex, genetic predisposition, general diseases, nutrition, daily routine, alcohol, substances inducers or inhibitors of the MFO. The MFO induction (eg. of some ChlOC - lindane, keltan, melbecs) assist both accelerated detoxification and to accelerate the "lethal synthesis" - eg. for some PhOC (parathion, metilparathion, ftalofos) wherein in the metabolism are formed much more toxic (hundreds of times higher anticholinesterase activity). It has been found that certain pesticidal mixtures consisting of PhOC, ChlOC, derivatives of carbamic and dithiocarbamic acid can have a highly toxic effect in comparison with the individual components, but in most cases (suitable mixtures thereof), decrease the toxicity of the combinations containing PhOC and ChlOC. (Some medications (phenobarbital) are also strong induc-

ers of the MFO and can be used as a prophylactic exposure to pesticides or treatment of intoxication.) Basic requirement when choosing a pesticide and evaluation of selective toxic action, involving high efficiency respect to insects and maximum safety for warm-blooded animals and man. For example, pesticides Actellic, Hortet, Valekson, Antio have up to 10 times more effective than Metafos for insect (by CL_{50}) and at the same time have from 200 to 2000 times less toxic to warm-blooded animals and man.

ORGANOPHOSPHORUS COMPOUNDS

First organic phosphate - tetraetilpyrophosphat (TEPP) was synthesized in 1854, but the development of PhOC was in the thirties of the twentieth century when in Germany synthesized the so-called "Nerve paralytiq gases" - zarin, tabun and zommer (chemical warfare). During the 40s and 50s are synthesized many new PhOC-insecticides, mainly in Germany (Schroeder).

General characteristics. Organophosphorus pesticides due to a number of benefits have largest application. In agriculture, used as insecticides, acaricides and defoliants. Some of them apply for disinsection - in domestic animals, on premises. PhOC, are complex esters of a number of acids - phosphoric acid, dithiophosphoric, thiophosphoric, pyrophosphoric. The main advantage over other pesticide that decompose relatively quickly in the environment. The resistance varies depending on the type of compound - from one day to several months. Some of systemic insecticides, however, can be saved and up to one year (sayfos, formothion).

Toxicological characteristics and mechanism of action.

Organophosphorus pesticides are compounds of varying toxicity to humans and warm-blooded animals. They enter the body by inhalation, skin and digestive tract. Typical of these compounds that can enter the body through intact skin without causing irritation (this represents a great danger to workers). After absorption in the body of larger amounts of PhOC was observed inhibition of cholinesterase (ChE) - an enzyme that hydrolyzes acetylcholine to choline and acetic acid. Specific AChE (acetyl cholinesterase) locates in ganglionic and myoneural synapses in the central brain structures and in erythrocytes, and pseudo-cholinesterase - mainly in plasma and liver. PhOC generally inhibit both enzymes, which resulted in nerve ganglia, in myoneural synapses and brain structures accumulate acetylcholine. Occurring endogenous acetylcholine poisoning, which determines the main clinical syndromes of poisoning: muskarinic, nicotinic and cerebral.

Organophosphorus pesticides inhibit other enzymes like other esterase, oxidase, lipase and proteinase.

For preliminary and ongoing sanitary control in working with PhOC importance is the study of serum ChE. In lowering the ChE more than 30% from baseline, workers must be removed from work. Symptoms of poisoning occur in lowering ChE with more than 50%, while chronic exposure due to adaptation processes symptoms can occur at very low levels of ChE - 15-20% from baseline. A specific antidote for PhOC is atropine.

CARBAMATES

General characteristics. Specimens of this group are used as insecticides, fungicides and herbicides. Carbamate pesticides are complex esters of N-methylcarbamate (baygon, dekrezil, sevin, mezurol) and N-dimethylcarbamic acid (pyrrolyl, RP-62). Naphthyl and cresyl esters of methylcarbamic acide are less toxic to warm-blooded, compared with dimethylcarbamic acid derivatives which contain in their molecule heterocyclic radicals (pyrrolyl). Carbamates do not accumulate in the environment and not general phytotoxicity.

Toxicological characteristics and mechanism of action.

Carbamate compounds mainly enter the body by inhalation and dermal route. They can cause acute poisoning, and, like PhOC inhibit the activity of ChE. This explains the similarity of the clinical picture in both groups pesticides. Unlike PhOC reactivation of the enzyme it is very fast and occurs and after having taken blood (this requires analysis of ChE be carried out within 2-3 hours of taking the sample). Upon termination of exposure to carbamate preparations symptoms disappear mostly after 1-2 hours and there are no known cases of chronic poisoning. In toxicodynamics esters of methylcarbamate (sevin) are essential damage to the endocrine organs, disruption of oxidative process and nucleic acids metabolism.

DITHIOCARBAMATE COMPOUNDS

General characteristics. Dithiocarbamate compounds are widely used in agriculture mainly as fungicides in the treatment of fruit, vegetable and cereal crops, or in seed treatment. The derivatives of dithiocarbamic acid (DTCA) as pesticides are used the salts of methyl-DTCA (karbation), dimethyl DTCA(TMTD, ferbam, ziram), ethyl-bis-DTCA (zineb, maneb, nabam, policarbazin) and ethyl- and methyl-esters of DTCA (preparation 23, zistogon). Furthermore, in agriculture are widely used different mixtures of dithiocarbamates (cuprozin - a mixture of zineb and copper salt of ethylene-bis-DTCA), deptan

C-45 (mixture of zineb and maneb) and mixtures of dithiocarbamates and other compounds such as kuprozan and miltoks, maneb and nickel sulphate. In the environment dithiocarbamates decompose relatively rapidly, whereby toxic volatile compounds (carbon disulfide, hydrogen sulfide, dimethylamine). Upon destruction of the dithiocarbamate into the environment and form stable compounds (dimethyl-DTCA, tetramethiltiourea, ethylenethiourea, dimethylamine). Ethylenethiourea is removed after heat treatment of food products treated with dithiocarbamates.

Toxicological characteristics and mechanism of action.

Dithiocarbamates may enter the body through the respiratory system, skin and through the digestive tract. Acute poisoning when they enter through the mouth and skin are not observed, given their low toxicity. Upon their receipt by the inhalation route are possible poisoning both the starting compounds of DTCA, and the products of their destruction (carbon disulfide, hydrogen sulfide, methyltioisocyanate). The majority of dithiocarbamates do not have a local irritating effect in single application on the skin, but often repeated contact sensitization is observed (cineb, maneb, TMTD, ciram) - dermatitis, bronchial asthma.

In animal experiments are established early and late genotoxic effects (embryotoxicity, teratogenicity, gonadotoxicity, blastomogenic and mutagenic). These effects occur in entering the body of amounts greatly exceeding the real ones. It is believed that these effects are due to their degradation products (ethylenethiourea, etilentiouram disulfid).

Dithiocarbamate compounds are distinguished by high reactivity to metals, which form metal complexes and thus influence the activity of many enzymes. They block the enzymatic conversion of inorganic iodine in an organic, and at chronic oral administration to experimental animals, can cause hyperplasia of the thyroid gland.

ORGANOCHLORINE COMPOUNDS

In 1874 synthesized DDT (dichloro-diphenyl-trichloroethane), but only in 1939, Paul Mueller discovers the insecticidal qualities and its commercial production, production of other ChlOC, and their use at 1945.

General characteristics. Organochlorine compounds (ChlOC) is applied in agriculture and forestry and for health purposes as insecticides, acaricides, fungicides. Their importance for elimination or reduction of yellow fever, malaria and other parasitic diseases transmitted by insects is extremely high, especially in tropical climates. In the past - 50-60 years, they are the most widely used insecticides, DDT, aldrin, dieldrin, chlordane, heptachlor, lindane and toxaphene. Their high persistence in the environment (around 20 years half-life) and their accumulation in warm-blooded animals and humans however, have led in recent decades to ban many of them.

ChlOC represent chlordeviratives of polycyclic hydrocarbons (DDT), compounds of cyclodiene row (tioneks), bicyclic aromatics - bichlorfeniles (keltan, methoxy), cycloparaffins (lindane), chlorinated terpenes and camphenes (toxaphene).

A distinctive feature of many ChlOC is the so-called process of **biomagnification**. This phenomenon is characteristic of compounds with much greater stability in the environment and in organisms and is characterized by a gradual accumulation along the food chain (ex. content of DDT in meat water gulls can exceed a million times its content in water ponds).

Toxicological characteristics and mechanism of action.

Organochlorine pesticides can enter the body through the respiratory system, skin or mouth. Unlike PhOC and carbamates penetration through the skin is less stringent, (depending on the solvent used). When ChlOC greater the risk of chronic poisoning (accumulation). Degrades predominantly in liver (inducing the MFO), central nervous system and gastrointestinal tract. Symptoms of acute toxicity is largely determined by the way of administration of the poison in the body. The mechanism of action of pesticides are associated with impairment of several enzyme systems (of the respiratory chain, carbohydrate metabolism), which often leads to tissue hypoxia. Hydrocarbons of the aliphatic range block the sulfhydryl groups of enzymes and deplete protein synthesis, and ChlOC diene synthesis by inhibiting the activity of cytochromoxidase.

In chronic exposure to ChlOC described cases of aplastic anemia in workers, and in animal experiments found carcinogenic effects of chlordane, heptachlor, toxaphene, aldrin and dieldrin.

SYNTHETIC PYRETHROIDS

Pyrethrum (extract from color chrysanthemum) is one of the oldest and high insecticidal activity of natural pesticides. It contains 6 active ingredients - pyrethrins (pyrethroids) - esters of pyrethrinic and jasmine acids. After World War II synthesized pyrethrins (synthetic pyrethroids) with molecules similar to natural but modified for stability.

General characteristics. Synthetic pyrethroids are among the most active insecticide (with low toxicity for humans). They are analogues of cyclo-

propanecarbonic acid. There are hundreds of commercial preparations containing pyrethrum and synthetic pyrethroids, often in combination with synergists - PhOC or carbamates. Apply for seed treatment, in vegetable and fruit growing, viticulture, rooms, in minimal concentrations - 0.01-0.1% active ingredient. Compared with PhOC at ten-rate lower concentrations they have the same effect on different insects. In the environment are degraded rapidly from 1 to 3 weeks after application. For example, in some of them (karate vastak, detsis) one day after treatment of fruit and vegetables remain negligible amounts of preparation.

Toxicological characteristics and mechanism of action.

Synthetic pyrethroids may enter the body through the respiratory system and digestive tract and fall on the skin. In humans, they decompose and emit much faster than in insects and fish - it also defines the very most toxicity to insects. Acute and hard poisoning in humans have not been reported. While exposure to pyrethrum are typical and frequent allergic contact dermatitis (described and allergic rhinitis, bronchial asthma, hypersensitivity pneumonitis, etc.) In synthetic pyrethrins allergic reactions have not been reported. If on skin can cause itching and burning, with no evidence of local inflammation. In rare cases can cause eczema, or skin inflammation.

The mechanism of their action is not fully understood. To clarify the causes of neurological disorders in mammals (seen only at high doses pyrethroids) were studied neural mediators and an increase in some of them (in insects, however, the main mechanism of their action is neurotoxic).

The toxicity of some pyrethroids is enhanced by inhibitors of MFO in mice, and they themselves to varying degrees induce mixed function oxidases. Some pyrethroids (permethrin, respethrin) cause carcinogenicity in experimental animals.

DINITROPHENOLS[1]

General characteristics. Nitrophenols are preparations with a relatively high toxicity, is very versatile in agriculture (such as insecticides, acaricides, ovicides, fungicides and herbicides). In fact, they are one of the oldest insecticidal agents - are used as such in the 19th century, and as herbicides applied by about 50 years. The most widely spread are selinon (4,6-dinitroorthocresol - DNOC) karatan, acricide and acrecs. Selinon contain 50% dinitroortocrezol and 50% fillers and has a insecticidal, fungicidal and herbicidal activity.

Toxicological profile and mechanism of action.

Dinitrophenol have different toxicity to humans and warm-blooded animals **(Table. 4)**

Dinitrophenol enter the body by breathing, digestive tract and through the skin, emit relatively slow (3-4 days) in the urine, unchanged or associated as glucuronide. Hepatic metabolism is negligible.

DNOC accumulates in the body. In blood this preparation is combined with plasma proteins and form compounds, kept for some time in the body. In warm-blooded animals is reduced to amino compounds. Emits significantly slow in the urine, where find as amidofenol.

The mechanism of action of dinitrophenols comprises impairment of enzymatic systems and blockage of oxidative phosphorylation. As a result, increased oxygen consumption and production of heat, impairs thermoregulation and increased body temperature. In poisoning by DNOC increases aerobic glycolysis due to hyperglycemia to yield a decline of glycogen in muscle and liver. During the summer days the risk of poisoning is greater, because the heat caused the increase in basic cellular metabolism.

Characteristic of systemic poisoning with these preparations is the total hypermetabolism, which is reflected in the clinical picture - profuse sweating,

Tab. 4. Dinitrophenols - administration and toxicity

Name	Active substance	Designation	Dosages and concentrations of application	DL_{50} mg/kg per os	Antidote	Quarantine - day
Karatan	dinoxan	fungicide acaricide	1-2 kg/ha 0,1-0,2% solution	980	–	21
Akreks	dinobuton	acaricide	7 kg/ha 0,1% solution	140	–	20
Selinon	DNOC	acaricide insecticide fungicide	10-20 kg/ha 3% solution	20-50	–	45
Acricide	binapacryl 40%	acaricide	0,1-0,2 %	150	–	21

[1] M. Lyapina

thirst, flushing, fever, anxiety or euphoria, tachycardia, hypertension and others.

Conflicting data on local irritation of DNOC. It is known, however, that commercial preparations based on DNOC irritate the skin and mucous membranes, and one of the chronic effects of these preparations is the development of dermatitis. Yellowing of the skin, nails and hair is observed upon contact with dinitrophenols; coloring of the sclera and urine is a sign of systemic absorption.

HERBITCIDES[2]

General characteristics.

Herbicides are pesticides that prevent or control the growth of weeds, or destroy them after their appearance. This chemical method of weed control largely replace mechanical (committed fewer people). The herbicides are now the most widely used pesticides - they make up 60% of total sales of pesticides in the US and 40% of those worldwide.

In the group of the herbicides include plant growth regulators, defoliants, and desiccants. Depending on the activity spectrum herbicides are non-selective, total, affecting all plants and selective affecting specific target weeds. In connection with the mechanism of action they have contact (attack plant parts in direct contact) and systemic (are absorbed by plants).

The herbicides are mainly the following groups: salts of phenoxyacetic acid and phenoxypropanoic, nitro- and chlorophenols, dipyridyls, triazines, carbamates and the like. Inorganic herbicides are dangerous and they are used less frequently. **Table 5** presents some organic herbicides with relatively weak health effects.

Tab. 5.

Name	DL_{50} (mg/kg) per os
Alachlor	1,000
Amitrole	1,000
Ammonium sulfamate	4,000
Atrazine	3,000
Bifenox	6,500
Dalapon	6,500
Dicamba	1,000
Diuron	3,500
Etfluralin	10,000
Glyphosphate	4,300
Linuron	1,500
Monuron	3,500
Oryzalin	10,000
Oxadiazon	3,500
Picloram	8,000
Prometon	3,000
Pronamide	5,500
Propham	9,000
Propanil	1,500
Simazine	5,000
Terbutryn	2,000
Tertrifluralin	3,500

[2] M. Lyapina

The herbicides are degraded in the environment to 1-2 years and in plants hardly found in good agricultural practice.

Toxicological characteristics and mechanism of action.

The major route of penetration in the body when applying herbicidesis is dermal; possibly penetrate into the body of individuals without occupational exposure - in the form of impurities in food or residues on crops. Herbicides generally possess low toxicity to mammals. There is relatively little information on the pharmacokinetics and their mechanism of action in humans. Certain herbicide formulations containing organic solvents, surfactants, emulsifiers, and other additives and binders, which may cause irritation of eyes, nose and throat (spray with herbicides), as well as dermatitis in prolonged skin contact. Some of the herbicides exhibit allergenicity and photosensitizing effect. There is evidence of carcinogenic effects of some herbicides (alachlor) in the experiment.

PHENOXYACETATIC ACID - DERIVATIVES

General characteristics. The herbicides of this group include 2,4-dichlorophenoxyacetic acid - (2,4-D - the most widely used in Bulgaria), 2,4,5trichlorfenoxyacetic acid (2,4,5-T), 2-methyl-4-chlorophenoxyacetic acid (MCPA), as well as their salts and esters. Formulations silvex, kuron and fenac are homologs of 2,4,5-T and 2,4DB, MCPB, MCPCA and MCPP are homologs of 2,4-D and MCPA, respectively. These are systemic herbicides relatively selective for deciduous plants used against weeds of cereals.

2,4,5-trichlorophenoxyacetic acid and its homologs are no manufactured in the USA, because combining these drugs with chlorinated dibenzodioxins (2,3,7,8-tetrachlorodibenzo-p-dioxin), known as strong carcinogens and mutagens. In some batches of 2,4-D is also found to be contaminated with less chlorinated dioxins, ex. dihlordibenzo-p-dioxin, but has not been established toxicological significance of any of these contaminants. On the other hand, the experimental data for the teratogenic and mutagenic action of 2,4-D are associated with contamination of the preparation with dioxins.

2,4-D is rapidly decomposed in the environment; water pollution with this preparation does not constitute a health problem.

Toxicological characteristics and mechanism of action.

Herbicides in this group mainly enter the body through the skin by direct contact with them (in the

preparation of mixtures, loading, etc.), inhalation and digestive tract. 2,4D, and most of the preparations of this group, not cumulative and excrete unchanged, easily and quickly through the urine.

The putative mechanism of herbicidal action of chlorophenoxyacetic acid includes simulated growth hormones (auxins) of the plants and their effects on metabolism. The mechanism of the adverse impact on humans is not yet fully understood. It is assumed weak blocking of oxidative phosphorylation, which is in extremely high doses resulted in increased production of heat and hyperthermia.

Some formulations of these compositions are strong irritants to the skin, eyes, nose, throat and respiratory tract and the gastrointestinal tract when ingested. In workers cause dermatitis and chloracne, discoloration or hyperpigmentation. There have liver, kidney damage and pancytopenia. There are reports of peripheral polyneuropathy, after dermal exposure to 2,4-D in relatively high doses for a few days. Experimental studies suggest that at a dose of 40 mg/kg per day for a month 2,4-D resulted in an increased incidence of cerebral tumors. Cases of lung cancer, soft tissue sarcomas and lymphomas in humans is also associated with exposure to herbicides of this group.

DIPYRIDYLS

General characteristics. The main representatives of this group of herbicides are paraquat (gramoxson is an aqueous solution of technical paraquat dichloride, used and methylsulfate of paraquat) and diquat (trade names reglon and agral). These are non-selective contact herbicide, widely used (especially paraquat) against weeds of vineyards, orchards, as defoliants and desiccants, cleaning of weeds in ponds. Paraquat is less stable in the environment. Diquat absorbed intensive in soil where dissociate under the influence of soil bacteria. However, some quantities of preparation, are found, and the seventh month after treatment.

Toxicological characteristics and mechanism of action.

Occupational exposure dipyridyls can enter the body through skin in contact with solutions, as well as breathing and digestive tract. The penetration of paraquat through the skin is facilitated when it is compromised the integrity. The contact of concentrated solution with the skin for a few hours, severe disability and symptoms of general toxic action - are described cases of acute toxicity and effect of aerosols of the preparation on the skin. Overall, paraquat is of the stronger toxicity of diquat - acceptable daily intake for paraquat is 0,002 mg/kg body weight for diquat - 0,005 mg/kg bodyweight.

Dipyridyls cause tissue damage as well as in plants and in mammals, by the generation of oxygen free radicals.

These herbicides (especially paraquat) have a pronounced irritant effect regardless of the route of entry into the body. They damage the epithelial tissues such as skin, nails, cornea, gastrointestinal and respiratory tract, and kidneys and liver. Contact of concentrated solutions with nails can result in breaking up and their fall. Aerosols of paraquat entering up in the lungs, causing irritation and inflammation.

Ingestion of paraquat clinical picture develops in three phases: early - abdominal pain, nausea, vomiting, a second phase with symptoms of liver and kidney damage and late phase - dyspnea due to pulmonary edema, which quickly evolved into pulmonary fibrosis (the ingestion of diquat does not lead to the development of lung fibrosis). Described are also the occurrence of the development of pulmonary fibrosis after exposure to paraquat by skin. The development of lung damage is facilitated by the active accumulation of paraquat in the lungs and its slow transmission thereof. In the mechanism of the described effects lies infringement of leucine metabolism of the alveolar epithelium and the surfactant. Impaired integrity of lipid membranes and phospholipids is due to lipid peroxidation.

Were reported hundreds of deaths worldwide due to ingestion of small amounts of paraquat. In order to reduce their frequency is exploring the possibility of adding an emetic to formulations of paraquat.

PREVENTIVE MEASURES DURING TRANSPORTATION, STORAGE AND USE OF PESTICIDES

From hygiene measures are particularly important replacement and banning of persistent and highly toxic pesticides from the first and second group and the introduction of effective but with low toxic action and quickly degradable pesticides (eg. synthetic pyrethroids).

To improve working conditions for loading and unloading of the preparations required mechanization of the main processes. Workers must wear work clothing and personal protective equipment. During the journey and work is forbidden to drink, smoke or eat. Provide bathe and change clothes after the completion of transport.

Pesticides stored in warehouses. Built on no less than 100 m away from residential buildings, barns or warehouses to forage, 200 m away from ponds and water sources, 50 m away from the main and 25 m of secondary irrigation canals. The floors need to be

adapted so quickly and easily cleaned and washed. Wash down water are discharged into a special built shaft. Highly toxic and volatile preparations can be stored in separate rooms from less dangerous. It is in the stores to have ventilation, running water, overflow and recuperation of preparations to become mechanized. The staff of the store should work required with special clothing and personal protective equipment.

Pesticide products under hygienic requirements must be placed in special metal or plastic containers or containers with hermetic closure to have durable labels in Bulgarian, contain the exact name of the pesticide, data on his toxicity instructions on how to use precautions and first aid for intoxication. Metallic and plastic packaging can be reused only for storing pesticides.

The preparation of the working solutions is carried out in pre-prepared containers intended for this purpose. Empty containers are treated with 5% caustic soda and burned in furnaces for solid waste.

The application of volatile products (PhOC, derivatives of chlorophenoxyacetic acid and the like.) in the warmer seasons of the year must be carried out mainly in the morning hours when the air temperature is not higher than 28 ° C and a wind speed of 3 m/s.

Always before work is tested functionality of machines. Workers who sprayed with knapsack sprayers should be a distance of at least 5 m apart (preparations first category should never be applied using knapsack sprayers). Tractors used in plant protection practice must be tightly closed cabins. Processing of aircraft allowed to land located at least 500 m away from the last buildings of the settlements and 200 m away from livestock farms. Treatment of areas with airplanes inhabitants of settlements, adjacent blocks shall be notified by the respective municipal councils at least three days pore (specify the day, time, place and used pesticides). For use of pesticides in personal holdings are allowed only preparations III and IV class.

Work clothes are deactivated for 2-3 hours in a 3% solution of bicarbonate of soda or concentrated soap solution, then launder. After each spraying workers must provide mandatory bathing or washing.

Working with pesticides undergo preliminary and periodic medical examination. At work are allowed only healthy persons and 18 years old. Do not allow people with skin, respiratory, infectious diseases, neurological disorders, mental disorders, chronic renal and hepatic disease, or used before alcohol. Also not allowed pregnant women and nursing mothers. Periodic check-ups are carried out 2 times a year by the therapist, neurologist and dermatologist (working with PhOC is compulsory control of ChE in blood serum).

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8 DUST AEROSOLS

In his life and work the man is inevitably exposed to dust impacts. Dust is one of the most important and universal influencing factors of the environment, which harms not only millions of industrial and agricultural workers in the world, but a large part of the population living in contaminated areas.

Collective term dust (dust aerosol)[1] includes a variety of organic and inorganic substances with the different in nature and force damaging impact, united by common physical property - highly fragmented particles that because of its small size and mass and relatively large surface stay in the air for differently long periods of time.

A huge number of manufacturing industries in which the person is exposed to the dust factor. To so-called dusty proceedings concern the open and underground mines for ore- and coal mining; the extraction of raw materials and the production of construction materials - stone, gravel, sand, etc.; metallurgy; mechanical engineering; textiles; glass and porcelain-faience industry, agricultural production, construction and others.

8.1. CLASSIFICATION OF THE AEROSOLS

According to the mode of preparing the aerosols are primary - (by disintegration and condensation) and secondary. Disintegration are obtained by mechanical grinding of solids, such as carrying out drilling works, blasting, seizure of extracted mass - in mining, hydro- and road construction, demolition, construction and repair of furnaces, logging and woodworking. A characteristic feature of disintegration particulate matter is that most of them have an irregular form. Condensation aerosol form in the condensation of vapour or gas after high temperature effects on substances that are in solid form at ambient temperature - dust-processes of melting metal and silicate minerals, in industries like metallurgy, glass and porcelain industry, electric- and oxygen welding, etc. Condensation aerosols are distinguished by disintegration in a smaller size, a narrow range of dispersion, usually have regular spherical shape and have a tendency to form agglomerates confluence of a large number of particles. Secondary are those powders that at some stage of production or end in packaging, transport and bulk materials are in powder or sedimented on working equipment, floor, windows, etc., and at work and in creating air currents pass again in the air of the working environment.

Basic parameters of dust aerosols necessary for hygienic health risk assessment and prevention of adverse effects are: type of dust; dispersibility and other physico-chemical characteristics of the powder; duration of exposure; concentration and pathogenic properties.

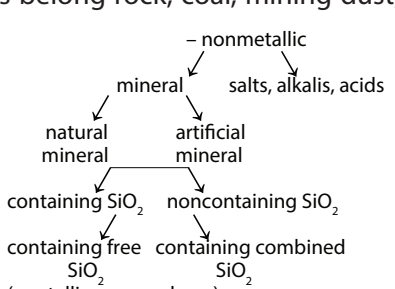
Type of powder. Type dust is divided into organic, inorganic and mixed.

The **organic powders** in turn are *natural* - plant (pollen, flower powder, seeds, cotton, linen, etc.) and animal (animal dandruff, hairs, microorganisms and their spores, mites, parasitic elements leaking by urine and feces of infected animals and people or formed during the process of decomposition and decay of organic matter, etc.) and *resulting from human activity* - synthetic powders (paints, plastics, pesticides, pharmaceuticals, synthetic fibers, detergents powders and so on..).

Inorganic powders are classified as:

– metal (metal oxides and condensation aerosols)

Metal powder and metal oxides are formed in the metal industry, such as condensation aerosols in welding, melting and casting. The natural mineral powders belong rock, coal, mining dust, dust of no



[1] The dust in the air is referred to as a colloid system in which the fragmented particles were dispersed phase and the dispersing medium is air, and therefore also means the concept of a dust aerosol.

ore minerals and others. To artificial mineral powders belong ceramic fibers, glass wool, stone wool. Aerosols of salts, acids and bases are formed in the production of mineral fertilizers, cement, condensation silicic acid and the like. Dusts rich in SiO_2 are free quartz, cristobalite, tridimit, and by synthetic - with crystal structure - for ex. koezit and with amorphous - aerosil, vulkasil, silica gels and the like, causing disease silikoza[1]. Exposure of powders rich in SiO_2 lead to develop silicatoses - asbestosis, talkosis, olivinosis, nefelinosis, apatosis, caolinosis and others. Exposure to dusts containing no SiO_2 leads to the development of aluminosis, baritosis, stanosis, berylliosis, manganoconiosis, pneumoconiosis of oxygen- and welders and others. Prolonged inhalation of nefibrogenic dusts leads to dust pneumonia - hammer man lungs and others.

In real terms the most commonly found mixed powders, for example: mineral-metal (a mixture of iron and silicon) - when welding; organic and inorganic - in textile production powder is mostly herbal, but contains impurities of mineral (soil) powder and so on. Dust particles can be carriers of the microflora (eg., grain dust can contain spores of various fungi).

Dispersion. The degree of dispersion powders are divided into: **dust** - the size of dust particles above 10 μm (these particles are precipitated under constant acceleration in static air according to Newton's second law (G = m.g) and rapidly sediment); **cloud** (microscopic dust) - the size of dust particles from 10 μm to 0,1 μm, which precipitate significantly slower with constant speed[2]; **smoke** (ultramicroscopic dust) - the size of dust particles below 0,1 μm - in static air they perform Brownian motion and practically no precipitation.

The greater is the dispersibility of the powder aerosol, the greater is its **physico-chemical activity** (this especially applies for the degree of adsorption). Dust tend to adsorb ions from the air, gaining positive or negative charge (dust acquire an electric charge and the formation of dust - in the process of weeds, crushing etc.). ***Electric charge status*** increases with increasing temperature and decreases with increasing humidity. Electric charge status powders is important for their stability.

The property of dust particles to adsorb gas molecules in the air results in the formation of a kind of buffer strips around dust which prevents the agglomeration and slows them precipitation. Another feature is the ***hydroscopic capacity*** of dust particles - absorbs moisture they increase its mass and faster settle.

The ***solubility*** of the powder particles - its speed, as well as the adsorption properties are closely related to the dispersibility and the total surface area of the powder aerosol. By reducing the size of particles and increases their surface accelerates dissolution. Many organic powders which mechanically traumatize and damage the mucosa of the upper respiratory tract, if it were soluble would have such effects, i.e. their insolubility is unfavorable, but the opposite - eg. it is at toxic acting aerosols.

8.2. DEPOSITION OF DUST PARTICLES INTO THE RESPIRATORY SYSTEM

From the dispersibility of the powder (particle size) depends on the depth of penetration into the respiratory system and the nature of the particulate damage.

"Precipitation" is the amount of dust that settle in different areas of respiratory in inhalation and exhalation (expressed as the difference in the concentrations of dust in inhaled and exhaled air). "Clearing" is the elimination of particles by ciliated epithelium, lung macrophages depending on where the deposition. "Retention" is the accumulated amount of dust in the alveolar region for a specified time after completion of primary bronchial purification, or 20 hours after the last inhalation.

Dust particles inhaled with the air can reach, and accumulate in various areas of the respiratory system, depending on their aerodynamic size, of geometric dimensions and shape of the airways, which carry and characteristics of breathing (in through, the nose, mouth, mixed, depth of breathing). A large part of them back to the breath, while others are eliminated by treatment mechanisms of the respiratory system. Important in terms of pneumoconiosis danger are those which are able to penetrate deep into the lungs and form there lasting depot. This fraction of the total suspended in the air of the working environment dust is referred to as *respirable* - this dust particles with a size of 0,5-5 μm.

The first protective barrier for dust were upper respiratory tract - so eg., almost all particles larger than 10 μm are held primarily in the nasal cavity, and breathing through the mouth only a very small percentage of them to penetrate the bronchi of the second order. In the nasal cavity main sewage safeguards are nasal hairs, released from the lining of nasal mucus, rich in lysozyme, in which stick dust, vibrating cilia of the epithelium and the nasal con-

[1] Disease described from Paracelsus - in 1530, Agricola - 1556, Ramazini - 1700. Term silicosis is proposed by Visconti - 1870, from latin silicium - silica.

[2] Now it is considered that the particle sizes up to 100-200 μm are precipitated at a constant speed by the law of Stokes. Effect on the rate of precipitation and has a flocculation process of the powder aerosol, depending on the humidity and the velocity of the air, the type and size of particulates.

cha, creating a highly undulating topography of the inner nasal surface. This air moves circularly with the greatest speed (120-150 cm/s) and therefore plays a major role inertia precipitation, called impaction. Precipitated particles are exported by the secretions of the pharynx and are eliminated either by absorption or by expectoration or mechanically cleaned (blowing, sneezing, etc.). Breathing through the mouth of the particles precipitated mainly by impaction in the oral cavity and pharynx, and eliminated by ingestion.

With the advent in the trachea and in bronhi in deeper parts of the respiratory system, where the air is moving at a slower speed (in bronchioles velocity up to 1 cm/s) a major role in the precipitation of the powder play sedimentation and diffusion. As it is less than the speed of the inhaled air and thus longer path, and as a greater mass of particles, the greater their sedimentation. Diffusion is important for particles with a diameter lower of 0,5 μm. They perform unruly (Brownian) motion, bump into walls of the airways and are precipitated thereon. Airway to the terminal bronchioles are covered with ciliary epithelium and mucus glands whose secretions is moved proximally from the ciliated epithelium. This is an effective means of eliminating dust particles before they reach the alveoli - so-called muco-ciliary escalator (function is difficult in inflammation or hypersecretion - smoking, irritating gases). In the alveolar region, lacking the ciliary epithelium, clearing of particles is relatively slow. An important role here play phagocytic cells. In the tracheo-bronchial tree to retain 10-15% of the particles size of 3 μm, and 40% of them reach the alveolar region. Almost all particles smaller than 0,5 μm reach the alveolar region but of which approximately 20% are preipitated (some of them are absorbed by phagocytes and are transported by muco-ciliary escalator, others cross the alveolar walls and penetrate the lymphatic lung system, rest is excreted in exhaled air). Around 80% of the inhaled dust is eliminated due to a combination of defense mechanisms. In persons who perform heavy physical work (large volume of breathing air), or who breathe mostly through the mouth are greater amount of large particles in different sections of the respiratory system.

8.3. CHEMICAL COMPOSITION OF THE DUST AND FIBROGENIC ACTION

In the proceedings are most often heterogeneous particulate aerosols, including ingredients with different chemical and biological action. The chemical composition of the dust determined biological activity - its fibrogenic, allergenic, toxic and irritation action. Fibrogenity of dust particles is determined primarily by the content of free silica.

Silicon is prevalent element in the earth's crust, occurring mainly in the form of silica. Quartz is a mineral composed entirely of free silica, but its crystals are mixed. Differ α- and β-quartz, α- and β- cristobalit, α- and β- and γ- tridimit - these are high-temperature crystal modifications of both the crystalline and the amorphous silica. Crystalline quartz (sand) is almost insoluble in water, even more slowly dissolves its deep layer, which has a great importance for the development of fibrosis. The amorphous silica has a greater solubility and is unstable at high temperature effects, it passes in tridimit, which in turn is converted into cristobalite at higher temperature impacts. Calcination of the amorphous silica increases the amount of free silica in it by 20-30%.

Various modifications of the quartz vary in their fibrogenity. Amorphous silica does not provide intense fibrosis. Cristobalite and tridimit have a more potent fibrogenic effect than quartz. Some crystalline modifications of silicon dioxide (stishovit) are biologically inert. Much fibrogenic activity to the lungs show crypto- (micro) crystalline forms of free silica (flint, chalcedony).

Fibroactivity of free silica was increased significantly in the presence of components such as lead, copper, arsenic, mercury, manganese, uranium and the like. Dust exposure required for the occurrence of the disease is reduced. There are particulate ingredients with antagonistic activity (reducing fibroactivity of free silica) - iron, coal, aluminum, hematite, limestone and the like.

Organic powders contain specific biologically active components **(Table. 1)**.

The relative role of each of these components, either individually or in combination with each other, has not been fully studied. Relatively more data is the role of the present in most organic powders bacterial endotoxins. Endotoxins are lipopolysaccharide components of the cell membrane of Gram-negative bacteria. The biological response of the human body upon inhalation them is expressed in an acute inflammatory reaction in the mucosa of the respiratory tract with invasion of neutrophils and the subsequent activation of other cells and secretion of inflammatory mediators. Another specific component of the organic powder is (1 → 3) - β-D-glycan contained in the cell wall of molds and certain bacteria - it enhances the inflammatory response to endotoxins and alter the function of inflammatory cells, especially macrophages and T-lymphocytes. Some of these biologically active agents in organic dusts - eg. proteins and enzymes, are allergen causing development of hypersensitivity pneumonitis.

Tab. 1 Main biologically active agents in organic dusts

Type organic dust	Biologically active agent
Vegetable powders	Tannins, histamine, alkaloids (nicotine), cytochalasin
Animal powders	Proteins, enzymes
Microorganisms	Endotoxins, proteases, mycotoxins (1 → 3) - β-D-glucans

8.4. EXPOSURE TO DUST AND THE RISK OF LUNG DAMAGE

Important factor in assessing the risk of dust impact was the exposure (E), which is equal to the multiplication of the time - T (working shifts in days, months and years, the whole service), in which man it has been exposed to dust and average concentration - $C_{avr.}$ of total inhaled dust and fine (respirable - below 5 μm) fraction in the breathing zone of workers during this period.

$E = C_{avr.} .T$

(This formula is valid for non-toxic powders with predominantly fibrosogenic action).

In assessing the impact of the so called powder depot (cumulative exposure) in late silicosis or silicotuberculosis essential to have "average stay of dust in the lungs," which is calculated by the following formula:

$AS = E/2+ DP,$

where : AS is the average stay of dust, E - the time of dust exposure (in years) and DP - dust-free period.

Concentration (mass) of dust into the air, along with the exposure time is of primary importance for the emergence of powder pathology and is an indicator of the efficiency of the preventive measures. Adverse effects of dust on the health depends both on its total mass (the concentration of total dust) and the concentration of fine powder - less than 5 μm (determined by weighing method and expressed in mg/m^3).

Data necessary for assessing the actual exposure to dust for each worker and for risk assessment of damage to health are available on individual sampling. It is realized with personal sampling devices located in the worker's breathing zone and provides information on its dust load for one shift.

Other significant factors for the occurrence of lung disease are the parameters of breathing defining of: anatomy and functional condition of the airways, way of breathing - in through the nose or through the mouth, posture, microclimates, the hard of labour, individual characteristics of body and others. Generally may indicate that the greater amount of powder has gone into the lung and longer dust is stored, the greater the likelihood of damage. Also however, the massive "dust stroke" lead rapidly to disease, especially in highly toxic dusts. The amount of the deposited quartz (Q) in the lungs is calculated as follows:

$Q = k. c. t,$

where k is a factor depending on the individual characteristics of the respiratory system, c - the concentration of fine (respirable) fraction of dust in the worker's breathing zone and t - the duration of dust exposure.

The risk (R) of disease of the lungs is a function of the amount of the powder depot (Q), its specific biological effect (a) and the time of his stay in the lung (T):

$R = f. (a. Q. T)$

8.5. EXPRESS EXPERIMENTAL METHODS TO DETERMINE THE EXTENT OF FIBROGENITY AND SILICOSIS HAZARD OF POWDERS

For predicting disease risk in exposure to silica-containing powders production are developed express experimental methods to determine the extent of fibrogenity and silicosis hazard them. This is the method of "subcutaneous pocket" in which 50 mg of studies powder suspended in a suitable nutrient medium, is administered subcutaneously in the back of the test animal. In place of the application of the powder to form a body of oval shape, which dissection at 7th day of the experience and its diameter is measured linearly (to 0.01 cm of the dimentions). On the basis thereof calculate the area (S) of the projection of the cavity filled with connective tissue, as determined and the amount of oxyproline (connective amino acid) in it - in mg/g tissue.

According to these indicators powders can be graded on silicosis hazard:

First grade - very high silicosis hazard with an area of subcutaneous pocket over 10 cm^2 and content of oxyproline in the tissue above 70 mg/g.

Second stage - high silicosis hazard, an area of the subcutaneous pocket of 6 to 10 cm^2 and containing oxyproline in the tissue of 45 to 70 mg/g.

Third stage - medium silicosis hazard, an area of the subcutaneous pocket of 4.5 to 6 cm^2 and oxyproline content in the tissue of 35 to 45 mg/g.

Fourth stage - low silicosis hazard, an area of the subcutaneous pocket of 1.5 to 4.5 cm^2 and oxyproline content in the tissue of 20 to 35 mg/g.

Fifth stage - no-silicosis hazard, an area of the subcutaneous pocket to 1.5 cm^2 and oxyproline content in the tissue to 20 mg/g.

8.6. HEALTH RISK FROM EXPOSURE TO DUST

Health risk is considered as a function between the severity of possible injury (degree of silicosis hazard) and the likelihood of its occurrence. The probability in turn is a function of the incidence of professional diseases and the exposure time.

Using the formula for the definition of risk (R) as a value determined by three parameters - probability (P) exposure (E), consequences (damage) (D)

$$R = P \times E \times D,$$

on propose risk assessment of lung damage from exposure at certain type of dust or powders with unexplained biological effect on humans. The probability (P) is equivalent to the frequency (F):

F = number of persons with proven professional disease of lung/100 workers.

Exposition (E) is calculated as:

$E = N \times T$, where N is the value expressing the ratio of the measured concentration of dust in the breathing zone of workers to MAC, and T is the exposure time

$T = T_1 \times T_2$, where

T_1 is the exposure time of the powder during the working day/week, for which are adopted the following ratios:

0.1 - very low (up to 1 h weekly)

0.2 - low (up to 1 h per day)

0.3 - medium (to 1/3 of working days)

0.4 - high enough (1/2 of the working day)

0.6 - continuous (all the day)

T_2 is the total duration of dust exposure (in years)

Consequences (damage) (D) equalizing by S (area of the ellipse of subcutaneous pocket) in determining silicosis hazard of dust in cm^2.

The risk (R) on the development of dust pathology:

1. to 7.5 - very limited, acceptable risk

2. from 7.5 to 30 - a small risk, it is necessary attention

3. 30 to 60 - measures are needed to reduce risk

4. from 60 to 260 - need immediate improvement of working conditions

5. 260 - leave off work to eliminate the risk

8.7. ADVERSE HEALTH EFFECTS FROM EXPOSURE TO DUST

Although the powders have very different physicochemical properties and chemical composition, their adverse health effects can be classified into the following guidelines: toxic action, locally irritable and corrosive, allergenic effect, specific action, carcinogenic action.

The main disease caused by exposure to dust are:

Lung disease - are caused by the accumulation of dust in the lungs and include the development of fibrosis, bronchitis, asthma and lung cancer.

The dust pathology is primarily pulmonary pathology causing occupational disease pneumoconiosis[4]. Initially as pneumoconiosis are designated all dust lung disorders, but subsequently only include those where there is development of pulmonary fibrosis (of varying degree and nature). In etiological sign has 6 major groups pneumoconiosis: **silicosis** - the most typical and fast growing pneumoconiosis caused by dust containing free crystalline silica; **silicatoses** - by powder comprising salts of SiO_2 (bounded silica), for example. asbestosis, talkosis, olivinosis, nefelinoza etc.; **carboconiosis** - carbohydrate meals - eg. coal dust (anthracosis), powdered graphite, carbon black, coke, etc; **metaloconiosis** - powdered metals (metal oxides), for example aluminosis, baritosis, siderosis, manganoconiosis etc.; **pneumoconiosis of mixed powders** with different content of free and bounded silicon dioxide, for example siderosilicosis, antracosilicosis etc.; **pneumoconiosis from organic powder** (plant, animal, synthetic) eg. tabacosis (tobacco powder) bagasosis (reed), byssinosis (cotton), amylosis (flour), from wood dust, plastics, etc. In these pneumoconiosis can occur and dominate other effects besides fibrosis - ie. asbestosis is often combined with lung cancer (mesothelioma, cancer of the bronchus), silicosis (less silicatosis) combined with pulmonary tuberculosis - silicotuberculosis; in pneumoconiosis from organic dust prevalent allergic reactions - hypersensitivity pneumonitis, bronchoconstriction, bronchial asthma; in carboconiosis (anthracosis etc.) is a very common lung emphysema and slowly developing interstitial diffuse fibrosis, etc.

Systemic effects - occur with absorption in the blood of inorganic toxic dusts containing elements such as lead, manganese, cadmium, mercury, as well as absorption of certain components (also with toxic action) of the organic powders.

[4] Pneumoconiosis - a term first introduced by F.A. Zenker in 1866 from greek pneumon - lung and konis - dust.

Metal fever - is observed at inhalation of finely dispersed condensation aerosols of oxides of zinc, magnesium, copper, derived from the melting of metals. It has been reported that inhalation of aerosols, resulting from the melting of aluminum, antimony, cadmium, copper, iron, manganese, nickel, selenium, silver and other also cause metal fever.

Allergic and hypersensitivity reactions - in the inhalation or skin contact with vegetable powders (flour, cereals, certain types of wood dust, medicinal plants), as well as some organic and inorganic materials (chromium, nickel, drugs, enzyme preparations used in the rubber industry as auxiliary materials accelerators, stabilizers, etc.).

Bacterial and fungal infections - exposure to dusts containing live microorganisms (anthrax in woolen powder) or powder of cereal, nuts, containing pathogenic fungi.

Irritative action - such effect have acids, bases, etc. It is well known that chrom containing dust cause ulceration and perforation of the nasal septum.

Another very common occupational lung pathology - chronic dust bronchitis is also associated with irritation of dust on the mucous membranes of the airways - mechanical, chemical, allergenic effect of the dust aerosol. These effects start from the nasal cavity, pharynx and larynx, especially aggressive is silica containing powder (rapid development of hypertrophic and atrophic catarrh of the upper respiratory tract, followed by atrophic sclerosing bronchitis with frequent metaplasia - after 15-20 years of service). Potentiating effect have other production factors - excessive heat or cooling microclimate, irritant gases, work loading, also available respiratory diseases and smoking.

Injury to the eye mucosa - conjunctivitis and keratitis, caused by the mechanical and chemical action of metal and mineral dust aerosols on the eyes.

Skin contact various kinds of dust cause a variety of local disability - eg. eczema, dermatitis of allergic nature; argirosis and anthracosis in skin due by impregnation of dust particles of coal and silver; dermatitis and eczema, acne, furuncles, injuries to the sweat glands, sores, etc.

Carcinogenic effects - inhalation of asbestos dust (mesothelioma, cancer of the bronchi); metal aerosol - nickel, chromium, arsenic, etc.; powder with enhanced natural radioactivity, or a combination of powder with the radioactive effect of radon and smoking (potentiating effect).

Radiotoxic action - stochastic and non-stochastic damage to organs and tissue from inhaled radionuclides.

8.8. PREVENTION OF ADVERSE EFFECTS

Basic preventive directions can be classified as: 1. Technological 2. Sanitary-technical 3. Hygienic 4. Medico- prophylactic.

The most effective and radical are the **technological and sanitary-technical solutions** limiting dust formation and dust scattering.

These include:

– radical changes in technological processes with intensive dust: maximum avoid dry techniques processing- eg. introduce of water perforators in Bulgaria in 1956 .; implementing effective means of wet dedusting - using tools and methods for increasing the wettability of water - placing in blasting holes in the plastic cartridges filled with water or pastes prepared on the basis of cellulose or bentonite (which led to a reduction of dust in underground mines from 20 to 80%); implementation of uninterrupted work precesses managed automatically, mechanization and hermetisation.

– the use of powders and raw materials can be minimized by introducing pasty and granular (eg. in plastic production).

– encapsulation and placement of local exhaust ventilation with mandatory dust hold before the release in the atmosphere.

– the transport of raw materials is carried out by shorten the path, using the band sealed, screw or pneumatic, applying periodic spraying with water.

– isolated in separate premises processes, machinery and equipment with intensive dust.

– establishment of effective general exchange ventilation of descending type and local aspiration or encapsulation also from descending type accordingly particularities of production.

– in ventilation with recirculating air, content of dust in the recirculating stream can be up to 10% of MAC for work environment.

The group of hygienic and medical preventive activities include:

– removal of the settled dust in workplaces, best by pneumatic suction with industrial vacuum cleaners.

– use of appropriate personal protective equipment (masks, goggles) and dust protection clothing.

– organizing systematic control of dust in the air*.

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* **air in work environment:**
- mean-shift MAC for dust to 2% free crystal SiO_2
  - inhalable fraction - 10 mg/m^3
  - respirable fraction - 4 mg/m^3
- at higher SiO_2 or other substances - lower MAC - 3-8 mg/m^3

air settlements:
- dailly average MAC - 50 μg/m^3 FDP_{10}
-average annual MAC - 30 μg/m^3 FDP_{10}

– conducting preliminary and periodic medical examinations - every 12 or 24 months involving therapist, ORL, dermatologist, ophthalmologist.

– ensuring the safety professional catering for workers (diet B) - rich fat (PUFA), proteins (cystine, cysteine, methionine), calcium and vitamins (vit A) food, inhibits the development of silicotic process.

– providing conditions for good personal hygiene (two-sector cabinets for work and personal clothing, bathrooms, laundry, etc.).

– organizing photiriums - with UV-exposure, individual inhalations with saline and sodium carbonate solutions (5% solution of NaCl, 2% soda solution) to restore the self-cleaning function of the bronchial tree.

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9 CHEMICAL FACTORS WITH CARCINOGENIC EFFECT

Carcinogenic effect as general biological phenomenon is a process of converting a normal cell into a tumor under the influence of carcinogens. Tumor transformation is observed under the influence of three main groups of carcinogens: chemicals, radiation and oncogenic viruses in all species.

It is estimated that currently 30-40% of the population in industrialized countries developed malignancies during their life. The main part of cancer in humans are caused by environmental factors (70-80%), as a leading place among them have chemical pollutants (70-85%). Today, modern man consumes more than 60 000 chemical substances. Around 16% of them were tested for carcinogenic activity on animals. Till now experimentally found carcinogenic activity of more than 1,000 chemicals, of which more than 40 have proven carcinogenic to humans.

9.1. METHODS FOR EVALUATION AND CLASSIFICATION

Carcinogenic risk of chemicals and industrial processes are evaluated using the following basic methods:

1. Epidemiological studies: include descriptions of individual cases or observations of large groups of people for an extended period of time (15-30 years) exposed to certain chemical substances. Data from these studies are most important, because through them is estimated actual carcinogenic risk in humans.

2. Experimental studies. Data from experimental animal studies are essential in assessing the carcinogenic risk because epidemiological studies are possible only after 20-40 years of presence of chemicals in the environment. The study of a substance for carcinogenicity lasts from 3 to 5 years and takes place in three stages: preparatory, chronic experiment and evaluate the results.

3. Express tests. The classical methods of experimental oncology are labour-intensive and expensive, and therefore in recent years have been proposed more than 100 express test. They were developed on the basis of certain basic characteristics of chemical carcinogens: induction of chromosomal aberrations and reparative DNA synthesis in cell cultures, the use of bacterial systems, blast-transformaion of cell culture, disruption of the cell genotype in the form of point mutations. Practical most widespread testing methods have mutagenic action of chemicals as an indicator of carcinogenic activity. Systematic studies have found that 90-95% of chemical carcinogens tested on bacterial test systems from mutant strains (Salmonella typhimurium - 5 strain, method of B. Ames et al. - 1975) are proven mutagenic item, i.e. disclosed is a high correlation between the ability of a substance to induce mutations in microbial test systems and the development of cancer in experimental animals (potential carcinogens in humans). According to the modern concept of the mechanisms of tumorigenesis, carcinogens with mutagenic effect are genotoxic unlike of epigenetic which acting by others, often not well defined biological effects, associated with functional changes in the genome - violation of physiological and regulatory processes in cells, disrupting permeability cell membranes and the interaction between the cells. Prognostic significance of rapid screening test systems does not exceed 90-95%, ie they are binding, but can not replace experimental studies (allowing, however, be screened).

Based on the data of the main methods for assessing the carcinogenic risk of chemicals and industrial processes, WHO experts offer their division into three groups:

1. First group - chemical substances, mixtures and industrial processes that are **carcinogenic to humans** (asbestos arsenic, chromium, 2-naphthylamine, benzidine, vinyl chloride, benzene, etc.). In them a causal relationship between exposure to the agent and the development of cancer in man. Development of tumors in various types of lines or animals using various dosages and routes of administration of the test substance.

2. The second group - chemical substances, mixtures and industrial processes that **are proba-**

bly carcinogenic to humans. In subgroup 2A are included substances, and industrial processes for which the evidence is relatively strong by those of subgroup 2B (aflatoxins, acrylonitrile, diethyl sulfate, beryllium, etc.). In the evaluation of carcinogenicity of the substance of this subgroup main are results obtained in the experimental studies.

3. A third group - chemical substances, mixtures and industrial processes that can not be accepted or rejected as carcinogenic to humans (aniline, dichlormethane, dieldrin, saccharin, lead and its compounds, reserpine, hematite, etc.). The available data are insufficient in quantity and quality.

9.2. MECHANISMS OF CARCINOGENESIS

According to current studies carcinogenesis associated with the most common and basic life events and characteristics of the species, rather than separate and specific to a particular type or tissue structures and functions. In chemical carcinogenesis establish some basic laws; 1) the chemical induction of cancer process is a result of too specific interaction between the carcinogen and cellular targets; 2) the cancer has monoclonal character, ie the tumor arises from changes in one cell and its progeny; 3) the progression from normal to malignant phenotype comprises a plurality of phases with varying degrees of stability and reversibility; 4) carcinogenic effects depend on the dose, they accumulate and are almost reversibilic; 5) modifying factors can accelerate or inhibit the process of carcinogenesis. The mechanisms of action of carcinogens associated with their metabolism. Some of these are direct acting, but most require metabolic activation by cellular enzymes (MFO), which results in highly reactive so-called carcinogens. The isolation of the final carcinogens is difficult because of their high volatility and reactivity. These activated metabolites interact with the nucleophilic centers of cellular macromolecules (DNA, RNA, proteins) and form covalent bonds with them.

Experimental and epidemiological studies of carcinogenesis indicate a latent period and phasic nature of tumorigenesis. For people duration of **latent period** varies from 4-6 years for leukemia induced by ionizing radiation to 40-50 years on some cases of mesothelioma induced by asbestos, for most tumors that period averaged 12 and 25 years. The duration of the latent period showed comparability in various experimental animals and humans, according to life expectancy.

In the process of carcinogenesis often differ two main stages. The first stage is the initiation and the respective agents that cause it are so-called initiators. The second stage is the stage of the promotion, and the agents are called promoters. The sequence is obligatory (if initially treated with promoter tumor can not be obtained). The stage of initiation is short - from minutes to the time required for passing of a cycle of cell division. In this stage, a normal cell turns into a neoplastic, most likely through cell mutation, without excluding the participation of epigenetic mechanism. As a result, yielding the so-called latent or dormant tumor cells. Stage of promotion is long - from weeks to two years. During this stage the latent tumor cell begins to divide and eventually leads to the appearance of tumors. Promoters in most cases realize its effect at two levels - by influencing the regulatory genes via interaction with cell membranes, i.e. their action is paragenetic. Known combination of agents that cause the two successive stages, and a biological model is the one with urethane as the initiator, croton oil - promoter and the mouse skin as a system for developing the effect - skin tumors. As initiators in animal experiments have used many chemicals (carcinogens) - polycyclic aromatic compounds aminoazo compounds, aflatoxins, aromatic amines, nitrosamines, and as promoters - surfactants, phenols, bile acids, DDT, phenobarbital, saccharine and the like.

Two stages not the only mechanism for the development of carcinogenesis. In many cases, this occurs after contact with an agent. This is a characteristic of exposure to sufficiently high doses of chemicals - strong carcinogens, as well as with ionizing radiation, possibly with oncogenic viruses. In some production conditions this mechanism of carcinogenesis can be leading, while in pollution of ecosystems to chemical pollutants and unhealthy lifestyles, leading can be combined action of the initiators and promoters.

Carcinogenesis is not a one-way process. It is possible reversibility of every stage of its development before the emergence of a malignant tumor. This regression is observed in both experiments, and in humans in a clinical setting and is the more common as more initial stages of carcinogenesis is. Rare cases of reverse development of already occurring malignant tumors have no relevance.

9.3. DOSE AND HEALTH RISK

Carcinogenic effects are dose-related, such as: 1) the higher doses give a higher percentage of tumorigenesis and decrease latency; 2) repeated multiple smaller doses, regardless of the timing of the different exposures lead more often to tumorigenesis compared to a single (or a few) large dose; 3) a combination of several carcinogenic factors more

frequently causes on the target tissue the synergistic or additive effect (less antagonistic action); 4) any exposure to carcinogenic creates a risk of cancer for all the life.

For many toxic action substances is a threshold dose below which there are no adverse changes (ie. non-stochastic effect).

When factors of carcinogenic effect is difficult to determine whether there is a threshold dose - it is assumed that they have stochastic effects on linear (non-threshold) depending dose - response. A number of arguments, however, are held against the concept of non-threshold carcinogen - eg. agents may react with other cellular nucleophiles (proteins, non-critical segments of DNA); be rapidly metabolized when passing through the liver; occur off of mutations in the DNA or destruction of the neoplastic cell by the immunogenic mechanisms, prior to the occurrence of tumor cloning. Also, for cancer to occur under the influence of a number of carcinogens is necessary to have previously tissue damage (hyperplasia, necrosis), which is the threshold effect - i.e. then there is a threshold for an initial toxicity below which there was no damage, it should have and the threshold for secondary formation tumors. These arguments, however, are not generally accepted because they can not prove accurate in experiments on animals and studies in humans.

Regardless of whether there are threshold doses or not, today there is much evidence of dose-response effects of carcinogens - eg., that large doses lead to a greater probability of developing tumors; to a higher incidence of mortality and a shorter latency period compared to lower doses or concentrations. In this connection, a number of authors consider that the threshold concentrations of chemical carcinogens can be established. For this purpose comes in experiments with a variety of experimental animals, in a variety of ways of introducing and gradually decreasing the dose to establish the doses or concentrations that do not cause tumors and pre-tumor proliferation during the life of the experimental animals; also identified in the experiment noblastomogenic doses to be compared with the results of the quantification of carcinogens in the environment in which they live or work certain population groups, as well as the frequency of relevant cancers in them.

Transfer of results of animal experiments on humans is quite naturally taking into account a series of factors - eg. the body mass of the various organs of animals and humans, their physiological and biochemical characteristics, etc. It is recommended that the use of various mathematical models and appropriate formula. After all the calculations is required, as in toxic substances the introduction of ratio of safety, but with larger values - 100 and more.

9.4. MODIFYING FACTORS

Carcinogens do not operate in isolation on biological systems. Many of on biological factors (genetic, immune, sexual, age, nutrition, enzyme induction) can have a stimulating or inhibiting effect on carcinogenesis.

Species and genetic specificity. Some species are relatively resistant or more susceptible to the effects of certain carcinogens. These differences are most often a result of certain genetically determined properties of the type - eg. in underdeveloped sebaceous glands of the skin, thereby hindering the penetration of the carcinogen; inability to induce the MFO, due to not forming carcinogenic metabolites, etc.

A number of tumor localizations can be observed more frequently in blood relatives (brothers and sisters). These are cancers of the breast, colon, bladder, kidneys, lungs, prostate gland, stomach; melanomas and some sarcomas. The risk of developing some of these tumors in blood relatives is 3-4 times higher than that seen in control families or among the general population of the inhabitants. This is explained with genetically determined metabolic competence of the hepatocytes and the cells of other organs to metabolizable fallen into the body chemical substances, wherein the active carcinogens degrade to harmless products, or vice versa, from the inactive chemical substances are formed intermediate and final carcinogens.

In connection with this modification of carcinogenesis are important factors that **induce enzyme systems** associated with metabolic processes of carcinogens. Here are some essential polycyclic aromatic compounds (methylholantren), organochlorine pesticides (lindane), food (spinach, cabbage, salads), drugs (luminal), smoking and chronic consumption of alcohol.

Experimental established a number of inhibitors of carcinogenesis contained in fruits and vegetables, cereals and legumes, such as vitamins A, E, C, PP, B_2, carotenoids, indoles, coumarins, aromatic isothiocyanates, flavones, plant sterols, protease inhibitors.

Gender differences. Different sensitivities of sexes to carcinogens can be explained by differences in hormonal profile, anatomical structure of some tissues and organs and their physiological activity. It is known for example that estrogens stimulate and testestosterone inhibit carcinogenicity of chemical substances, MT-virus, ionizing radiation in tumorigenesis of breast cancer.

Age differences. Experimental and epidemiological studies have shown that adolescents individuals are much more favorable terrain for the course of carcinogenesis in action of carcinogenic factors. Most likely, this is due to high proliferative activity of tissues, the relative immaturity of the immunological defense of the organism, of the immaturity of certain structures and enzyme systems.

9.5. CARCINOGENS IN PRODUCTION

Professional chemical carcinogens are major sources of air pollution, food, water and soil. Initial assumptions about the low incidence of occupational cancer of 1-3% were not compliant with such factors as the prolonged latent period and synergies in the combined action of some carcinogens. Now, according to some authors 20-40% of cancers are or will be in the coming decades professionally conditioned **(Table. 1).**

Data from epidemiological studies indicate the presence of an increased risk for occupational exposure to inorganic and organic compounds **(Table. 2)**:

Asbestos. In workers of asbestos productions register an increased incidence of lung cancer and mesothelioma. It is noted that asbestos is over 3,000 different applications in industry and households. High concentrations of asbestos in the environment are found around manufacturing industry and enterprises. It is experimentally demonstrated that the incidence of tumors is influenced by the shape and dimensions of the fibers. Most are carcinogenic fibers with a diameter less than 3 μm and a length below 5 μm. Established a synergy when combined action of asbestos and smoking.

Arsenic and arsenic compounds. In miners involved in the extraction of gold, cobalt and copper ores, that are high in inorganic arsenic compounds is detected an increased risk of lung cancer. The risk of lung cancer is greatly increased in the metals refineries, where workers inhale high concentrations of arsenic **(Table. 3)**. In literature there are not a few reports of skin and lung cancer in growers used arsen containing insecticides.

Chromium and chromium compounds. In the workers involved in the production of chromates, there was about a 20 times higher risk of developing lung cancer compared to the corresponding control groups. In experimental studies have induced tumors in laboratory animals after treatment with various chromium compounds (calcium, strontium and zinc chromate).

Nickel and nickel compounds. Cancer mortality in the nasal cavity at workers in nickel refineries is 150 times higher, and lung cancer 5 times higher than in the general population. In these workers is increased and the risk of cancer of the larynx and stomach, as well as various sarcomas. The risk now associated mainly with the powdering of nickel ores. For carcinogens adopt nickel sulphide, nickel oxide and carbonate. Intramuscular administration of these substances to rats leads to the development of fibrosarcomas and by inhalation - tumors of the lungs.

Tab. 1. Productions in which the possible development of cancer (in M.L. Fischman et al., 1995)

Production	Probable or suspected agent	Target organ
Production of aluminum	Polycyclic aromatic hydrocarbons	Lung, bladder
Production of auramin	Auramin	Bladder
Manufacture and repair of footwear (some professions)	Benzene	Leukemia
Gasification (coal)	Polycyclic aromatic hydrocarbons	Lung, bladder, skin, scrotum
Production of coke	Polycyclic aromatic hydrocarbons	Lung, ?kidney
Furniture production	Wood dust	Nasal cavity (mainly adenosarcomas)
Casting of iron and steel	?Polycyclic aromatic hydrocarbons, silicon, metal aerosols	Lung
Production of isopropyl alcohol	Diisopropyl sulfate, isopropyl oils	Paranasal sinuses, ?Larynx
Production of fuchsin	Fuchsin, ?precursors (incl. Ortotoluidin)	Bladder
Purification of nickel	Nickel oxides, nickel subsulfid	Nasal cavity, lung, larynx
Rubber industry	Aromatic amines, ?solvents	Bladder, leukemia, stomach, lung, skin, colon, prostate, lymphoma
Underground mining of hematite (exposure to radon)	?Radon	Lung

Tab. 2. Production carcinogens (by Encyclopaedia of Occup. Health and Safety, 1998)

Carcinogen	**Target organ**
4-aminobiphenyl	Bladder
Arsenic and its compounds *	Lung, skin, ?liver angiosarcoma
Asbestos	Pleura and peritoneum (mesothelioma), lung, ?larynx, gastrointestinal tract, kidneys
Benzene	Leukemia
Benzidine	Bladder
Beryllium and its compounds	Lung
Bis (chloromethyl) ether **	Lung
Vinyl chloride	Liver (angiosarcoma),? brain, lung
Coal tars and resins	Skin, scrotum, lung? bladder
Wood dust	Nasal cavity
Ethylene oxide	Leukemia
Mustard gas	Larynx, lung
Ionizing radiation	Leukemia, skin and others.
Cadmium and its compounds	Lung
Mineral oils, raw or slightly processed	Skin, scrotum,? lung
β-naphthylamine	Bladder
Nickel and its compounds (? Oxide and sulfide)	Lung, nasal cavity
Radium	Bones (sarcomas)
Radon	Lung
Soot, tar *	Skin, lung,? bladder
Talc containing fibers azbestformeni	Lung? mesothelioma
Ultraviolet radiation	Skin
Chromium (VI) compounds *	Lung, nasal cavity

* Compounds with a carcinogenic effect in humans are not well defined
** And technical chloromethyl ether which contains 1-8% bis (chloromethyl) ether

Tab. 3. Carcinogenic risk in the copper production - CP (by Tzolova S., 1995)

CP	Type of study	Number of persons	Follow-up period	Exposure	Common relative risk (%)
Tacoma, USA	Cohort	525	1949-1973	AsY	304.8*
- " -	- " -	2802	1940-1976	AsY	194.9*
- " -	- " -	- " -	- " -	AsA	- " -
Anaconda, USA	- " -	8047	1938-1963	AsA	329*
- " -	- " -	8045	1938-1977	AsA	- **
- " -	- " -	1800	1938-1979	AsA	-
Garfield, USA	Proportional mortality	244 died	1959-1969	-	481*
Ooita, Japan	Cohort	2675	1949-1971	-	-
Sweden	- " -	3916	1928-1981	AsA	372*
- " -	- " -	3710	1958-1982	-	232*
- " -	Case-control	29 dead of lung cancer	1960-1976	AsA	510*
9 workshop copper electrolysis USA	Cohort	4802	1946-1975	-	97

* $p<0.05$ ** Relative risk in contingent no-estimated
AsY - Arsenic in urine; AsA - Arsenic in air

Aromatic amines. Of aromatic amines with underlighted carcinogenic properties to induce bladder tumors are β-naphthyl amine and benzidine. Epidemiological studies have found that the highest risk in workers exposed to β-naphthylamine - 61 times more than in the whole male population and benzidine - by 19 times. Carcinogenic amines are applied in cable production, the paper industry, in the production of pigments and dyes. With increased carcinogenic risk and are working with benzidine in publishing laboratories.

Heterocyclic and polycyclic aromatic hydrocarbons in soot, coal tar and mineral oils. A number of experimental studies have shown that contact with soot, coal tar, creosote, shale oils, asphalt, smoke that is produced by burning organic matter, carries a high risk of developing skin cancer (cancer of the scrotum in chimney sweeps, and workers exposed on tar and mineral oils), and in fewer degrees of lung cancer, bladder and digestive system. These substances are produced experimental tumors in many animal species of both sexes of almost all organs and systems. Polycyclic aromatic hydrocarbons exhibit strong carcinogenic and for cells and organs, cultured in vitro.

Vinyl chloride. Epidemiological studies show that exposure of workers to vinyl chloride increases the carcinogenic risk of developing angiosarcoma of the liver, brain tumors, lung cancer, chemo- and limphopoetic system. Following oral or inhalative treatment of experimental animals with vinyl chloride are observed most frequently angiosarcoma of the liver.

Benzene. When workers having prolonged contact with benzene establishes an increased risk of acute no-lymphocytic leukemia. Relatively few however, successful attempts to induce tumors with benzene in an experiment with animals.

Several studies have established an increased risk of cancer of the paranasal sinuses in the wood-processing and furniture industry, production of shoes. The increased cancer risk associated with release of large amounts of **wood dust** in the wood-processing industry and the use of several **chemicals** in other industries that are carcinogenic to humans and experimental animals (chromium and chromium salts, arsenic and arsenic compounds, asbestos, benzene, mineral oils, tars, isopropyl alcohol, etc.).

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10 FACTORS ACTING ON THE IMMUNE SYSTEM

10.1. IMMUNE SYSTEM

Object of the immune system is to protect the living body from external aggressors (bacteria, viruses, fungi, nutrients, chemicals) through the construction of a protective immune response against foreign macromolecules (called antigens). Large biological macromolecules are natural antigens, while smaller molecules can not induce an immune response if they are not associated with a carrier (usually proteins) - haptens.

Immune competence has two main components:

1. Humoral immunity - immunoglobulins, antigen-specific antibodies, B lymphocytes
2. Cellular immunity - T lymphocytes, subpopulations of their, T-cell recognition, lymphokines, T-B cell cooperation and regulation of immune response.

Immune defenses of higher organisms is done on two levels:

a) antigen nonspecific, primary implemented by polymorphic-nuclear leukocytes, macrophages, natural antibodies, opsonization and phagocytosis

b) antigen-specific - characterized antigen recognition, primary or secondary immune response

All of these, called immunocompetent cells derived from pluripotent stem cells. A portion of the precursor stem cells are "brought up" in the thymus and are transformed into T-lymphocytes, which are present in thymus-dependent areas of spleen, and lymph nodes and constitute 60-85% of peripheral blood lymphocytes. Precursor B cells mature under the influence of the fetal liver and bone marrow and make up 7-23% of human lymphocytes. Genetically defined cell surface markers allow you to identify and distinguish lymphocyte types. Stem cells are also precursors of the monocyte-macrophage and polymorphs-nuclear lines of cells that play an important role as in non-specific and like wise in antigen-specific immune defense.

Lymphocytes can be activated, either directly by the antigen or indirectly by macrophages (via interleukin-1, IL-1) which were exposed to the antigen. The latter normally are destroyed by macrophages, but a small part of them is processed and presented to the lymphocytes which are activated. T-lymphocytes bear on their surface receptors, through which recognize foreign materials. Activation of B-and T lymphocytes by clonal proliferation leads to amplification of the immune response. Upon stimulation part of T-lymphocytes differentiate into cytotoxic T-lymphocytes, while others respond by formation of soluble mediators, termed lymphokines, possessing an extremely broad spectrum of activity. Most of the newly immunocompetent cells involved in the development of an immune response, but some of them remain "dormant" as memory cells. Upon re-exposure to the specific antigen, the memory cells are activated and generate a large number of responsive cells (via IL-2), leading to intense immune response.

The selectivity of the humoral response against foreign molecules or pathogens is mediated by antibodies. Antibodies are gamma globulins that identify foreign antigens and activate certain protective systems of the body. They consist of heavy and light chains making up the functional domains and are formed by plasma cells. There are five classes of immunoglobulins (IgG, IgA, IgM, IgE, IgD) each of which has a specific heavy chain **(Table. 1)**.

Despite large differences in the nature of the humoral and cellular immune response, various

Tab. 1. Human immunoglobulin classes

Type	Mean serum concentration (mg/dl)	Molecular weight	Half-life in plasma (days)	Biological function
IgG	1200	150 000	23	Fix complement precipitin
IgA	280	160 000 550 000	6	Secretory antibodies, anti-virus protection
IgM	120	900 000	5	Fix complement, agglutinins
IgD	3	180 000	3	In lymphocyte receptor in newborns
IgE	0.03	190 000	2.5	Reagins antibodies

cell types cooperate with each other and through complex mechanisms set and the two arms of the immune response. Certain subpopulations of T-lymphocytes (T-helper and T-suppressor) exert a positive and negative control and on B- and on T-lymphocyte function. Other lymphocytic cells called natural killer (NK cells) participate in non-specific defense of the body and are potentiallycy cytotoxic for various malignancies and virus-infected cells, and another subpopulation T cells involved in antibody-dependent cell-mediated cytotoxicity. Furthermore, in the effector phase of the immune impairment involved and other components (biological systems, cells, etc.): complement, prostaglandins, cyclic nucleotides, eosinophils, platelets, hormones, neuro-endocrine links, psychological stress, which determines the complexity of the immune response.

10.2. BASIC MECHANISMS OF IMMUNE DAMAGE

Currently defined at least 4 major types of immune responses conditioned tissue damage, depending on the effector mechanisms. In many diseases, however, operate more than one effector immune mechanism.

Type I - quick type hypersensitivity

Reaction develops in sensitized individuals immediately after contact with the antigen, most often in people with a history of IgE-mediated asthma or rhinitis (atopics). Fc-portion of the formed IgE binds to surface receptors on basophils and mast cells, resulting in the release of vasoactive mediators. Depending on the organ involved clinical manifestations are rhinitis, bronchospasm, urticaria, angioedema or hypotension (by this mechanism occurs occupational allergic asthma and rhinitis). By IgE-mediated mechanism than fast, immediate reactions can occur and late-phase reactions - 12-24 hours after the initial reaction, and are characteristic of occupational asthma when using detergents, in bakers and others.

The fast type hypersensitivity evidenced by skin tests with relevant allergens in vivo and immune tests which prove specific IgE in serum.

Type II - cytotoxic effects

In them a cell antigen is a protein or is attached to the cell surface. Low-molecular substances (eg. haptens) can induce the formation of IgG or IgM antibodies with anti-hapten specificity. Upon re-exposure on the cell surface to form antigen-antibody complexes. The destruction of target cells (mostly erythrocytes or pulmonary basic membranes) is realized by killer cells that have receptors for the Fc-portion of IgG, as well as activation of the complement cascade.

The demonstration of type II reaction going through the test of Coombs, who demonstrated IgG antibodies on the surface of red blood cells.

Type III - immunocomplex reactions

They occur when the binding of soluble antigen and free IgG and IgM antibodies in immune complexes, which leads to complement activation. May develop as local, Arthus reaction (immune complexes are formed in situ in the skin) and systemic serum sickness (circulating complexes are deposited in different tissues). The deposition of immune complexes leads to the development of the effects of chronic inflammation with the inclusion of the coagulation, complement and other systems, the secretion of vasoactive amines, cytokines, oxygen radicals and other factors and cells that lead to the death of the tissue and fibrosis.

The presence of antibodies of type III immune reactions can be demonstrated through the establishment of circulating immune complexes and complement activation in the conventional way.

Type IV - cellular immune responses

This type of immune response, also called delayed-type hypersensitivity, occurs when the antigen binds to the sensitized T lymphocytes and form a series of cytokines. It does not require humoral antibodies and complement. Located in skin Langerhans cells absorb and processing fall antigen and present it to the CD4 + T lymphocytes. Upon repeated exposure, they are activated and secrete lymphokines. Lymphokines exert its effect mainly in the neighborhood surrounding environment by enhancing a number of biological activities, but their main function is to attract and activate macrophages and new immune cells. Clinically occurs most often as contact dermatitis. At 24-48 hr immune infiltrate manifests itself as erythema, induration and itchy, and sometimes vesicles and skin necrosis.

Demonstration of delayed type hypersensitivity in vivo done by skin test by standardized antigen.

10.3. HEALTH EFFECTS OF IMMUNOTOXICITY EFFECTS

Known and relatively well known are as innate, genetically determined T-, B- or combined immunodeficiency, and diseases associated with acquired immunodeficiency. Possible health effects of immunotoxicity effects can be related both to suppress or enhance the normal immune response, and the development of aberrant one. The most typical clinical manifestations of abnormal immune response can be summarized as follows:

1. Suppressed resistance to infections.

Several harmful environmental factors can lead to suppression of the immune response: ionizing radiation, metals (lead, cadmium, cobalt, beryllium, gold), polychlorinated biphenyls, chemotherapeutic and immunosuppressive drugs. They may be affected various components of the immune system:

- immunodeficiency of T-cell subpopulations - mainly associated with severe viral and opportunistic infections due to intracellular pathogens and fungi (chicken pox, tuberculosis, listeriosis, aspergillosis, etc.).
- deficient in B-cellular functions - most often associated with severe bacterial infections (streptococcus, Klebsiella pneumoniae, Neisseria, etc.)
- impaired phagocytosis and intracellular killing - may manifest as recurrent purulent infection, chronic skin disease
- deficiency of the complement system - manifested by recurrent infections with streptococci, Haemophilus influenzae, Neisseria meningitis

2. More frequent occurrence of carcinomas.

Congenital immunodeficiency, such as Ataxia telangestasis, syndrome Wiskott-Aldrich and severe acquired immunodeficiency diseases are associated with a high incidence of lymphoreticular malignancy, Kaposi's sarcoma and others. Prolonged treatment with immunosuppressants on transplant patients or patients with autoimmune diseases is also associated with a higher incidence of malignancy. In cancer patients after prolonged chemo- or radiation therapy occurs more frequently second malignancies such as leukemia, macroglobulinemia, myeloma, another cancer, associated probably with immunodepression.

3. Emergence of autoimmune diseases.

Currently the emergence of autoimmunity as a consequence of immunotoxicity effects is quite debatable. Described are a number of drug-induced autoimmune diseases-lupus-similar syndrome, myasthenia gravis, autoimmune hemolytic anemia. There are many similarities between idiopathic and drug-induced autoimmune diseases, but there are significant differences between them. In the past there are two characteristic features: gradual onset - 3-6 months after exposure, and clear remission following termination of exposure to harmful agents.

Possible mechanisms of induction of autoimmune response of chemical hazards are: a) simple activation of latent disease or cross immune reactivity to chemical, b) directly imunopharmacological or immunotoxicity effects on the immune response - eg. in action of chlorpromazine, hydralazine, methyldopa, penicillamine, procainamide.

4. Hypersensitivity reactions.

Well known that most of the immunomodulating agents may also cause hypersensitivity reactions. Clinically, the most common are fatigue and febrile conditions and are the expression of a pharmacologically immunomodulation associated with activation of macrophages and release of interleukins IL-1 and TNF. Chemical immunostimulation can also increase the risk of a true allergic diseases, regardless of the allergen. There is evidence for increased production of IgE antibodies in the action of chemicals and drugs. Each of 4 effector mechanisms of immune damage (but most likely in combination with others) may underlie of clinical-proven allergic reactions as a result of toxic pollutants in working and environmental surroundings.

5. Altered hepatic metabolism.

Inhibition or stimulation of liver enzymes from harmful chemicals or immunomodulatory therapeutic agents leads to distortion in the normal biotransformation in the cells, which may lead to an accumulation of active metabolites, abnormal toxicity and interleukin production. For similar mechanisms for example affect phenobarbital and polycyclic aromatic hydrocarbons that damage cytochrome P450 monooxygenase functions. Immunostimulation is also a factor that can modulate hepatic enzymatic metabolisation of chemicals.

10.4. ALLERGENS CAUSING PROFESSIONALLY-DEPENDENT SENSITIZATION

Professional allergen is any substance present exclusively or predominantly in the workplace, which can cause by immune or non-immune mechanisms hypersensitivity reaction in exposed workers. With the development and introduction of new technologies is constantly increasing and the number of potential allergens.

Plant allergens. Herbal ingredients, oils, resins and juices can cause asthma, rhinitis, urticaria or contact dermatitis. Pollen from grasses and trees can sensitize agronomists, florists grain-producers, gardeners. Known role of gluten - a common antigen for various types of flour (wheat, corn, barley) for occupational asthma among millers, bakers, confectioners. Inhalation of wood dust from oak, pine, beech and some exotic trees - mahogany, teak, western red cedar and others can cause occupational allergic diseases. Some medicinal plants - primula, valerian, aloe, lime, causing the occurrence of bronchial asthma in pharmacists and herbalists. Castor seed, oil plants cake, tobacco, soya flour, cotton, flax, hemp, jute, dust from raw coffee have similar effects

on exposed them workers (interesting is that during firing of green coffee are destroyed macromolecular substances, play a role allergens).

When workers in the textile industries inhalation of dust from cotton, linen or hemp causes so-called byssinosis. The pathogenesis of the disease is not yet fully understood, but it is assumed that develops histaminoliberation without prior sensitization of inhaled dust.

Animal antigens. By epidermal waste (dandruff and hair) come epialergens. They include dandruff and hair of man and animals, feathers and down of birds, fish scales. Secretions, mucus, urine, feces, insect venoms may also be allergens. Endangered professions are those of veterinarians, breeders, workers in vivariums, zoologists, poultry-farmers. Ingredients of cuticle (artropodin), haemolympha, excrement (proteins and glycoproteins) a number of parasites - ticks (home, flour), insects (corn beetle), moths (wheat moth) and sericin - protein cover fibers of silk, are also professional allergens. Allergenicity of sheep wool and unrest dust increases during their storage in humid and warm rooms due to the development of fungal and bacterial microflora. Pearl dust causes sensitization and asthma among jewelers.

Bacteria, fungi and their spores. Most important for professional pulmonary pathology as antigens are thermophilic actinomycetes, enzymes of Bacilus subtilis and fungi - Alternaria, Penicillum, Aspergilus and others. Risk are workers employed in the production of wood flour, leather, detergents, cheese, beer, mushrooms and more. Inhalation of allergens causes hipersensitiv pneumonitis.

Chemical allergens. A huge number of organic and inorganic substances can cause occupational sensitization. Of organic matter allergenicity have gasoline, naphtha, chloramin, formaldehyde, acrolein, trichloroethylene, benzene, phenol and derivatives, mineral oils, coal tar, aniline, organic dyes used in the textile industry (indigo-, azo-, nitro- and nitrozo dyes) , epoxy resins, etc. Ursol and in particular derivative hinondiamin causes symptoms of respiratory allergy in working with hair dyes. Pesticides, especially chlor- and organophosphorus also have allergenic potential. In many professions - workers in the chemical industry, painters, bookbinders, producers of shoes and handbags, workers in woodworking and furniture production risk of sensitization in contact with lacquers, paints and adhesives. In the production of plastics etiologic factors for the occurrence of occupational allergy can be used monomers, accelerators, plasticizers and colorants.

Metals. Metals and their salts are important etiologic factor for allergic conditions. Working with nickel is one of the most common causes of contact dermatitis. Allergenicity have also chromium, platinum. It is believed that the metals and the salts act as haptens, causing subsequently immune response.

Lawrence (1981) graded heavy metals according immunotoxic properties as follows: mercury > copper > manganese > cobalt > cadmium > chromium. According to him, zinc and iron showed no significant effects, while lead and nickel, lead to an increase in antibody-forming cells.

The metals can affect the immune system in several directions: to influence natural immune resistance (antigen - nonspecific immune response); cause immunosuppression; to connect to its own proteins and make them autoantigens (gold- and mercury induced nephritis in humans and animals); to induce reaction of respiratory hypersensitivity (chromium and development of asthma in atopics).

Occupational lead exposure shows that the main immunotoxicity effects are most likely associated with proliferative ability of lymphocytes (amplified) without major changes in humoral immune indicators (except for a reduction in secretory IgA in saliva).

The mechanisms by which heavy metals exert their immunomodulatory effects are noth entirely clear. It is generally accepted that metalothioneins - small, cysteine-rich proteins (6-8 kDa) which bind heavy metals have Immunosuppressive properties.

Drug allergy. Professional are exposed workers human and veterinary medicine, pharmacists, workers in the chemical and pharmaceutical plants. The most common allergens are antibiotics, anesthetics, sulfonamides, analgesics, corticosteroids, neuroleptics and others. The mechanisms of sensitization to drugs can be immune or pseudo- and non-immune and clinical manifestations - local or generalized, systemic.

Food additives and impurities are common allergens - the different colors of food (eg. tartrazine), preservative (benzoic acid, sodium bisulfite, acetylsalicylic acid), antioxidants (ascorbic acid, lactase, salt of citric acid), impurities (metals, pesticides, ingredients of plastics, detergents, etc.).

The work environment various antigens are found in various combinations with one another and with the physical factors, impacting on the body complex. The induced immune response can be carried out in more than one immune mechanism.

10.5. IMMUNO-DEPENDENT OCCUPATIONAL DISEASES

Most common occupational diseases in the pathogenesis of immune mechanisms involved are allergic asthma and rhinitis, hypersensitivity pneu-

monitis and contact dermatitis. Their development depends on the individual predisposition, the type of allergen, and the extent of exposure.

Allergic asthma and allergic rhinitis are some of the manifestations of I-type IgE-mediated allergy. Generally, genetically predisposed individuals (atopics) are prone to sensitization by inhalation of allergens of high molecular weight (proteins), while the inhalation of small molecule antigens, no-connection with the presence of atopy in the individual and sensitization. Still not fully understood the mechanisms of pathogenesis of non-allergic (non-immunological) asthma.

Hypersensitivity pneumonitis were known in 1713, were described by Ramazzini as pneumonia-like illnesses occurring after contact with powders in some productions - i.e. as occupational diseases. Subsequently, they are disclosed various forms - farmer's lung, bagasoza disease, cheese producers disease, lung of mushrooms, etc.

Hypersensitivity pneumonitis is an allergic disease of the lung parenchyma by inflammation of the alveoli and the interstitial spaces, induced immunologically from acute or chronic inhalation of various organic powders or of occur in these bacteria or fungi (thermophilic actinomycetes), as and animal products (dried excrement). The allergen enters as particles that do not exceed 3 μm, and reaches the alveoli in normal breathing.

Many of the allergens associated with hypersensitivity pneumonitis, except immunogenicity possess other biological properties relevant to pathogenesis. Such are the adjuvant properties, which is due to additional non-specific activation of macrophages and non-immunological activation of the alternative complement pathway.

A long time as a major pathogenetic mechanism of hypersensitivity pneumonitis was assumed III-type - reaction of Arthus, caused by immune complexes of inhaled allergen by precipitating IgG antibodies and subsequent complement activation. Evidence in this direction are found in more than 90% of patients antigen - specific precipitating antibodies. However, in 50% of persons with similar exposure and without clinical symptoms were also detected precipitating antibodies, suggesting that these antibodies are associated with the effects (exposure) to the allergen, and not necessarily by the presence of lung disease.

There is also an opinion that the disease is mediated predominantly by IV-type - T-lymphocyte effector mechanisms. In support of this are histological studies showing the infiltration of lymphocytes, neutrophils and macrophages, interstitial fibrosis, granulomas. Experimental studies in animals also confirm the assumption that the primary pathogenetic mechanism engages the effector T-cells.

Clinical manifestations, the latest experiments conducted on animals, epidemiological data and research samples from bronchoalveolar lavage showed a complex mechanism of disease with involvement of specific cell-mediated immunity, and nonspecific biological effects of inhaled material and only a small role (if can speak of such), circulating antibodies.

Contact dermatitis is erythematous skin disease, classic manifestation of cell-mediated immunity, wherein the main pathogenetic role is played by sensitized T-lymphocytes. It is assumed direct contact of the allergen with the skin. There are numerous allergens leading to disease - metals (chromium, nickel, cobalt), and organic compounds - ingredients in cosmetics, detergents (enzyme preparations), preservatives (formalin), cutting oils, drugs, used in the rubber industry as auxiliary materials accelerators, anti-oxidants, vulcanizators, stabilizers. The majority of them are haptens, but the source and nature of the vectors in the skin are not known.

As a special case here is regarded **photoallergy** - manifestation of cell-mediated hypersensitivity to some substances which are converted into immunogens under the action of sunlight. Photosensitising considered as being phototoxic reaction, associated with changes in pyrimidine bases of DNA or changes in its double helix. It is known photosensitizing effect of some medications - sulfonamides, phenothiazines (endogenous activity) and phototoxic effect of coal tar, tar and derivatives (exogenous action).

Unlike the typical effects of some occupational hazards and drugs on the immune system, relationship immune response- harmful substances from the environment are unclear. It was found that the total load of antigens from the environment suppresses the immune system leading to an immune abnormalities, multisystemic and psychological diseases. Typical symptoms are impaired self-esteem, chronic fatigue and neurotic symptoms. Harmful agents of the environment include exhaust from road transport, smoke, perfumes, solvents, formaldehyde, chlorine and fluorine compounds in drinking water, chemical additives to foods and more. No exact mechanisms of damage to the immune system by these agents, but it is the role of IgE, immune complexes, and other yet unidentified mechanisms.

10.6. DIAGNOSIS OF IMMUNE IMPAIRMENT

Study of immune changes in humans exposed to potentially harmful agents is much more complicat-

ed compared to that in the experimental animals. It is necessary to comply difficulties as logistical problems, appropriate controls, magnitude and nature of exposure, drug abuse, illness.

The human population is heterogeneous and genetically different and individual variation affects on results. People are exposed continuously to stimuli from the environment, and their ability to meet the foreign materials is supported by immunological memory. For some infectious microorganisms, eg., the immune response, however, does not lead to their complete elimination, and more likely to their "soft" integration into the genome (Herpes, cytomegalovirus, Epstein-Bar viruses, etc.).

Essential are limitations of methodological approaches to the study of people. Applied noninvasive methods carry less information, and methodology established by WHO to study the immune response was built to demonstrate abnormalities of the immune system in prominent medical conditions - immunodeficiencies and more discreet departures are difficult ascertainable. For some immune parameters are essential and age, gender, additional disease therapy - factors which, if not comply can be a source of misinterpretation of results.

Obtaining usable data for immunotoxicity effects of some environmental factor and its use for risk characterization begins with initial epidemiological study. The most common model is a cross-section study in which exposure and disease state (in this case a change in immune function) are measured once or within a short period. Immune function of "exposed" individuals was compared with that of unexposed, appropriately selected group.

Actual laboratory studies of the immune response are usually selected standard and are aimed at characterizing the overall immune competence (first stage) and to characterize specific immune response to toxic materials and mechanisms of occurrence of aberrant immune response (subsequent stages). There are some differences in the combinations of methods applied in different countries, such studies are combined in one-moment scheme or sequential-deepening order. On **Table. 2** is a combination of methods recommended by the World Health Organization (EHC 180).

Tests for the study of immune competence

After detailed and focused history and initial studies of: blood (blood count), pulmonary function tests (FEV1, FVC, EFF_{25-75}, PEFR) or provocative samples (eg. with methacholine) is directed to proceed laboratory testing of the immune response.

Skin tests. Scarification and intradermal skin tests are useful in identifying sensitization to number of inhaled protein antigens such as ticks, dust mites, feathers, pollen and extracts of suspected high-molecular-weight antigens of working environment. Low-molecular materials usually do not give positive rapid skin response until are no-conjugated with carrying protein- ex. serum albumin. The initial positive reaction from a redness and vesicles may be followed after 4-6 hours of IgE-mediated late response, expressed by erythema, induration, pruritus on application site.

Testing by patches. Patches used in the proof of contact skin sensitization. The test was performed with standard impregnated with antigen patches, which are applied to the skin and results were read after 24 h. A positive reaction consists of erythema, induration, and in some cases and a vesicle. In addition to standard commercial kits comprising a set antigens (American Academy of Dermatology) in the same manner can be applied and suspected harmful antigens from the environment.

Table 2. Methods recommended by WHO for the study of immunotoxicity

1. Blood cell counts and differential counts
2. Antibody-mediated immunity (one or more of the following)
- a primary antibody response to protein antigens (eg. epitope tagged flu vaccine)
- serum immunoglobulins IgG, IgA, IgM, IgE
- a secondary antibody response to a protein antigen (diphtheria, tetanus or poliomyelitis)
- natural immunity to blood group antigens (anti-A and anti-B)

3. Phenotypic analysis of lymphocytes by flow cytometry
- superficial analysis of CD3, CD4, CD8, CD20

4. Cellular immunity
- skin testing for delayed type hypersensitivity by Multitest Biomerieux R
- primary reaction of delayed-type against the protein (keyhole limpet haemocianin)
- proliferation by permitting antigens (secondary response)

5. Autoantibodies and inflammation
- C-reactive protein

Titers of autoantibodies against nuclear (ANA), DNK, mitochondrial antigens
- IgE against allergens

6. Measurement of non-specific immunity
- the number of NK cells (CD56 or CD60) or cytolytic activity against K562
- phagocytosis (NBT or chemiluminescence)

7. Clinical Chemistry

Examination of antibody-mediated immunity in vitro. The study of the humoral immune response include measurement of serum concentrations of immunoglobulins, antibody formation after immu-

nization and screening of natural immunity.

For the determination of serum total and alergenspecifically IgE antibodies are most popular radio-immunosorbent (RAST) and the enzyme-linked immunosorbent (ELISA) methods. In these, inert particles/surfaces are coated with antigen and incubated with serum. The formed antigen-antibody complexes were visualized by an anti-human conjugate with isotopes or enzymes. The latter are measured by suitable apparatuses-counters or spectrophotometers.

The common IgG, IgM and IgA antibodies in serum most often were measured by radial immunodiffusion in gel or turbidimetry. The demonstration of precipitating IgG can be done by test Ouchterlony - cross-diffusion in gel, where antigens and antibodies from serum diffuse against each other and form a precipitation line. This test is useful to define the III type immune responses.

Tests for cellular immune response. Various tests in vivo and in vitro are used for the study of cellular immunity: number of T- lymphocytes and their subpopulations, cutaneous delayed hypersensitivity, lymphocyte proliferation in vitro and blast transformation under the influence of antigens and mitogens.

The modern approach to determine the number of lymphocytes and their subpopulations, as well as some of their functions is by flow cytometry. Using specific monoclonal antibodies against different characteristic epitopes of a cell surface, associated with fluorescent substances, labeled mononuclear cells from peripheral blood and account their % or absolute number of an automated flow cytometer. Deviations in the number of cells or in their ratio may be associated with defects in their ability to recognize antigens and the regulation of T-cell function, leading to immune incompetence or autoimmunity.

Delayed type hypersensitivity indicates the ability of the individual to manifest pre-built T-cell response and in vivo was examined by cutaneous application of antigens which normally induce such a response. Is used to demonstrate T-cell-mediated immune deficiency (i.e. performed with a commercial kit Multitest R, Institute Merieux, Lyon, France, containing 7 standardized antigen, positive and negative controls, placed in a special applicator). The results were evaluated after 48 h by measuring the diameter of the hardening and calculating the sum (scoore).

The proliferation of mononuclear cells in addition to the authorizing (recall) antigens is an way for the study of delayed hypersensitivity in vitro and proof of type IV immune reactions as pathogenetic mechanism of immunotoxicity disabilities. Measure blast-transformation and newly synthesized DNA and the expression of some activation markers IL-2R (CD25), CD69, HLA-DR, etc.

Tests for the study of non-specific immune defense. Studied the number of so-called NK cells (by flow cytometry) or their cytolytic function through their ability to cell line K562. A significant role in non-specific immune defense play and polymorphonuclear leukocytes, whose function phagocytosis is frequent object of study. The activation status of neutrophils and platelets can be examined by flow cytometry with appropriate labeled antibodies: CD11b, CD14, CD16, CD54, CD63, etc.

The classical complement system is composed of 9 components (C1-C9) and a number of regulatory proteins. Many important biological activities in the inflammatory response and in natural resistance to infection are disclosed at various times of the classical and alternative pathway activation of the complement system. Laboratory testing of serum hemolytic complement activity (CH50) is a useful test, but similar and focused information can be obtained through the study of fragments of complement - C3, C4, C1 - inhibitor, C2 and others.

The classification of the immune response in predominantly **Th1 or Th2 type of cytokine secretion** (Mosmann & Coffman 1989) and modulation from harmful environmental opens new perspectives for the characterization of immune mechanisms by which damage occurs.

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10.7 IMPACT OF PHYSICAL FACTORS ON THE IMMUNE SYSTEM

Relatively scarce and conflicting data about the impact of physical factors on the immune system. The biological effects of physical factors are due to the direct impact on the cell and subcellular structures and by means of - by neurohumoral and neuroreflex mechanisms. Recent advances in the study of the interaction between the nervous and immune systems show bidirectional links between them through common receptors and biologically active substances, cytokines and neuropeptides.

NOISE is one of the most common environmental stressors and in working environment. Adverse effects on the immune system can be either by means of neuroendocrine disorders, and directly immunity. In both cases extremely important clinical implications are distortions in common to neuroendocrine and immune system mediators.

It is considered that the noise impact on the immune system manifests itself in the following biological effects:

1. Reduction of anti-infective immunity
2. To promote the development of autoimmune and allergic processes
3. Reduction of antitumor immunity.

Exposure to noise distorts the function of cell membranes and biological barriers, increasing their permeability, which leads to entry into the bloodstream of antigen-carring scars of another origin and opportunity for development of autoimmune response.

In experiments carried out on animals exposed to a short-time (30 min to 1 h) exposure to noise levels of 100 dB report decreased secretion of superoxide radicals from neutrophils and IL-1 from macrophages. Increased titers of serum thymulin, increase the weight of thymus and the number of thymocytes. In experiments with 3 weeks of exposure to noise, it was found that the tested immune parameters are changed in the direction of immunostimulation or immunosuppression, depending on the exposure duration: after 24 hours increased serum levels of IgM, decreases in the phagocytic activity and increased activity of NK-cells, after 7 days, a decrease of the proliferation ability of spleen lymphocytes upon stimulation with mitogens and NK-activity is increased: after 3 weeks increased slightly the proliferation ability of splenic lymphocytes and NK-activity reduced.

INFRASOUND AND ULTRASOUND. Scarce and conflicting data about the effects of infrasound on the immune system. Eg. experimentally it has been found that the effects are dependent on the levels of sound pressure - frequency infrasound with 8 Hz and sound pressure of 80 dB has an immunostimulating effect, and at 130 dB induce immunodeficiency.

It is known that the smaller the intensity of the ultrasound has a positive biological effect, stimulating the vital processes. Therapeutic applications have resorption, fibrinolytic, vegetonormaliziation, desensitization and immune stimulating effect of ultrasound. In conditions of experiment it was shown that exposure to low frequency ultrasound to stimulate the functional activity of macrophages and their metabolism, as well as the specific immune response.

Larger intensities have a destructive effect on the cell protoplasm. It highlights the importance of free radicals, that could mediate the production of yet unidentified organic radicals, also cause DNA damage found during exposure to ultrasound.

THE VIBRATIONS are a physical stressor that by afferent and efferent connections with the participation of the central nervous system and affects the immune system.

The assumption of a possible link between occupational exposure to vibrations and development of scleroderma was first expressed back in 1914 by Bramwell. Currently it has formed the hypothesis that the possible development of collagen and be professionally conditioned. Similar is the pathological picture of the vascular damage in vibration disease and scleroderma-rupture of the inner elastic membrane of the vessels with subsequent focal cell proliferation and regenerative formation of collagen and elastin. In addition, endothelial damage caused directly from the vibrational impact may cause platelet-adherence, activation and the release of mitogenic mediators - such for example platelet derived growth factor (PDGF), which enters the vessel wall and causes local smooth muscle proliferation.

Studies in the field of vascular pathology indicate the important role of leukocytes. Main role in micro-

vascular occlusion is attributable to polymorphonuclear leukocytes (PMN), which are the most numerous, considerably larger than lymphocytes.

In subjects with a phenomenon of "white fingers" (VWF) found subpopulation harder and harder deformable therefore activated leukocytes. It is considered that the vibration exposure causes local activation of leucocytes and increase their adhesion to the endothelium. The release of leukotrienes, cytokines and chemoattractants by damaged endothelium, and ischemic and "captured" PMN enhances the development of a pathological process by increasing the expression of adhesion molecules on endothelium and leukocytes, and further recruitment and activation of PMN - there is a vicious circle.

In support of the hypothesis of activation of leukocytes in vibration exposure is established increased production of leukotriene B4 and free radicals by PMN in patients with VWF. LTB4 is a potent chemotactic and aggregation mediator and a rise in plasma levels, in turn, further activated leukocytes.

In the latest research has explored the role and defined serum levels of intercellular adhesion molecule-1 (sICAM-1) and interleukin-8 (IL-8) in patients with VWF. Found a significant increase in the levels of sICAM and reduction of IL-8 in persons with VWF. Elevated levels of sICAM-1 reflect the increased leukocyte adherence and extravasation in the extracellular matrix which may contribute to the development of microcirculatory pathology. The reduction in levels of IL-8 in these patients probably reflects slow blood flow in the microcirculation.

It is known that the development of vibration disease, the severity and nature of its course may be different in people working under identical conditions. Perhaps it depends on biological, genetically determined characteristics of the organism determining unequal susceptibility to the effects of industrial vibration. Not exclude that the autoimmune component is basically a negative course of vibrational disease with the development of degenerative-dystrophic permanent damage. Susceptibility to immunodeficiencies and autosensibilization associated with antigenic determinants related to HLA-system. It has been found that the antigen HLA-DR5 may have a role as a prognostic marker indicative of increased risk of adverse course of vibration disease. Conversely, antigens HLA B35, B40 and DR2 perform protective function to the impact of industrial vibration.

ELECTROMAGNETIC FIELD (EMF). The accumulated data are still insufficient and contradictory, but identifying certain trends in the immune response depending on the type and duration of exposure and the type and intensity of exposure.

Quantitative assessment of lymphocyte subpopulations, defining their functional activity, of serum immunoglobulins, phagocytic indicators and antinuclear antibodies in professionally exposed to Ultra-High Frequency (UHF) electromagnetic fields. The majority of controlled indicators show dynamism and expressed phase character in its quantitative variation depending on length of service and the trend in the first five years is to increase.

Sets are an increase in IgM - primary immunoglobulin in the primary immune response, which gives base to assume the role of microwave electromagnetic fields as antigen non-specific immunostimulant.

There are changes in lymphocyte subpopulations, as total T-lymphocytes are reduced, and suppressor-cytotoxics - increased. There have been reports of generating an autoimmune response upon exposure of the test animals.

In studies carried out on peripheral blood mononuclear cells, exposed to extremely low frequency electromagnetic fields was an increase in production of IL-1 and IL-6 from monocytes. Express assumption is that it is possible monocytes are the target of these EMF.

Interest is the possible carcinogenic effects of EMF with frequency 50Hz, sources that can be power lines and household appliances. In the studies on isolated human lymphocytes is not apparent increase in the frequency of spontaneous emergence of "micronucleus" and genotoxic effect.

In booming fields of application of high-frequency EMF (3-30 MHz) and increasing the number of radio sites (mainly antenna systems of broadcasting stations) more people are exposed to their action. Scarce and conflicting data about the impact of these EMFs on the human immune system. Experiments on animals have shown: establishment leukopenia and lymphopenia with monocytopenia; increasing the number of basophils and reducing the number of neutrophils; There is a clear correlation between the effect and degree of the impact (field strength).

Higher voltages field (400V/m at 24 MHz) stimulate autoimmune reactions in the body and lower (200 V/m) induce delayed-type hypersensitivity.

IONIZING RADIATION. Studies were carried out on changes in humoral and cellular immune response upon exposure to ionizing radiation. Changes in the humoral immune response is characterized by: 1. extending between an antigenic stimulation and antibody production 2. reducing the rate of increase in serum antibody levels 3. reduction of their maximum concentration. The study of serum IgG, IgA and IgM in workers in uranium mines show

phase character and dynamics in its quantitative variation depending on the time of exposure. There is a suppression of cell-mediated immune response. Detected is a difference in sensitivity of different lymphocyte subpopulations, the most sensitive to the effects of ionizing radiation are CD 8 + suppressor T cells. Increased interrelation helper-suppressor (CD4/CD8) speaks for selective recovery of CD4 cells after irradiation in occupational exposure.

They found data of the role of immune changes for the occurrence of some diseases of the population in areas contaminated with radionuclides (Chernobyl accident). It is found interrelation of an autoimmune response to antigens of thyroid, microsomal and thyroglobulin to the observed thyroid hyperplasia. There are also antibodies to the antigens of the lens, which also implies involvement of autoimmune mechanisms in the development of cataracts in humans exposed to chronic effect to low doses of ionizing radiation.

ULTRAVIOLET RADIATION. Ultraviolet radiation affects human health in different ways. Acute reactions are skin erythema and photodermatoses and by eye-keratitis and conjunctivitis. In chronic exposure to UV radiation increases the risk of developing various types of skin cancers and cataracts. Damaging the immune system and DNA.

Experimental studies have shown that radiation in the UV-B range cause suppression of contact hypersensitivity reactions, reduction in the number and functional activity of the Langerhans cells in the skin, and changes in the distribution of lymphocyte subpopulations in peripheral blood. Repeated exposure of mice with radiation in the UV-B range induce immunological changes, leading to suppression of rejection induced by radiation in the UV-B range tumors in syngenic mice. The impact of the rays in the UV-B range and leads to suppression protection against of primary, induced by radiation in the UV-B range tumors in the skin of influenced animals. It is considered that the strongest immunosuppressive effect have the UV-rays in the range 260-270 nm.

They studied the effects of ultraviolet radiation on the immune system and activation of viral and other infectious diseases. Assumed probability of exposure to ultraviolet light to activate the HIV virus and to accelerate the onset of the disease.

TEMPERATURE. It is known that physical factors such as sunlight, heat, cold, mechanical injury can cause urticaria. These are 15-20% of its chronic forms.

The physical forms of urticaria are divided broadly into primary (IgE-mediated) and secondary. For reagin-mediated solar urticaria is assumed that antibodies react with modified or synthesized de novo antigens under the influence of the sun. The origin of antibodies of class IgG and IgM in cold urticaria are associated with the occurrence of cold-induced neoantigens (by exposure of new antigenic determinants or modified proteins).

ATMOSPHERIC PRESSURE. The conditions of reduced atmospheric pressure and hypoxia, cause marked changes in the immune system, increasing the amount of lymphocytes, monocytes, functional activity and the concentration of NK-cells, a reduction in CD4 + and CD8 + lymphocytes. It is assumed that NK-cells are particularly sensitive to hypoxia.

With prolonged stay under increased atmospheric pressure observed phase reaction in the functional activity of polymorphonuclear cells and monocytomacrophagic row - after initial increasing should decrease. There is also an increase in aberrant metaphases of lymphocytes.

The study of the mechanisms and processes, underlying the immune response, allowing to look for new approaches to assess the impact of physical factors on the body, and to take appropriate preventive measures.

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10.8 POSSIBILITIES FOR TREATMENT OF ADVERSE FACTORS BY ANTIOXIDANT SUPPLEMENTS

Number of environmental factors, physical, chemical, biological, social (stress), affect human health and have markedly deleterious effect on the immune system. This effect is the reduction of its own resilience to a variety of bacteria and viruses, the greater possibility of sensitization of the body, a predisposition to the development of autoimmune diseases and malignant. Numerous epidemiological and clinical studies prove the primary role of prophylactic nutrition to reduce the harmful effects of these factors and prevention of many chronic and degenerative diseases.

The increasing knowledge of the reactive forms of oxygen, oxidative stress and the role of antioxidants in the diet showed that feeding can modify, positively or negatively, oxidative damage to the cell structures are involved in the pathogenesis of many diseases.

The active oxygen species are produced as by-products during normal cellular metabolism, mainly in mitochondria. These are hydroxyl radical, superoxide anion, hydrogen peroxide, nitrous oxide and others. Under normal conditions, the cells have a well-developed antioxidant protection reduces the impact of reactive oxygen species. When production of these reactive species has increased to the degree that overcomes the antioxidant cellular protection are talking about oxidative stress. Because these molecules are partially reduced oxygen products, they are highly reactive and interact with other biomolecules, which may lead to lipid peroxidation, oxidation of the nucleic acids and proteins. Therefore, oxidative stress plays an important subsidiary, role in the pathogenesis of numerous chronic and degenerative diseases.

Antioxidants are substances that directly or indirectly protected the cells against the harmful effects of xenobiotics, toxins, drugs and carcinogens. Antioxidants are divided into three groups:

- antioxidants in food - vitamins C, E, carotenoids, flavonoids and other plant polyphenols;
- metallo-enzymes such as catalase, superoxide dismutase, glutathione peroxidase, etc .;
- molecules such as glutathione, albumin, bilirubin and uric acid.

Dietary antioxidants reduce oxidative damage by:

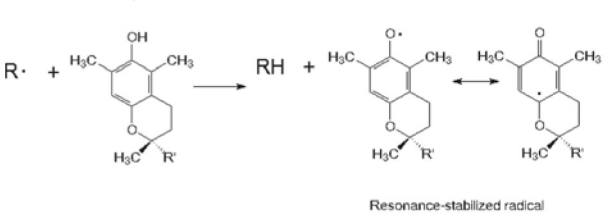
- inhibiting the processes of formation of reactive oxygen species;
- regeneration of antioxidant vitamins;
- supporting and inducing effect on antioxidant enzymes and detoxifying agents.

To increase the antioxidant defenses of the body may further be included in the diet increased intake of antioxidants in the form of dietary supplements. This is especially exposed to adverse chemical environmental factors (eg. production of petrochemicals, plastics, synthetic fibers). Where are limited opportunities for primary prevention of risk management strategies to enhance adaptation capabilities of the organism are fully appropriate. The antioxidant vitamins E, C, beta-carotene are a necessary component of a safety professional catering. In prophylactic purposes are accepted and some trace elements (selenium, zinc, manganese), directly involved in the enzymes metabolizing toxic substances.

VITAMIN E

Vitamin E is the main antioxidant in the lipid phase of biological membranes and lipoproteins. The name vitamin E includes eight compounds (four tocopherols and four tocotrienols). Alpha-tocopherol is the most biologically active. Natural alpha-tocopherol (d-α-tocopherol) is a single stereoisomer and is 2 times more biologically active than the synthetic form (dl-α-tocopherol), which is an equimolar mixture of eight stereoisomers. Their different biological activity due to the differences in their incorporation in LDL- and HDL-cholesterol, affinity of tissue to the natural form and much faster metabolism for synthetic form.

In membranes tocopherol is reacted with a lipid peroxyl and alkoxyl radicals to give a relatively stable lipid hydroperoxide and tocopheroxyl radical, interrupting the chain reaction of lipid peroxidation:



Various studies suggest that the protective effect of vit.E in terms of chronic diseases, can be attributed not only to its powerful antioxidant action but also to other poorly understood mechanisms such as participation in cell signaling mechanisms.

In addition to maintaining the integrity of cell membranes, alpha-tocopherol protects the unsaturated fatty acid in LDL-cholesterol from oxidation. It has been shown that the changed oxidized LDL-cholesterol plays an important role in the development of atherosclerotic changes in the blood vessels.

Several epidemiological studies have shown that low consumption of vitamin E increases the risk of cardiovascular diseases. WHO sponsored studies to determine the presence of low serum levels of vitamin E as a very important risk factor for developing heart disease.

Conversely, use of high doses of vitamin E in the treatment of patients already diagnosed with heart disease show conflicting results. In 1996, Cambridge Heart Antioxidant Study conducted with more than 2,000 patients with proven coronary atherosclerosis, indicates that supplementation with 400-800 UI/day synthetic alpha-tocopherol, for 2 years, reduces the incidence of non-fatal myocardial infarction by 77%, but generally total mortality from heart disease has not changed significantly.

It is believed that many cancers are the result of oxidative DNA damage. Placebo-controlled study in Finland seeking preventive effect of vitamin E in lung cancer in male smokers demonstrated 32% decrease in the incidence of prostate cancer in patients receiving 50 mg of synthetic alpha-tocopherol, over a period of 5 years.

Main sources of vitamins. E are vegetable oils, nuts and grains. The recommended dietary intake for adults of alpha-tocopherol is 15 mg/day (22,5 UI). This amount is primarily necessary to prevent the symptoms of deficiency (neurological), rather than for prevention of chronic diseases. It's hard to consume more than this amount of food without increasing the intake and fat. To prevent chronic diseases such as cardiovascular, neurodegenerative and some types of cancer is recommended intake of 200 UI (133 mg) natural vitamin E daily during meals.

However, vitamin E affects the clotting process at very high doses can lead to bleeding. On this basis calculated maximum allowable daily intake of vitamin E (natural and synthetic) - 1000 mg.

CAROTENOIDS

In the human diet most often present carotenoids alpha/beta-carotene and beta cryptoxanthin which are pro-vitamin A activity as well as lycopene, lutein and zeaxanthin. In plants, carotenoids play an important antioxidant function as deactivated singlet oxygen, formed during photosynthesis as high effective is lycopene.

Laboratory studies show that carotenoids inhibit lipid peroxidation. The system of conjugated double bonds determined absorption in the visible spectrum. This is of particular importance to the eye, wherein the lutein and zeaxanthin effectively absorb the blue light, preventing the photoreceptor cells of the retina from damage.

Although the results of epidemiological studies have shown that a high dietary intake of rich in carotenoids fruits and vegetables reduces the risk of cardiovascular disease and certain types of cancer, high doses of beta-carotene does not reduce the risk of these diseases in large randomized clinical trials. Back, two such studies found that high doses of beta-carotene (more than 20 mg/day) increases the risk of lung cancer in smokers and former workers in the asbestos industry.

Beta-carotene has vitamin A activity and included in multivitamin preparations as 2 μg beta carotene (suplemented) provides 1 μg retinol (3,33 UI vitamin A). The recommended daily intake of retinol is 1000 μg (3330 UI vitamin A)

Epidemiological studies have shown that men with high intake of dietary lycopene (tomatoes and derivatives) have a lower risk of developing prostate cancer. As a dietary supplement lycopene is available in doses of 5 to 15 mg.

Lutein and zeaxanthin are carotenoids in the retina and lens of the eye. Scientific evidence shows that consumption of at least 6 mg of these can reduce the development of age-related macular degeneration and cataracts.

ASCORBIC ACID

Vitamin C is the most effective water-soluble antioxidant and most important for extracellular fluid. Ascorbate effectively binds to a variety of reactive oxygen species. Vitamin C has and tocopherol regenerating effect by reducing tocopheroxyl radical to tocopherol.

To prevent deficiency recommended daily intake for adults is 70 mg. Numerous, mainly prospective cohort studies have shown that the amount of vitamin C necessary for the prevention of chronic diseases is significantly higher (100-300 mg). The allowable maximum daily intake for vitamin C is 2 g.

METALLOENZYMES ANTIOXIDANT

Enzymatic antioxidants - superoxide dismutase (SOD), catalases and peroxidases, have emerged as early as the evolutionary development and are established in all eukaryotes.

SOD are a family of metalloenzymes containing copper, zinc and manganese. They catalyze the conversion of superoxide radicals to hydrogen peroxide and oxygen. Daily recommended dietary intake for adults of zinc, copper and manganese, respectively - men 11 mg, 900 μg, 2,2 mg; women 9 mg, 900 μg, 1,6 mg. The maximum allowable daily intake of these elements is 40 mg, 10 mg, 11 mg. Prolonged intake of zinc in doses above 50 mg/day cause a shortage of copper and iron in the body.

Selenium dependent enzyme glutathione peroxidase is of primary importance to eliminate peroxides (hydrogen peroxide and lipid hydroperoxides) in the cell, while forming water and alcohols, and using reduced glutathione. Its importance to the body as demonstrated by Keshan disease - cardiomyopathy that develops in endemic areas of Keshan province in China, where the soil and hence the food is very low in selenium. It is assumed that oxidative stress caused by selenium deficiency leads to emergence of more virulent strains of some viruses that affect the heart muscle.

There is a reverse correlation between cancer incidence and selenium content in soil, water and food in different geographic areas. Other studies also show inverse correlation between cancer incidence and level of selenium in serum and nails. In the US in a placebo controlled clinical trial proves that selenium intake of 200 μg/day for an average of 7.4 years reduces the risk of prostate cancer by 51%.

Furthermore, increased activity of antioxidants selenoenzimes and other mechanisms have been proposed for the protective effect of selenium in respect of cancer diseases. Such are improved immune function, influenced metabolism of carcinogens, increased levels of selenium metabolites suppressing tumor growth.

The recommended daily intake for adults of selenium is 55 μg. Average diet provides about 100 μg. High doses can be toxical-maximum tolerable daily intake of 400 μg.

FLAVONOIDS

Flavonoids are a diverse group of polyphenolic compounds of plant origin. It is believed that many of the biological activities of flavonoids due to their antioxidant potential.

With used as food additives are used oligomeric proanthocyanidins, monomeric catechins from green tea, kvertsetin, extract of ginkgo biloba, silymarin and others.

Industrial oligomeric proanthocyanidins (OP) are obtained as extracts from grape seeds (92-95% OP) and mediterranean pine bark (80-85% OP). They represent dimers, trimers and tetramers of catechin, epicatechin and epicatechingalat.

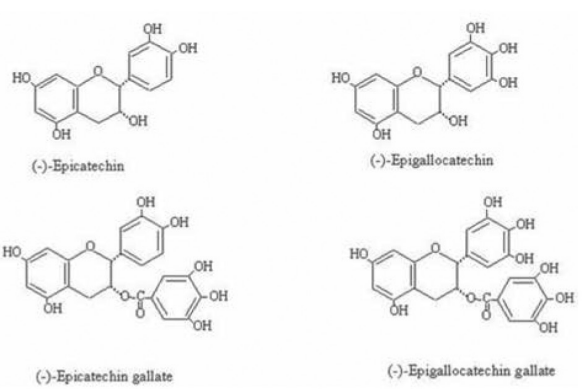
Epidemiological WHO studies show that at comparable levels of plasma cholesterol and other risk factors, the incidence of heart diseases in France is more than half lower than in North America. This discrepancy known as the "French paradox" is confirmed by other studies indicating that moderate consumption of red wine (rich OP) probably reduces the risk of cardiovascular disease.

OP used clinically for half a century they have held very in-vitro and in-vivo studies demonstrating their powerful antioxidant, collagen stabilizing, anti-thrombotic, anti-inflammatory and vasodilating action. OP have affinity for blood vessels, which accumulate in the greatest concentration and exert vasoprotective effect.

Their antioxidant activity involves direct interaction with various radicals, storing and regenerating effect of alpha-tocopherol, inhibition of xanthine oxidase, chelating iron and copper ions. In vitro studies have shown that OP are significantly more potent antioxidants from vitamin C and vitamin E. This has led to numerous, and conducting in vivo, studies demonstrating that administration of OP significantly increased plasma antioxidant activity and reduce the tendency of LDL to oxidation by free radicals.

With prophylactic dosage is 50-100 mg/day, and for therapeutic purposes (problems in blood circulation such as varicose veins, edema, etc.) is 150-300 mg/day.

It is known that a high consumption of green tea, in some areas of Japan is associated with a significant reduction in the case of cancers. Green tea contains a significant amount (up to 30% of the dried drug) **monomer catechins** (epicatechin, epigallocatechin, epicatechin gallate, epigallocatechin gallate):



In addition to the strong antioxidant activity in vitro conditions are proven and various other mechanisms (stimulation of detoxification enzymes, etc. .) contributing to the anti-cancer activity of green tea.

With prophylactic dose is 100-300 mg/day of a standardized extract (60-80% polyphenols).

Quercetin is the most common flavonoid in plants such as occurs in the form of glycosides (rutin, kvertsetrin, hesperidin, etc.). In comparative in-vitro studies it shows the strong antioxidant properties as compared with other flavonoids. It has a strong anti-inflammatory and antiallergic action, making it useful in complex therapy of asthma, allergic rhinitis, rheumatoid arthritis and the like. Quercetin is a potent inhibitor of the enzyme aldose reductase in the eye lens and has a protective significance in the development of cataracts in diabetic patients. Apply in high doses (1,2-1,5 g/day) due to a very weak absorption in the gastrointestinal tract. Recently, using an analogue thereof - quercetin chalcone in a lower dose (750 mg/day) due to its high bioavailability.

Standardized extract of **Ginkgo biloba** contains 22-27% flavonol glycosides and terpene trilacotones 5-7%. Many clinical studies have confirmed its effectiveness in subjects with symptoms of impaired cerebral blood flow (impaired memory, tinnitus, etc.). The extract was administered at a dose of 120 mg/day. The effect is observed after several months of supplementation.

Proven hepatoprotective effect (chemical damage, viral hepatitis) has extract of milk thistle, contains flavonoid mixture **silymarin**, the effect is due to the antioxidant action.

The intake of flavonoids is well tolerated without side effects. As a general problem of all flavonoids is their low and unpredictable resorption. Developed extracts of grape seeds, green tea and ginkgo biloba, in which the flavonoid compound has been associated with soya phospholipids, leading to significantly higher absorption and effect.

Drugs with antioxidant activity

Conducted in our research in petrochemical plant have shown abnormalities in antioxidant defenses and a health risk to those exposed. It has been found that by increasing the concentration of benzene in air, increasing the level of malonic aldehyde in the blood - a biomarker of lipid peroxidation. It is also a significant decrease in antioxidant status - measured activity of the enzyme glutathione peroxidase and reduced glutathione concentrations.

Studied is the ability to counteract oxidative stress by using a food supplement "Oxiprevent"(supplement contains a complete blend of antioxidants). As a result, the intake significantly improved antioxidant status of exposed workers as all biochemical indices recovered to normal values.

An increasing number of immunocompromised people and the frequency of individuals with an antibiotic resistance in the treatment of various acute and chronic infectious diseases. Also common are hypersensitivity reactions to antibiotics and the manifestation of their immunosuppressive effect.

One way to overcome this problem is prophylactic use of immune stimulating agents.

Food additive "Respistim plus" (an improved version of Oxiprevent) is a combination of strong immune-stimulating lyophilized bacterial lysate and a full range of antioxidants. The bacterial extract contains lipopolysaccharides, peptidoglycan, muramyl dipeptide, tayhoic acids of the most common bacterial cause of respiratory diseases. In experimental and clinical studies have demonstrated a general stimulant properties on various immune system components such as phagocytosis, complement, interferon, secretory IgA, alveolar surfactant, B- and T- helper lymphocytes and others. and specific immune stimulation - synthesis of antibodies to the bacterial species from which it is isolated lysate.

Administration of "Respistim plus" fell nearly 10 times respiratory infections in soldiers, exposed daily on stress and overheating weather.

Other combination preparations with antioxidant activity are: Enogriyn instant drink Oxibor forte, Triovit, Phytophanere solaire.

Drugs with eye protective effect containing a combination of lutein and other antioxidants:

Stel and Stel forte, Eyelut, ICaps, Vitalux, Vitavision.

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11 HYGIENE REGULATION OF ENVIRONMENTAL FACTORS. ASSESSMENT AND MANAGEMENT OF OCCUPATIONAL RISK - APPROACHES AND MODELS

Throughout his life, a person is under the constant influence of various environmental factors. Modern hygiene justify preventive measures is based on the concept of the primary role of environmental factors (OS) in the etiology of human diseases. Balancing the relationship of the "Environment-man" for the purpose of primary prevention of diseases is reached with hygiene regulation of environmental factors, which is the theoretical foundation of hygiene and has important practical significance. It is used in solving the events of technological, sanitary and urban character and is the basis for health legislation.

The basis of hygiene standardization standing notion that only certain combination of various environmental factors is optimal for humans. In a significant change in the composition and intensity of environmental factors disturbing the unity between body and environment. In such conditions, or completely exclude the possibility of existence of the organism or therein develop pathological alterations occurring in the form of acute and chronic diseases. In less deterioration of the environmental qualities observed mobilization of protective-adaptive mechanisms of the body, in which adaptive reactions can go in pathologies.

Hygiene norm is the maximum safe quantitative physiological level, which is set for factors in differentiated levels (minimum, maximum and optimum). It ensures the preservation of health in the broadest sense, including genetic and reproductive health of both the individual and the entire population. Subject to the hygiene standards, human performance is not reduced and not worsen the conditions of life and work.

In different sections of the hygiene norms of environmental factors has its own specifics. Moreover, modern methodical approaches to hygienic normalization factors in different environments based on general theoretical principles. Theoretical basis of hygienic normalization is general biological status of the threshold effect on the body of the factors of the environment and the theoretical possibility of reproduction (modeling) in experimental conditions on animals pathological reactions man and extrapolation of the results of experimental studies on people[1]. The threshold values are the smallest concentrations (dosage levels) that lead to statistically significant deviations of indicators of vital activity of animals (people) in chronic (acute) experiment. In determining the threshold of action is necessary to establish physiologicaly permissible fluctuations of indicators and to distinguish the "norm" of "prepathology" and to obtain information about prepathological state in experimental and clinical studies, were used adequate functional tests with load. The threshold value of the factor is needed in order to determine the maximum no-effect dose (concentration, level). Eg. for each toxic substance is determined limit of effective concentrations (doses) under which does not occur harmful effects. These safe concentrations usually are several times lower than the threshold. To justify the limit concentrations (MAC) or doses (MAD) of harmful substances are conducted experimental toxicological studies in acute, subacute and chronic experiments on animals and epidemiological studies.

From acute experience obtain information about the degree of toxicity, range of toxicity, the magnitude of the DL_{50}, species and gender sensitivity of experimental animals to the test substance. The results of subacute experiment allow to judge the degree of expression of the cumulative properties of the substance. In the chronic experience are established threshold (threshold of chronic exposure to the substance) and sub-threshold (maximum no-effect) concentration. The maximum dose no-effect of the test substance is used to justify the MAC (MAD). Experimental studies carried out on sensitive for test substance, type laboratory animals. In epidemiological studies, studied the health status of the population exposed to this toxic substance.

[1] Possible experiments (modeling) and people under strict conditions that guarantee non-injurious to health

11.1. ENVIRONMENTAL FACTORS

The problem of hygiene regulation of environmental factors is complex, as opposed to factors of work environment, they are characterized by low intensity, but operate continuously throughout the life of the individual, irrespective of age and profession, there is diverse combination of factors that act simultaneously on the body in a complex interaction between them.

Historically may be noted three areas of hygiene standardization environmental factors:

1. The basis of hygiene standardization adopt the characteristics and parameters of a factor in the clear (without anthropogenic impact) environment. It should be borne in mind, however, that "pure nature" does not always offer optimal conditions for the organism. For example, in some areas settled natural deficiency or excess of micro- and macro- elements that need to be adjusted. On the other hand, many so-called harmful factors in certain quantitative relations appear as bioelements.

2. Regulation of communal factors to comply with the technical parameters achievable, according to the development and capabilities of technological progress. From the hygienic point of view maximizing this approach it is also unacceptable as these parameters may incidentally coincide with optimal conditions and harmless to the body. Modern equipment and technologies enable a high degree of purification in relation to a number of factors, the achievement of which in some cases is not necessary and beneficial for the body, while in terms of other factors still no workable solutions to achieve optimal parameters.

3. Physiologically justify hygiene standards for determining harmless to the body and generations parameters active factors. This is the basic and correct modern approach.

Important from a methodological standpoint is acceptance of certain parameters for harmful substances and factors in the environment not as norm, but as MAC, i.e. concentrations that are not normal, but are the limit- i.e. not distort hygiene comfort, have no direct or indirect adverse effects on the man and his descendants, not decrease performance and his confidence, no harm plants and animals. It is therefore necessary to move from the definition of the minimum levels of environmental factors, to the next stage - and from the optimum parameters. For example, a MAC (maximum value according to Decree № 9 of 2001 on the quality of drinking water in the country) for the content of fluoride in drinking water is considered 1,5 mg/l (risk of developing fluorosis); the minimum concentration (MIC) is 0,5 mg/l (prevalence of dental caries) and optimal necessary parameters (ONP) are 0,7-1,0 mg/l.

Existing hygiene and are **temporary** and need systematic reassessment and improvement based on monitoring of environmental factors and studies their effects on the body.

In hygienic regulation of air pollutants is taken into account that their impact is at the entire population, including and the most sensitive part of it - children, the elderly, chronically ill and in throughout the day and throughout their lives. In this possible reflex action, resorptive effect and indirect effects of chemical compounds on the environment and living conditions of the population. Therefore hygiene standards for ambient air are lower than those quantitative levels of harmful factors that can lead to reliable diversion of some of the indicators for normal vital activity of the body in this population. Experimental studies to assess the biological activity conducted in two stages. The first stage being studied in humans (volunteers) threshold of odor, irritation and reflex reactions in short-term effects of the substance. The information obtained is used to justify the maximum single concentration. The second stage is held to justify daily average concentration for preventing resorption effects of harmful substances during their prolonged inhalation entering the body. To determine the MAC experiments are conducted on laboratory animals undergoing round-the-clock inhalation effects of the test substance over the course of 3-4 months, which is determined threshold and sub-threshold concentration from resorption activity of chemical compounds. In this case, if the MAC does not establish by reflex action, sub-threshold concentration is justified MAC of this substance in the air. In hygienic regulation of air pollutants accepted the principle of the limit (most sensitive) indicator. For example, if the smell of the substance is felt at concentrations that do not have a harmful effect on humans (resorptive activity) and environmental, norms becomes by the threshold of perception of smell.

MAC substances in the waters of the reservoirs are established based on medical and biological research, starting from the priority of hygiene requirements to ensure the safety and welfare of the population for water. Development of hygiene standards provides comprehensive research of the substance in 3 major signs of harm:

1. toxicological studies of the influence of the substance on the body;

2. influence on organoleptic properties of water;

3. impact on the sanitary regime of the basin.

In establishing standards for drinking water can be tested only the first two signs of harm.

The study of the effects of substances on the organoleptic properties of water aims to establish a threshold concentration for their impact on the smell, the taste, color, transparency (turbidity).

The study on the sanitary regime of reservoirs aims to establish a threshold concentration in their influence on the processes of biochemical oxygen necessity, mineralization of nitrogen-containing organic substances and develop of aquatic saprophytic microflora.

The study of toxic effects of chemical aims to establish the maximum no-effect concentration (MNC), ie of sub-threshold concentrations of sanitary-toxicological sign of harmfulness. This happens in toxicological experiment on laboratory animals or by predicting the value of MNC with subsequent extrapolation of data on humans.

Guiding principle in regulation of toxic substances into water is the principle of limiting indicator of harmfulness, i.e. accepted for MAC lowest set of values from above 3 (2) symptoms of harm.

Security of scientifically based hygiene norms is checked in practice through comprehensive studies of reservoirs under natural conditions using epidemiologic methods to study the health of the population.

In hygienic regulation of toxic substances in the soil examine their impact on these harmfulness indicators: translocation, air-migration, water-migration, and general-sanitary. For those indicators are established permissible concentrations of substances that do not affect the process of self-purification of soil, onto her microbiocenoses and exclude the possibility of passing the test substance into groundwater and surface water, plants and air in quantities exceeding hygienic norms for each of the mentioned objects of the environment. In normalization of substances in soil, as normalization of air pollutants and water, decisive is the principle of limiting indicator of harm - for MAC is considered the smallest of the four values established in the above indicators for harm.

11.2. WORK ENVIRONMENT FACTORS

Possibility of **hygienic regulation of chemical factors in the work environment** is based on the existence of dependency "dose-time-effect" and threshold for harmful action. Many known mutagens and carcinogens as are necessary for normal vital activity - trace-elements, for example selenium, arsenic, chromium and others. These compounds are dangerous only at a certain quantitative limit in the body when the protective systems are inadequate. This level corresponds to the threshold of the harmful effects of the poison. It is considered sufficient justification to be assessed as harmful alterations that go beyond the physiological norm for one or other indicator (it being understood exit outside the normal range 1,5-2,0 Sx or in many cases - 3 Sx) . For the most reasonable approach to adequate assessment of the significance of detected changes currently considered complex complete characterization of the state of the organism as a whole, which recognizes the dynamics of the process in time. Many important now is the question of criterial significance of the observed changes in the assessment of the effects of compounds in low concentrations. Prolonged contact with chemical compounds often no-leads to the formation of a symptom characteristic of chronic intoxication with industrial poisons, but working under those conditions there are signs of a decrease in the general resistivity of the body, increase the level of non-specific morbidity, changes in immunological reactivity. Criterion for assessing the harmful effects of chemical compounds is the preservation or enhancement of adverse effects after the recovery period, which is typical for poisons having far in the time type of action (carcinogenic, gerontogenic effects).

The methodology of the hygienic regulation of chemical compounds in the air of the working area provides specific staging that enables:

- quick scientific prediction of the degree of danger and toxicity of chemical compounds;
- establishing safe levels of exposure to experiment on animals;
- clinical- hygienic approbation for established in experiment MAC.

The first stage is held justify approximate safe levels of influence, with the help of calculations for various physico-chemical properties of the substance and experiment on animals - on indicators of toxicity in single and repeated (up to 1 month) impact. The calculation methods are based on the relationship between the parameters of the toxicity of the substances identified in short-term trials and established MAC of these substances in the environment. It was found that on the basis of dependence "dose-time" and "dose-effect" it is possible to predict the effects of chronic action using the results of the acute experience, and also to identify the relevant hygiene standards for harmful substances.

The second stage of the hygiene regulation of harmful substances are exploring their toxicity by inhalation effects on the body of laboratory animals, but also evaluate skin resorption and irritating properties, the nature of the action and the rate of accumulation of the substance in re-entering the body. Studies completed by conducting chronic

toxicological experiment, which aims to determine the threshold of chronic exposure to the substance. For the determination of MAC threshold value of a chronic effect dividing by ratio of the stock (uncertainty factor, modifying factor). The modifying factor is different for different substances (typically 10 but can be 100 and more) and depends on the characteristics of the biological activity of the substance, its cumulative properties and others.

Conducted the third phase clinical- hygienic and epidemiological studies in working in endangered industries are criteria for prognostic value of experimentally established MAC.

The basis of the norms of physical factors is also thersholdness. In the hygiene regulation of the negative high levels of physical factors (noise, vibration, electromagnetic energy, ionizing radiation, etc.) are justified safe for body-limit levels (LV) or doses (LD).

The purpose of **the hygienic regulation of noise and vibration** to determine the permissible dayly (or weekly) dose of energy for relevant factors, which provides in terms of employment (taking into account the gravity and tension of labour) prevention of functional disorders or diseases caused by these factors.

To assess the physiological changes resulting from the effects of noise or vibration are two approaches. The first is generally accepted and can be called concept of "eligible" change - change compared with criterial permissible alterations (not causing severe intensions and restore normal). The second approach can be called concept of "zero" changes when changes in the indicators do not go beyond the physiological norm, i.e. dose is no-effect.

Given the specificity of noise-vibration factor, defined the basic principles of hygiene standardization, including physical, medico-biological, and social-hygienic aspects:

1. The hygiene regulation of noise and vibration in the production environment must be taken into account interaction is system "man-machine-production environment." Scientific and technological revolution reduce manual labour in manufacturing, increased role of knowledge workers. While previously in the "human-machine" focuses on the coordination of the design of the machine with anatomy-physiological characteristics and parameters of the body, it is currently leading conformity of the machinery and labour microenvironment with neuro-psychic possibilities of man.

2. Hygiene regulation of noise and vibration is based on health and performance criteria to assess the impact of factors on the whole organism in the process of work, given the intension and hardness of labour. The degree of harmfulness of noise and vibration judging by the results of the assessment of the health status of workers: morbidity and clinico-physiological studies in dynamics during the working day and the recovery period. Starting from the nature of the effect of noise on the whole organism, is establish concept of bioequivalence to the effects of the influence of noise and nervous load (confirmed in manufacturing and experimental studies). In their joint action, their adverse impact on the body is additive.

3. The adverse effects of noise and vibration is estimated as by their direct effect on adequate functions of analysts, and in mediated reactions throughout the body. The selection of adequate and informative methods for testing must be based on modern ideas about ways of receiving information in the body of the current stimulus, but along with the study of hearing, skin and vestibular analysts to also include indicators of functional state of nervous, cardiovascular and endocrine systems having general biological significance. About the dangers of noise or vibration is judged by the degree of fatigue onset and severity of remote consequences. Assessing the impact of noise is performed by specific changes in the auditory organ and nonspecific changes that appeared before them, which occur in disorders of neuro-psychic sphere - fatigue, dizziness, headache, sleep disorders and others. In assessing the impact of general vibrations into account the extent of violations comfort when working near (immediately) to sources of vibration.

4. The criterion for harmful effects of factors shall be established by changes in indicators of physiological functions compared with the norm and taking into account the prediction of irreversible changes in the magnitude of temporary changes the threshold of analyzer systems - used to assess the changes in physiological indicators during the 8-hour day, compared with their level before work (eg. it was found that the temporary reduction in hearing sensitivity after 8 h noise impact is equal in rank - in dB, of sustainable decreased auditory acuity after 10 years of working in continuous effect noise with the same intensity; such are the changes in vibration sensitivity and indicators of peripheral blood circulation in local vibrations). A distinctive feature of the hygiene regulation of noise and vibration is to conduct experimental studies on volunteers to exclude masking or enhancing effect of other factors of production.

Microclimate normalization of production is based on the results of studies for heat transfer from man to the environment and conditioned by his functional status of various systems of the body,

and the biological effect of various weather factors. Normalization is aimed at ensuring the two levels of the human heat condition (optimal and acceptable), taking into account its level of energy consumption and period of the year. Provides standardized requirements for individual microclimate parameters (temperature, humidity and speed of air movement) that in their joint action to provide the required thermal condition of the body.

As standards for optimal production microclimate accepted levels of individual components, which in systemic and lasting effect on the human body does not lead to significant changes in physiological status, help to preserve confidence and health of workers and create prerequisites for high performance and labour productivity.

Hygiene standards for microclimate must ensure levels of thermal conditions in the body of workers, where there is no tension on the mechanisms of thermoregulation or the same is weak. Developed a special classification of thermal conditions, at the basis of which are criteria to evaluate the function of thermoregulation and cardiovascular system, taking into account indicators for the energy value of labour. "Optimal" thermal condition is characterized by a lack of intension by thermoregulatory mechanisms, comfortable heat sensation, preserve human health and a high level of performance. In "permissible" heat condition has been some tension of thermoregulatory mechanisms, uncomfortable heat sensation, is possible decrease in performance, but there are no violations of health.

It is preferred that the complex evaluation of the microclimate in general; determining the degree of thermal discomfort/comfort; the risk of heat stress in acclimatized/noacclimatized, taking into account the intensity of work (metabolic heat) and thermo resistance (insulation) of clothing.

In the **hygiene regulation of the ionizing radiation factor** takes into account the specifics of its biological impact on the body. Dependence "dose-radiation-biological effect" occurs in two forms of biological effects: threshold (non-stochastic) and non-threshold (stochastic) effects. Normalization of non-stochastic effects is based on the concept of a threshold effect, i.e. determine a maximum physiologically harmless, quantified level of impact. In a study of stochastic (non-threshold) effects departing from the principle of probability of occurrence and not from the severity of the violations, which causes radiation. Specific hygiene regulation of stochastic effects is that in developing the system of radiation protection is considered the concept of non-treshold action whereby theoretically each dose ionizing radiation have mutagenic action.

Important practical matter the justification of norms, consistent with the simultaneous effect on the body of multiple environmental factors. In this account: the nature of the action - complex, combined or coupled effect of various environmental factors; bioequivalence (izoeffectiveness) of hygienic norms established for different environments and others. In **the combined action** of harmful substances can be observed different effects - an additive effect (the most common), the potentiation and antagonism. Upon receipt of a harmful substance in the body by both inhalation and oral route **(in-complex action)** toxicity becomes higher than the receipt of this substance only once route. In terms of the **coupled action** of factors of different nature can be observed strengthening (most common) or weakening of their action.

11.3. ASSESSMENT AND MANAGEMENT OF OCCUPATIONAL RISK - APPROACHES AND MODELS.

Risk assessment is a process in which data from experimental studies and epidemiological studies on humans are combined with information about the degree of exposure, to be able to quantitatively predict the probability of specific adverse health effects that would have occurred in exposed population. Health risk assessments is divided into four stages: risk identification, evaluation of dose-response relationship, exposure assessment and risk characterization **(Fig. 1)**.

Risk identification is the first and most easily step in assessing the health risk. This is a process of determining whether exposure to certain professional agent could increase the likelihood of adverse effects on the health of the exposed population. It is based on experimental animal studies and epidemiological studies of exposed persons in order to determine the health side effects when exposed to certain occupational hazards.

The evaluation of the dose-response dependence defines the relationship between the dose of exposure and the possibility of specific side effects. It requires information on methods of extrapolating the effects from high to low exposure and from experimental studies in animals to humans. There are some problems in extrapolating data from experimental studies to humans: **the various possible mechanisms** of action of some occupational hazards are not fully known; **doses used in experimental animal studies** are often very high and these doses are not commonly found in exposed individuals; **there is a difference between animals and humans** in relation to the metabolism of toxic

substances; **dose that is released in specific target organs** of animals is different from that in humans. The most accurate model for assessing human exposure based on data from experimental studies are physiologically based pharmacokinetic models for the effects of chemical factors.

Exposure assessment determines the magnitude and probability of accumulation of the toxic agent from the environment in the body. It is defined as an external (the concentration of different toxic agents in the environment and working environment) and internal exposure (accumulation of these substances in the body). For some chemicals, the actual exposure is determined directly by measuring the content of the toxic agent or its metabolites in certain body fluids, feces, hair, etc.

The most important part of the assessment - **the risk characterization** summarizes and interprets the information collected during the previous stages and identifies the limitations of this evaluation. It requires to determine whether in the specific workplace, there are significant health effects. Complete characterization must discusses previous levels of production factor in the environment and work environment and in the body, the pharmacokinetic differences between experimental research and studies in humans, the effect of the choice of the specific parameters for the exposure, statistical analyzes, as well as other factors that may affect the magnitude of the evaluated risk.

The influence of chemical and physical factors in the workplace on the cardiovascular system is one of the current problems of preventive medicine. The difficulty of diagnosing cardiovascular injuries from professional hazards arising from a number of reasons. These injuries (so-called. "Work-related" cardiovascular disease) are not specific and are expressed at increased risk for the development of hypertension, ischemic heart disease, cardiomyopathy or heart rhythm and conduction disturbances. Due to the multi-risk nature of these diseases, demonstrating the role of occupational exposure is very difficult. In addition, non-invasive methods to study the cardiovascular system recommended by WHO for epidemiological studies do not allow for safe diagnosis of CHD. In most of the studies used different methodologies, so the results can hardly be compared.

Production factors most likely damage the cardiovascular system are: some metals (lead, cadmium, mercury, cobalt, arsenic, antimony, yellow phosphorus), nitrates, carbon monoxide, hydrocarbons, carbon disulphide, pesticides, noise, vibration, high and low temperature, non-ionizing radiation and others.

Approaches to prove the causal link between work-related CVD and professional factors include:

1. Detailed occupational history to establish relationships over time between the occurrence and development of cardiovascular symptoms and risk factors of the working environment.

2. Establishment of exposure to occupational factor - daily, weekly, annual and cumulative (over the entire service).

3. Detection and evaluation of others (professional and domestic) risk factors.

4. Perform a general clinical examination with focused study of the cardiovascular system.

5. Use of diagnostic tests to identify the type and the degree of cardiovascular damage - ECG; 24-hour ECG monitoring; step test, possibly coronary angiography; 24-hour monitoring of blood pressure and others. In epidemiological studies, WHO recommends the use of ECG and its interpretation by the code of Minnesota and Rose questionnaire for angina pectoris and myocardial infarction.

6. The determination of the risk of impact of working factor under certain conditions of the working environment requires sufficient continuous and controlled epidemiological studies (case-control and cohort retrospective and prospective studies) and analysis of results through advanced methods of biostatistics.

Epidemiological studies and constantly perfected methods of biostatistics are of great importance in the assessment of occupational risk. There are three main types of epidemiological studies: descriptive, analytical and experimental. **Descriptive studies** can be used to prove the hypotheses and are relevant only for the characterization of the development of certain diseases. **Analytical studies** enable testing specific hypothesis and by calculating the degree of risk, to determine whether the etiologic factor associated with the development of disease: (1) What are other risk factors for this disease in the exposed population? (2) Whether the existing occupational exposure increases the risk of disease (using relative risk for measuring excessive risk)? (3) How many cases of the disease could be prevented if exposure is eliminated (using the methodology for calculating the attributive risk)? **Experimental studies** aim demand a strategy to influence the natural history of the disease, for example interventional studies to reduce risk factors, screening studies to identify the early stages of the disease, etc.

Fig. 2, 3, 4, 5, 6 and 7 presents an exemplary model for demonstrating a causal link between exposure to certain professional factor - vibration, and the probability of developing disease caused by it

(cardiovascular diseases and dyslipidemias). The proposed approach is based on research for health risk assessment and can be applied to study the effect of other factors of production on the cardiovascular system.

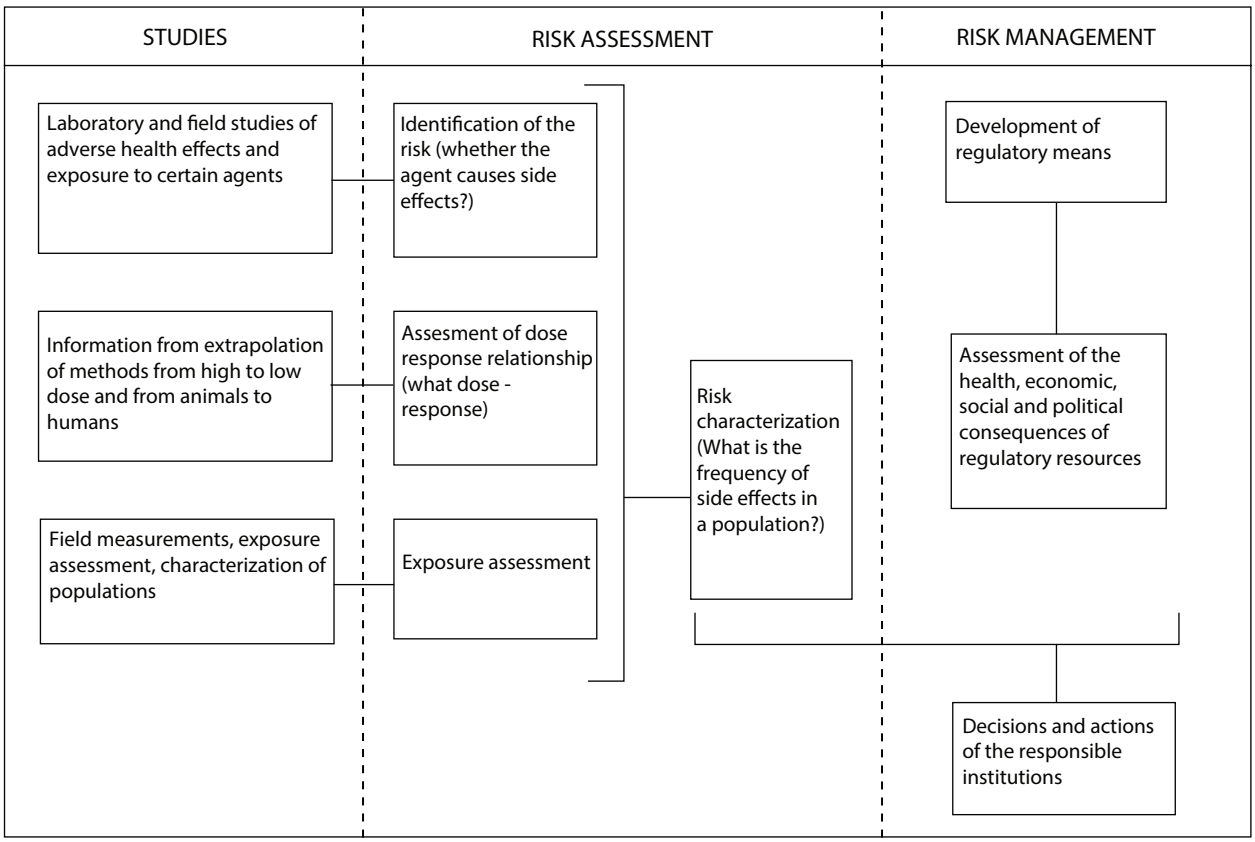


Fig.1. Elements for assesment and managing risk (of National Academy of Sciences, 1983)

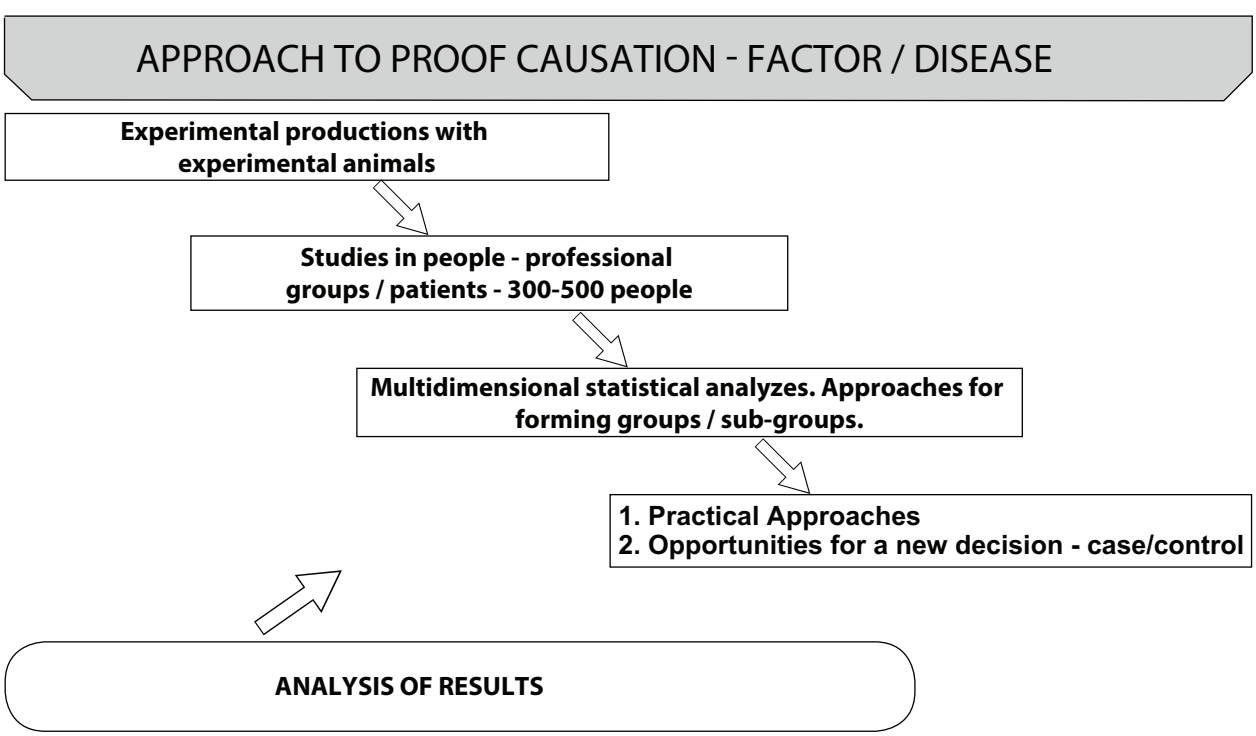


Fig.2.

Fig.3.

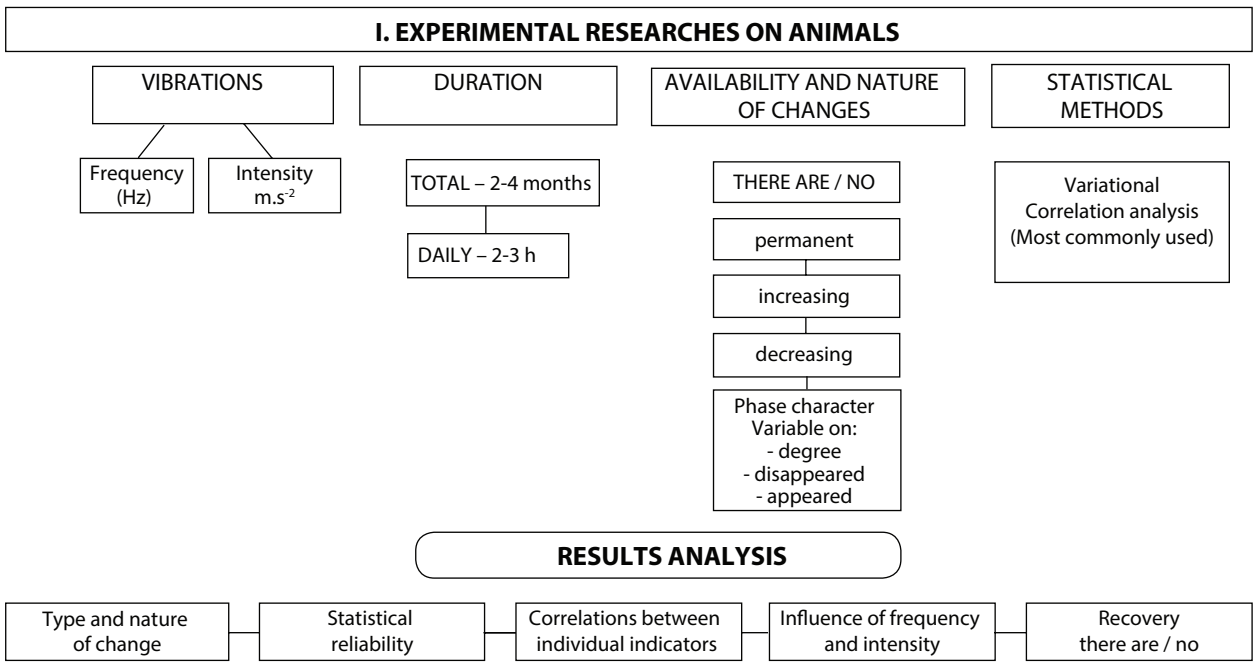


Fig.4.

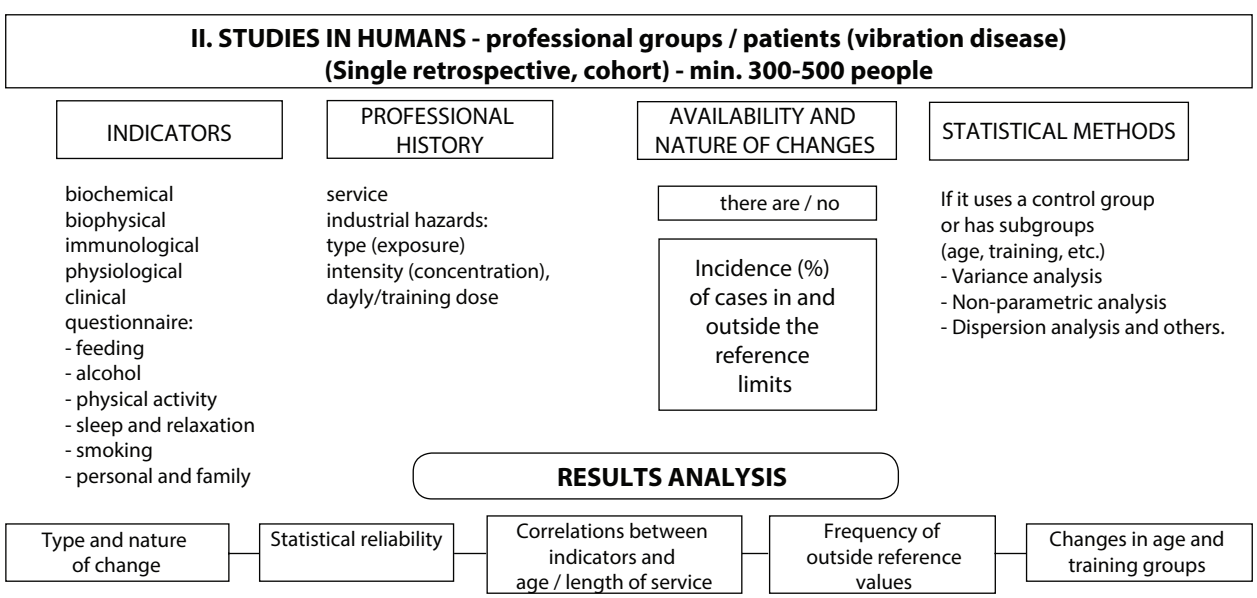


Fig.5.

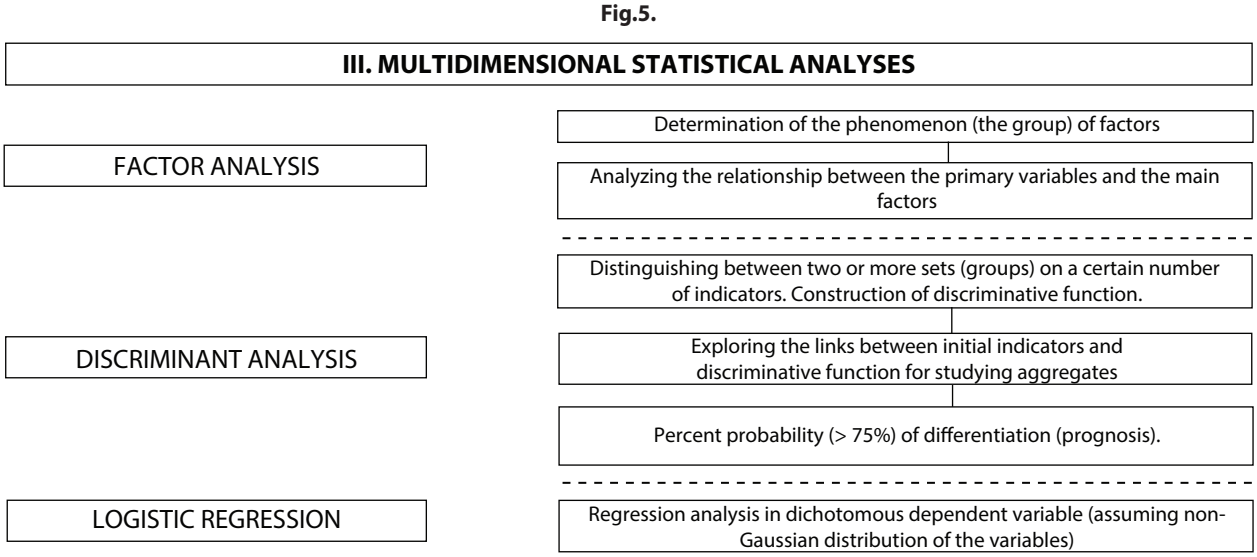


Fig.6.

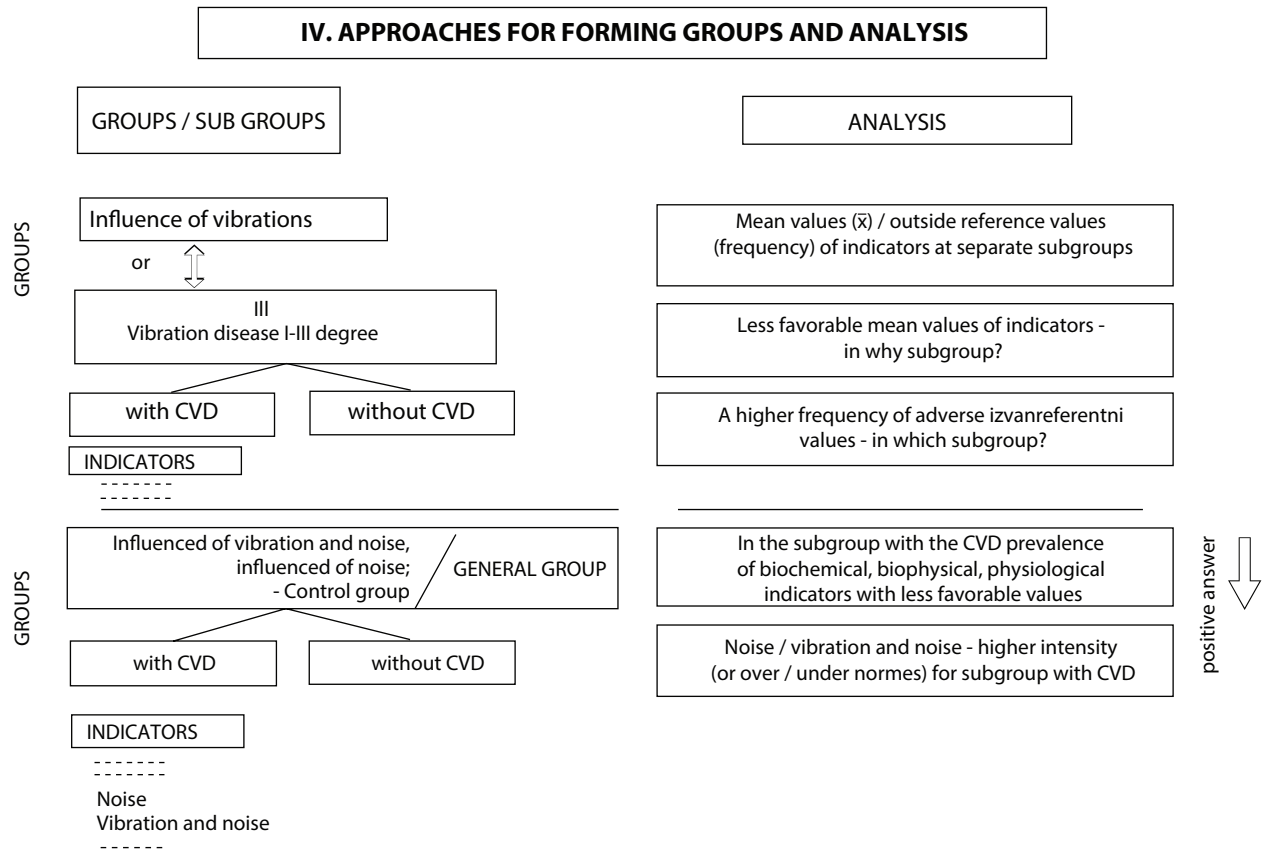
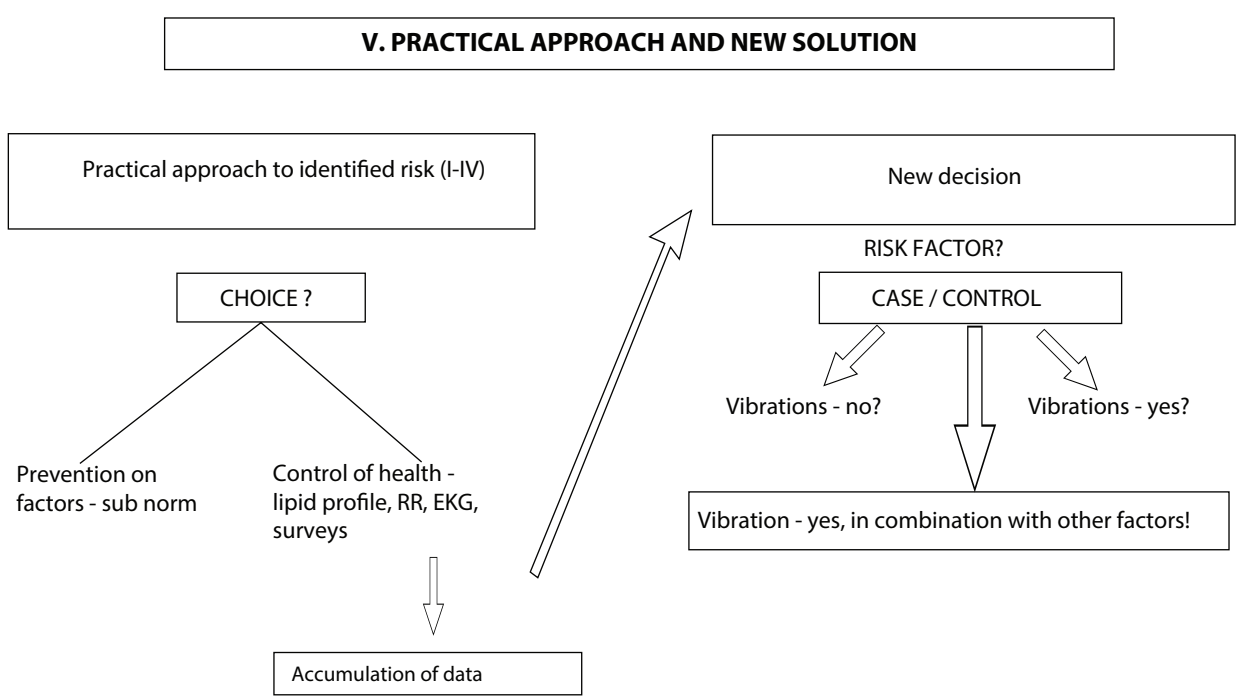

IV. APPROACHES FOR FORMING GROUPS AND ANALYSIS
GROUPS / SUB GROUPS
ANALYSIS
GROUPS
Influence of vibrations
or
III
Vibration disease I-III degree
with CVD
without CVD
INDICATORS
Mean values (x̄) / outside reference values (frequency) of indicators at separate subgroups
Less favorable mean values of indicators - in why subgroup?
A higher frequency of adverse izvanreferentni values - in which subgroup?
GROUPS
Influenced of vibration and noise, influenced of noise; - Control group
GENERAL GROUP
with CVD
without CVD
INDICATORS
Noise
Vibration and noise
In the subgroup with the CVD prevalence of biochemical, biophysical, physiological indicators with less favorable values
Noise / vibration and noise - higher intensity (or over / under normes) for subgroup with CVD
positive answer

Fig.7.


V. PRACTICAL APPROACH AND NEW SOLUTION
Practical approach to identified risk (I-IV)
CHOICE ?
Prevention on factors - sub norm
Control of health - lipid profile, RR, EKG, surveys
Accumulation of data
New decision
RISK FACTOR?
CASE / CONTROL
Vibrations - no?
Vibrations - yes?
Vibration - yes, in combination with other factors!

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12 PRODUCTION TRAUMATISM

Production trauma are injuries to the body, occurred suddenly under the influence of external factors, directly related to production conditions, that disabilities violate anatomical integrity of organ/tissue, and damage physiological functions. Possible mechanical, electrical, thermal, by ionizing and non-ionizing radiation, or chemical manufacturing traums[1].

For **industrial accidents** are all traumatic injuries, occurring during or in connection with work performed (and any damage to health occurs as a result of working in an emergency or abnormal conditions for a profession), if it led to temporary disability, invalidity or death of the employee.

Some non-industrial accidents are assimilated to production and recorded and reported as them. These are injuries occurring during any work done on the order or not, but in the interest of the enterprise, during breaks spent in the enterprise; when saving human life or public property; in outward or return of work; from the feeding place during the working day and the place to payment; in the performance of public duties in connection with the operation of the enterprise.

Production injuries are lightly, heavy, emergency and deadly. Lightly are those that do not lead to disability. Heavy lead to disability and emergency affects more than three people.

Following the production injuries (accidents) may experience temporary or permanent disability (invalidity). When permanent disability the victim usually retire.

All production injuries and treated as non-productive ones, which have caused loss of performance for one or more days, subject to registration within three days.

The analysis and study of the production traumatism is carried out using two basic coefficient. The coefficient of frequency (K_f) and the coefficient of gravity (K_g) is calculated based on: average number of personnel during the reporting period (A); total lost working days - B (on this indicator accidents are divided into led to the loss of working days: to 3; 4 to 17; over 17 and invalidity); the number of accidents occurred during the reporting period (C).

$$K_f = \frac{C_x 1000}{A} \qquad K_g = \frac{B(\text{total days})}{A}$$

Reasons for production injuries can be grouped as:

Technical inherent in the technology and use of technically outdated and dangerous production processes. Serious reasons are: inadequate equipment, lack of blocking devices on dangerous jobs, failure of machinery and equipment, lack of automatic and remote control units, imperfection of fences and protective devices and others.

Organizational reasons in the first place are the result of incorrect manipulation by workers due to inadequate instruction, training and supervision, or due to non-compliance with the rules of technical safety. After injury due to shortcomings in the workplace, poor order, poor maintenance of internal factory communications and transport, long working time, failure of or no-use personal protective equipment and more.

Reasons for **not complying with the requirements of occupational hygiene and sanitation, ergonomics and psychology of labour.** For example excessive heat production microclimate significantly reduces the speed of reactions, distributy and sustainability of attention, loaded significant cardiovascular and respiratory systems, which influenced to increase production traumatism. Low temperatures in addition to the general impact on the psycho-physiological functions can often be indirect cause of accidents - because wearing uncomfortable and heavy clothing and restricting freedom of movement. Significant impact on accidents at work and have the status of lighting, color dynamics, industrial aesthetic. Studies show that as improved brightness can be lowered in some cases by 40-50% the number of accidents and rational coloring of

[1] Similar in nature/reason are household injuries, but they are not linked to production.

the interior, plant and equipment - by 4-12%. Pulse and high-frequency noises interfere with workers to hear sounds (especially suffers perception of speech due to the masking effect of noise mostly on the perception of consonants). Along with that, caused by noise, fatigue and reduced efficiency of 30-60%, it is also one of the reasons for the increase in injuries in noisy industries. Fatigue and changes in the functional state of higher nervous activity under the impact of excessive concentrations of toxic gases, vapours and dust also contribute to increasing the level of trauma. General and local vibrations (especially in transport, military, vibration technologies in construction) lead along with objective changes in sensation (vibration, touch, pain, temperature) also to pronounced phenomena of subjective discomfort and fatigue, which increases the risk of accidents. In a significant physical strain, the level of fatigue and as a consequence of injuries and is also considerably overstated. There is also a strong correlation between the level of accidents and a number of ergonomic indexes. This is incorrect posture no-compliance requirements for working in the areas of accessibility, irrational design and maintenance of controls and means of presenting information, non-ergonomic work furniture, etc. It is believed that because of the failure to the ergonomic requirements and those of the psychology of labour arise about 18-20% of accidents. Important are the relationships within the workers. Studies show that 9 to 15% of an accident persons they have poor relations with colleagues, insufficient adaptation and non-inclusive to a group (eg. working together in difficult operations requires not only rational and synchronized interaction between all participants, but constant attention with a heightened sense of responsibility for the safety of others).

Reasons of the human factor. By 1960 almost equal importance has been serving the technical and human factors of production traumatism (till after 1929 began to recognize the importance of human factors). Now, however, Heinrich attached 88% of accidents in the US industry "personal dangerous actions", Barbier stated 90% of accidents in the French metallurgy and Coppee-Bolly - 98% of Belgian industry. Health is an important factor (among human reasons) to increase traumatism. It was found that the chronically ill (ulcer disease, diabetes, cardiovascular diseases - mainly hypertension, neurosis) were observed more frequently accidents. Gender also affects of trauma - women experience greater difficulty to adjust in a moment of danger; their bodies are more susceptible to poisoning; immediately before and during the first four days of the menstrual cycle, they sharply rising incidence of injuries, accidents, but are generally less severities, etc. Age to 30 years was also "cause" of more frequent injuries, as it is associated with a reduced self-control, impulsiveness and conceit, reluctance to seek help from colleagues. Frequent injuries seen with elderly workers associated with age physiological and psychological changes. There are hypotheses about "personal predisposition" to injuries - found that people with more accidents are very fast psycho-motor reactions, respond quickly and accurately to psycho-physiological tests. Such a "predisposition" however, is rather temporary - under the influence of emotions, stress, personal problems (very rare to meet people with enduring predisposition to injury). "Left-handing" can also affect the level of trauma - about 22% of workers with frequent accidents are left-handed, while workers with less accidents only 5% work with his left hand.

Other reasons - marital status, ethnicity, experience and training, operation, use of alcohol, motivation (satisfaction) of work and more. Family workers have fewer accidents than single people, which is explained by the increased sense of responsibility. Described are differences in the level of trauma depending on ethnic (racial) identity, but it is associated primarily with the family and social environment and conditions, not heredity. People with more experience and training allow fewer accidents - believes that after the first two to four years of work accidents decreased significantly. The mode of operation a day also has implications for injuries. Studies show that the risk of accidents in three-shift operation, as determined factor 1 for morning shift, is 1.79 for the afternoon and to 1.37 for night. Also highlight of accidents occur between the third and fourth working hour, at the end of the shift accidents are less frequent, but more weight. In only mechanical working operations, even with medium and high physical activity, occupational safety is higher during night shifts, while dealing with constant attention and focus on complicated problems is the opposite. Alcohol undoubtedly increases the severity and frequency of accidents.

Motivation (satisfaction) of the work is also an important factor for the occurrence or not of injuries. The nature of work, working conditions and friendly relations in team had a high influence for positive motivation to work, than wages and working hours. Workers who are satisfied with the work and occupied position, assuming accident cited as reason for more frequent personal reasons, while unsatisfied cited as the more common "objective" reasons, related to the profession and the work team.

Prevention of production accidents is aimed at eliminating or reducing the risk of multiple factors

(reasons) for injuries, it is directed primarily towards the most important of them - conditions the risk factors associated with the nature and conditions of production (profession) and training (if necessary and individual insurance) of workers for safe and trouble-free operation.

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E. Dincheva

13 WORK - AGE AND SEX

Study of aging changes in relation to the workload has its place in the prevention of many diseases associated with overloading the body and the development of stress. It is not possible to optimize workload, without complying with the age of the worker. The problem becomes even more complex, taking into account the fact that the rapid changes in the structure of production capacities and introduction of new technologies requires frequent retraining and changing jobs. Opportunities for requalification and training of new job also depend on the age. Actuality of the problem is increasing and of advancing recent demographic changes in the population structure.

Based on age-related changes and the integration of the individual in the social environment are many classifications of periods of working age. One of the most common classifications is that of D. Bromley (1974). According to her, working age divided into the following periods: 1). Young age - from 20 to 25 years; 2). Middle mature age - from 25 to 40 years; 3). Late mature age - from 40 to 60 years; 4). Before retirement age - from 60 to 65 years; 5). Retirement age - after 65 years and 6). Old age - over 70 years. This classification does not always coincide with the adopted legal decrees for retirement.

The large interindividual differences in the process of reducing the functional capabilities of people with respect to age, resulting mainly from the impact of environmental and social environment led to the formation of the concepts of "biological and functional age", ie the actual biological status of cells in various organs and systems and functional capacity, compared at the same chronological group. There is no clear correlation between biological and chronological age, and are pronounced individual functional differences in aging.

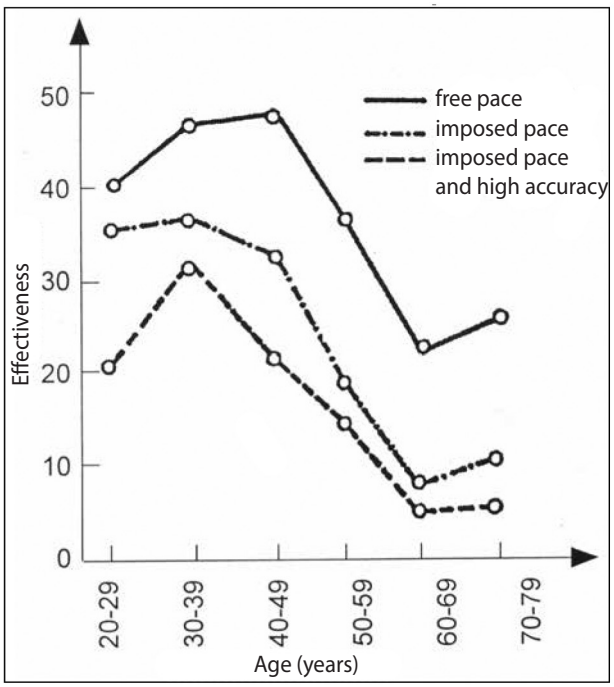
13.1. CHANGES IN PHYSICAL PERFORMANCE WITH AGE

Morphological and functional changes that occur in all organs and tissues in the body in the course of age development, led to the gradual decline of his physical abilities. Body mass of men over 45 years begins to decline (women this process began about 55 years). Height after 40 years also began to decline. Reducing absolute muscle strength starts even earlier - after 30 years, these changes are more pronounced for the large muscle groups of the legs and body and less in the smaller muscle groups of hands. Changes and endurance to static muscular effort. In aging form a peculiar muscle tone.

Along with the offense of supraspinal control due to changes in the CNS are observed and complicated changes in segmental motoneuron apparatus. It was found that in the elderly activity of alpha motoneurons (rapid, phases motoneurons) is reduced, and this at gamma motoneurons (slow, tonic motoneurons) increases. Imbalance between alpha and gamma motoneuron apparatus led to increased tension in skeletal muscles, which reduces the possibility of quick phases movements. With these changes, as well as degenerative changes in bone and joint system is explained reduced possibility of elderly to work at a rapid pace.

Advancing in age changes in psychomotor activity have some essential peculiarities. Typically, the ability to perform simple and uniform motion to a much lesser extent distorted as compared to the changes that occur in the execution of complex movements and actions. For example, actions in the course of which movement has to change speed and direction, usually, with increasing age, significantly delayed. It has been found that with age changes and strategy of execution a movement or action. As the extended time is mainly on account of extension of time pass of central nervous processes (less- or not for the motor time), usually the general balance of time is shortened. To the changed strategy goes that on account of the reduced speed allowed, however fewer mistakes. It was also found that with age, change (slow down) the most significant, such movements and actions with which the worker has at least experience. At age, unfavorable changes in terms of speed of movement is established when

Fig. 1. Results for different age groups depending on the pace and complexity of work



the worker has to work with precise movements in terms of the imposed rhythm, for example. when operating the conveyor **(Fig. 1)**.

Established facts regarding age changes in locomotor activity are important for the organization of labour. It should be borne in mind that changing labour task is detrimental to the worker. When this change is necessary, the new psycho-motor task should not be more complicated and more difficult than previously exercised. It is a new activity in the older worker to start after a period of training and adaptation to changed working conditions.

It is well known that lung volumes and capacities, in addition to gender and height and are highly correlated with age **(Table. 1)**. Along with the vital capacity and decreased respiratory reserve volume. In this is typical and relatively increased residual volume to total lung volume. Moreover, changing some basic indicators of the exchange of gases in the lungs. With age increases alveolar-arterial oxygen differentiation resulting in a reduced partial pressure of oxygen in arterial blood. The latter is most often associated with non-compliance of alveolar ventilation and blood flow to the lungs.

Well-known are also changes that occur with age with respect to the cardiovascular system. These are: reducing the efficiency of the heart as a pump, slow heart rate, decreased blood flow in the coronary vessels and the heart's ability to use oxygen. Rigidity of the large and small blood vessels and capillaries alter blood flow to the organs. Furthermore, considerable changes in the oxygen carrying capacity. This

Tab. 1. Vital capacity (x ± std in l)

Age	Male	Female
20 - 24	4,3 ± 0,3	2,8 ± 0,3
25 - 29	4,2 ± 0,4	2,8 ± 0,3
30 - 34	4,1 ± 0,3	2,7 ± 0,3
35 - 39	3,9 ± 0,4	2,6 ± 0,3
40 - 44	3,7 ± 0,4	2,4 ± 0,2
45 - 49	3,4 ± 0,3	2,3 ± 0,3
50 -54	3,2 ± 0,4	2,1 ± 0,3
55 -59	2,9 ± 0,4	1,9 ± 0,3

is due to changes in heart - vascular system, and the change of the hemoglobin content of the blood, the smaller arteriovenous differentiation of oxygen, the low efficiency of cell respiration. All these changes result in a decrease in physical capacity with age.

The maximum aerobic capacity (MAC) is highest around age 25 and then gradually decreasing. Characteristically, at the age grow increasing and interindividual variation in physical fitness. This is associated with both genetic and with a number of factors such as health, fitness level, diet and not least the influence of the profession and working conditions.

Studies in workers in Bulgaria showed the highest indicators of MAC in occupations with significant physical load - miners and forestry workers, the lowest values in workers from the chemical industry, which most often missing dynamic exercise and strong toxic-chemical factor **(Table. 2)**. Clarifying the impact of the occupation on physical capacity is important in two aspects - to seek professional factor that may modify age-related changes, and secondly how age limits in physical performance make it difficult for older workers to perform their professional duties and affect health them.

For prevention it is important that the load of older people should be dosed with great care and caution, strictly differentiated according to individual opportunities, avoid fling movements, spins and large static efforts. To maintain exercise capacity and delay aging changes it is very important that physical activity in young and middle age. This is particularly important in professions with marked hipodinamiya and hypokinesia.

13.2. CHANGES IN NEURO-PSYCHIC PERFORMANCE WITH AGE

Age-related changes in visual functions are essential for adaptation in work requiring sensorineural and neuro-mental activities. This is mainly due to the fact that although slowly they start too early, when the total neuro-mental performance is high. Knowing their first symptoms and their impact on the performances and the condition of the worker is important for rational organization of labour and

Tab. 2. Maximum oxygen consumption ($\bar{x}$-l / min) and a maximum pulse rate ($\bar{x}$-beats min^{-1}) at various professions

Study group	Age	Max.O_2 consumption	Pulse rate	Authors
Workers from chemical industry	20 - 25 26 - 30 31 - 35	2,37 2,50 2,29	174 165 158	N. Tsaneva at all.
Workers of engineering	to 20 21 - 30 31 - 40 over 40	3,12 3.00 2.80 2.60	187 184 181 180	G. Ganchev
Mechanics in agriculture	21 - 30 31 - 40 41 - 50 51 - 60	2.90 2.80 2.70 2.40	180 172 168 168	L. Mincheva
Forestry workers	to 30 31 - 35 36 - 40 41 - 45	3.26 2.88 2.96 2.81	172 167 170 159	I. Hadzhiolova et al.
Miners	21 - 30 31 - 40 41 - 50	3.40 2.54 2.49	– – –	L. Mintcheva et al.

maintaining the health and performance. Moreover, many modern professions and activities related to monitoring small objects and details or require continuous visual control, the great quantity of information, which requires a over-loading the visual system.

Age-related changes in front of the visual system mainly relate to the changes in the transmittance of light (change in the thickness and shape of the cornea changes in the vitreous, etc.) And to changes in accommodation functions (change in the thickness and density of the lens, reduction the elasticity and mobility thereof, atrophic changes in the ciliary muscle, etc.). These changes begin between 35 and 45 years of age and consist mainly of changes in visual acuity, which is one of the main demonstrated indexes for the capacity of the visual system. For hygiene of labour is important determination of visual acuity for near distance, as a large number of occupations associated with manufacturing, assembly or observation of small details and objects, the distance to the eye is 30-40 cm. In addition to visual acuity of static objects in employment matter and kinesthetic visual acuity, ie the perception of objects that move. Time perception of static and more of moving objects with age progressively increases. Undoubtedly, aging changes in time perception is also due to the changes that occur not only in the front parts of the visual system, but also to circulatory and metabolic changes in the retina that lead to narrowing of the visual field.

With age the elasticity of the lens decreases with which the nearest point of clear vision are increasingly moving away from the eyes. Develops so-called presbyopia (age hypermetrophia). Uncorrected or improperly corrected hyperopia leads to difficulties in the execution of work performed at near distance. To form a clear image on the retina accommodation apparatus is under constant tension. This leads to the development of fatigue, which manifests with visual discomfort, tearing, redness of the eyes, headache. Decrease in visual performance after 40-45 years requires special measures for work places. First, it is to improve the brightness and contrast and reduce glare. It is important to avoid overloading the visual system, which usually happens at the end of the month. Working with small details in older workers is very tiring. Recommended periodic eye examinations to detect the changes occurring in visual acuity and prescription needed glasses. When working on a video display with workers over 45 years recommended visual correction be of seeing the so-called semi-near distance.

Reducing the hearing with age - presbycusis, also has importance for the employability of older workers. The deterioration of auditory function consists in raising the hearing threshold in the range of 4000-5000 Hz. Changes in hearing sensitivity in exposure to excessive noise levels in industrial conditions initially were approximately at the same frequency range and are an expression of such pathological changes - degeneration of hair cells and damage to relevant cochlear structures, i.e. both factors have an additive effect and older workers are more sensitive to noise impacts. Later, with age auditory sensitivity decreases and in the lower frequency bands which leads to deterioration and speech intelligibility, respectively hinder workers communication.

Problems with aging changes in the neuro-psychological performance are very complicated because of the fact that with age the various psychophysiological indicators do not change equally and depend on a number of factors. Firstly these are indi-

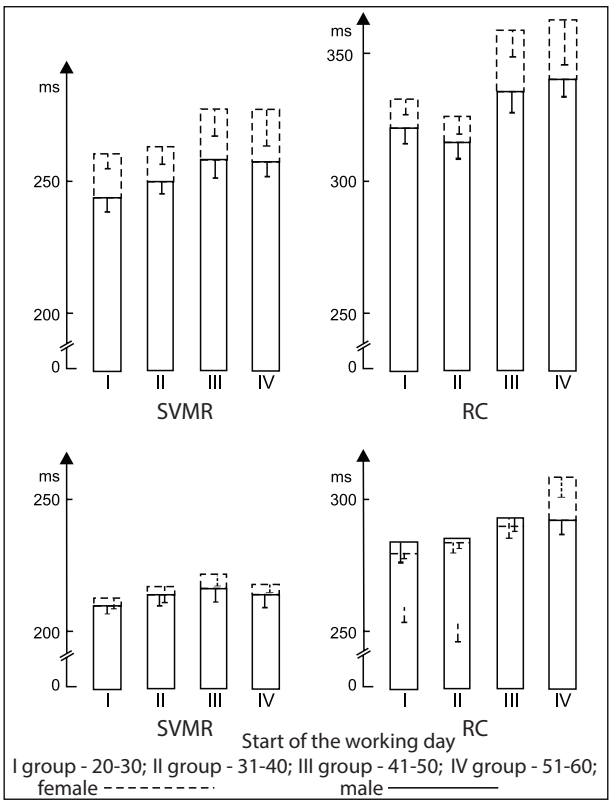
vidual characteristics and type of labour. It is known that most-exert neuropsychological functions remain preserved for a long time. Moreover, along with progressive degenerative changes develop a number of compensatory mechanisms that allow a long time to perform neuro-psychic activity. For example, the time to implement simple sense (visual)-motor reactions (SVMR) starts increasing with age, compared to the time for performance of complex tasks requiring engagement processes of discrimination signals, decision-making and more. Hormonal changes also play a role. Studies show that for example in all age groups after 20 years of age, men have a shorter response time than women. In men, at period of the working age, changes in time of simple reaction are negligible, while the changes in time of complex reactions (RC - reactions of choice) after age 40 are more pronounced. In women, both reactions in this age group are delayed. It should be emphasized that in both groups of persons age changes are more pronounced at the end of the day, when already there are signs of development of fatigue **(Fig. 2)**. Many authors highlight the fact that while older workers to perform more slowly neuro-sensory task, they make fewer mistakes in the appearance of distracting attention factors and the end result may not be worse.

The memory processes with age are not altered in equal measure. The primary memory (when the material is still repeated and is the focus of attention), age changed insignificantly, while changes in long-term memory are demonstratively expressed. Reducing the ability to memorize in the process of aging is also related with adaptability responses - age begin to prevail components of semantic and logical memory than mechanical. Semantic components are stored longer and acquire a relatively large share of total memory.

Higher intellectual processes also show dynamics with age, but the changes occur relatively late. A number of authors find that they are kept and even improved to 50 years of age. As has been shown improvement in solving arithmetic problems in 60-69 the men, and 50-57, the women. The explanation of this fact is most often associated with improving the functional condition of the body after completion of climacteric processes in previous decades and stabilization of the neuroendocrine regulation.

The age changes in neuropsychological performance should be considered in addressing issues such as education, training and retraining of workers, ergonomic problems of work places, the problems associated with the normalization of labour, which is directly related to preserving the health and employability of workers.

Fig. 2. Age changes in SVMR and in RC



13.3. ADAPTATION OF WOMEN TO WORK

The need of special attention to women's work stems from the fundamental physiological differences between the sexes and the underlying mechanisms that determine biological links between environment and health status. Moreover, the woman there are special periods such as period of pregnancy, lactation, hormonal changes of sudden exit of fertile age when they are more vulnerable. Besides professional commitment, housework and upbringing of children, and a number of psychosocial problems are additional stressors.

The woman has less physical strength than men - it has a smaller skeleton, shorter limbs - an average of about 7% of men. The smaller muscle strength is due to the smaller muscles and the greater amount of fat. The heart is at least about 10-15%, which results in the smaller stroke volume. In load stroke volume increased less and compensation at the expense of fast heart rate. At the same load women increased almost twice pulse rate. When thermal load woman also corresponds with a higher pulse rate. For physical efficiency is important, and the fact that the woman has a smaller chest and lower indices of lung capacity by about 15%. Hemoglobin in women is about 25% less quantity compared to men and the absorption of oxygen from the lungs is less.

These differences determine the lower working capacity of women - about 20-25%. The woman has only 55-65% of males opportunities for lifting and carrying weights. Therefore, women should not perform heavy physical work. In Bulgaria norms for different categories of physical work are about 0.5 kcal/min lower than those for men. During pregnancy, women have certain restrictions for heavy work due to fatigue. After diagnosis of pregnancy a woman should be transferred to lighter work. For women in production this problem exists, in connection with pregnancy and in relation to the menstrual cycle. Also early initiation of hard work is harmful to the young girl. Forced work posture leads to stagnation in the pelvis and favour the development of the reproductive system diseases.

No reliable data to show significant psycho-physiological differences between men and women. Individual authors emphasize some mental instability in women, greater emotionality and irritability, as well as no-precision approach when dealing with some individuals. It is known that women have a longer response time and a slower rate of voluntary movements. However, as all these activities largely depend on training and professional training, it is difficult to argue that these differences are physiologically determined (known practice, both in our and other countries that workers take jobs in industry often without any prior training and qualifications). In terms of skill, there is evidence that activities requiring fine adjustment attach better on women.

In literature there are no data to suggest that women are more sensitive to various toxic and chemical substances - toxic powders, aerosols, gases, compared with men. It has been shown, however, that during pregnancy and during lactation, certain toxic substances are particularly harmful for women. It is known adverse effect of lead, mercury, organic solvents and the like. These substances pass through the placental blood flow and harm the fetus. So there are laws and regulations that protect women during pregnancy and motherhood.

A number of factors in the work environment may adversely affect the female body to a greater degree. Important are the issues of security of women working in the conditions of ionizing radiation, particularly during the period of organogenesis in pregnant. The genital area of a woman apart from hard work affected by general vibration, infrasound, intense electromagnetic fields.

With regard to night work is considered that there is no physiological causes women to suffer harder than men. That women in night shift work decreased productivity more than men, is associated mainly with insufficient and disturbed sleep - social situation of women doing night work with them especially tiring, and therefore should be limited.

In terms of accidents in the industry, there is evidence that women make fewer mistakes and accidents than men. Normally accidents in enterprises are found in younger and older women compared to those of middle age. In domestic accidents, things are opposite, which again is due to their social engagement with family.

There are no data to suggest that outside of their particular periods, women are more susceptible to diseases. On the contrary, men have a higher incidence of mortality in all age groups - higher in them and fetal and infant mortality, the greater the mortality of inborn disorders. These differences indicate a role of genetic factors as well as in susceptibility to the development of certain diseases and certain hereditary diseases associated with a single chromosome in men, such as color blindness. It is also known that men risk of cardiovascular disease is greater than for women. It is considered that the younger women risk of cardiovascular disease due to the protective role of female sex hormones, especially estrogen. This explains the fact that the incidence of cardiovascular diseases in early menopause after ovariectomy after menopause is higher. It also assumes that a large amount of lipoplasmic proteins with high density in women have a protective effect on the development of cardiovascular diseases.

Significant interest is answering the question whether there are differences between men and women regarding the development of reaction to stress. During the rest or inactive state no differences in urinary catecholamines, but in situations of experimental stress - implementation of frustrating cognitive tasks, tasks that measured the response time, after exams, etc., excretion of adrenaline in men increases to a greater extent than women. Regarding the excretion of norepinephrine absent such differences. There are data showing that there were no differences between men and women in the development of stress responses in certain psycho-social conditions. For example, in cases where women exercise so-called male professions - drivers, responsible management activities, etc., differences in cortisol levels between the two groups were insignificant. It is suggested that psychosocial factors play an important role in the regulation of endocrine activity.

Given, more active involvement of women in difficult and responsible jobs, these data are important to physicians and psychologists of work in the development of medico-preventive measures - modes of work and rest, stress control and stress reducing programs, and to solve the problems of each case.

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13.4 OSTEOPOROSIS

Osteoporosis (OP) is the most common metabolic bone disease that according the World Health Organization occupies fourth place after cardiovascular diseases, cancer and diabetes in medical, social and economic significance.

OP is defined as the systematic osteopathy characterized by reduced bone mass per unit volume and violation of microarhitectonics of bone tissue.

Histomorphometric OP is defined as a reduction of the total amount of normal bone formed. Record the simultaneous reduction of the mineral substance and the bone matrix, and the ratio between them remains unchanged. The bones become porous and fragile, skeletal strength and endurance decline, which in turn significantly increases the risk of fractures with minimal trauma. Bone loss, most often occurs without symptoms at the onset of the first fracture.

Classification of osteoporosis: Osteoporosis is defined as *primary* or *secondary* based on the absence or presence of certain diseases, surgery intervention, and/or taking certain medications.

- ***Primary osteoporosis (involutional)***
  Given the clinical features, hormonal changes and the relationship of the disease with age and menopause are distinguished two types of involutional OP:

Type I - postmenopausal osteoporosis - it manifests itself in women 50 years of age and is associated with acute estrogen deficiency in menopause. Determined by the accelerated bone turnover, usually resulting in increased bone resorption.

Type II - senile osteoporosis. Occurs with age, affecting equally men and women over the age of 70.

Determined in age-related decrease in the body of the functionality of osteoblasts and secondary hyperparathyroidism, probably dependent of impaired calcium absorption in the small intestine. It is characterized by reduced bone turnover - relatively unchanged or reduced absorption, coupled with low bone formation

- ***Idiopathic Osteoporosis*** - rare forms of osteoporosis in children and adults (juvenile, premenopausal in women and senile in men)
- ***Secondary osteoporosis***, which is mainly to:

Hormonal disorders (amenorrhea, hypogonadism, hyperparathyroidism, hyper-tireoidism, hypopituitarism, insulin-dependent diabetes); **medication** (glucocorticoids, heparin, anticonvulsants, thyroid hormones, high doses of aluminum-containing antacids, immunosuppressants); **diseases of the digestive tract, cancer and genetic diseses** (malabsorption, chronic hepatitis, primary biliary cirrhosis and alcoholic, rheumatoid arthritis, leukemia, myeloma, lymphoma, and other neoplasms, osteogenesis imperfecta, homocystinuria, Marfan syndrome and Ehlers-Danlos).

Epidemiology of osteoporosis and related fractures. Medical and social consequences. OP can develop individuals of all ages, but most affected are women after menopause. Epidemiological studies indicate that 45% of women over 45 years of age and 15% of men over 50 suffer from osteoporosis.

The spread of OP grow worldwide. In the US, for example, according to the NHANES III cases diagnosed OP at 5-8 million for the period 1988 to 1994, rose to 10 million in 2000, and another 18 million Americans are at increased risk due to osteoporosis because diagnosed osteopenia (low bone mass).

In the countries of the European Union suffer from osteoporosis 40 million, representing 12% of the population.

In Bulgaria, according to a representative epidemiological study osteoporosis determine at 20.5% and osteopenia in 32.5% of the surveyed postmenopausal women. Extrapolating these data on the general population, the authors assume that about 820,000 women with low bone mass and increased risk of fractures.

It is assumed that 50% of women and 25% of men over age 60 will suffer at least one fracture during the remainder of his life. Patients over the age of 70, the curve of dependence - reduced bone / fracture risk is already exponential. It is important that in over 50% of the osteoporosis fractures are the result of a minor or moderate injury, as in the majority (76%) are due to a fall from a standing position or lighter falls.

Compared to the 70s, during the period 1989-1991 fractures of the limbs have increased by 14% (22% of women and 1% of men). It is assumed that if in 1990 worldwide were registered 1.7 million frac-

tures due to osteoporosis, then in 2050 they are expected to reach 6.3 million and in the European Union predicts that over the next 50 years will increase hip fractures more than 2 times - from 414 000 to 972 000.

The high prevalence and subsequent osteoporotic fractures determine the clinical and social significance of the OP (reduced bone mass, itself no clinical signs).

The most frequent and severe complications are **hip fractures**. It more than 50% of cases require prolonged hospitalisation, and 10% - permanent home patient care over a period of year. After severe hip fracture, approximately 11% of patients become disabled completely (only 30-50% recover completely).

The established high lethality due OP and fractures is associated mostly with that of the femoral neck fractures - 5-20% of patients die from complications during the first year. Generally such a fracture reduces life expectancy by 12-20%.

Second in severity and significance are **compression fractures of the vertebral bodies**. Established at 36% of older women. Spinal deformities due to compression fractures are the cause of persistent back pain, limited physical activity, expressed chest hyperkyphosis ("widow's hump"), loss of height, and 5% - reaching total disability.

Complications after **fracture of the radius in loco tipico** not so heavy, but in 29% to 44% of these patients establishes much pain, and 36-40% of them complain of weakness and numbness of the hand and after a year of trauma.

Due to the high frequency and severity of complications of osteoporosis fractures, economic losses associated with OPs are huge and are increasing every year.

In 1999, medical costs for treatment of fractures due to osteoporosis (without working day losses) common to Europe and the United States were 27 billion USD. In the next 50 years is expected to double these costs.

Risk factors for osteoporosis. Bone mass is accumulated in childhood, rising to a peak in late adolescence (so called peak bone mass - PBM) and after a period of stability began to decline. In this regard, two major pathogenic determinants of OP:

1. Gaining a relatively low peak bone mass (PBM) in the years of childhood and adolescence. In this case, even minimal bone loss with age will lead to low bone mass and high osteoporosis risk. It was found that PBM set at 50-70% (respectively for trabecular and cortical bone) bone mass in adulthood and is therefore predictive of the first order in etiopathogenesis of osteoporosis.

2. Significant bone loss (such as value, speed and duration) in the menopause and/or at a later age.

The level of bone mass, both in childhood and in later life is determined by a number of interrelated genetic, hormonal and behavioural factors affecting varying degrees accumulation of PBM or subsequent bone loss.

- ***Phenotypic and genotypic characteristics.***

As highlighted female sex was significantly affected by the OP and related fractures. These differences can be explained by the fact that men first accumulate significantly greater PBM because the 2 additional years of prepubertal growth and long period of pubertal maturation. On the other hand the absence of menopausal equivalent leads to a significantly later and weak age related bone loss.

Studies in children and adults demonstrate not only sexual but also expressed racial disparities that persist throughout life and set higher OP and fracture risk in individuals of Caucasian and Asian patients compared with those of Negroid.

Bone mass is under strong genetic control, evidence of which are based both on surveys of pairs of twins, as well as data from family studies. Accepts that up to 70% (50-90%) of the variability in bone density due to heredity. Made attempts to determine polymorphisms in several genes related to bone mass and / or fracture risk. These are: vitamin D receptor gene (VDR); collagen type 1 α1 gene (COL1A1) and connecting it proteins; estrogen receptor alpha gene (ER-alpha); transforming growth factor beta gene (TGF-beta); interleukin-1 beta gene and the like.

- ***Body constitution:***

Increased osteoporosis and fracture risk is associated with lower body weight, reduced thigh circumference, decreased muscle and fat mass and so on. For example, women with a higher body weight have lower bone loss, as weak and tall women develop more frequently osteoporosis.

- ***Factors of lifestyle:***

Nutrition - inadequate import of calcium and vitamin D, as a number of other minerals and vitamins - Mg, Zn, Cu, F and vitamins K, C, B_6, also the reduction in Ca absorption in the digestive tract due to decreased synthesis of vitamin D with age, a violation of the ratio of Ca/P due to excessive intakes of phosphorus in the diet.

Reduced physical activity and immobilization: Already in 1892 J. Wolff proves insufficient physical exercise (gravity or mechanical) adversely affects the bones, and bone strength and bone mass increases in response to increased physical activity. Today it is assumed that the adaptation of the bone to the effects of mechanical impacts occurs with changes, both in its size and in its structure. It is also under-

lined that bone mineral density in a certain skeletal point is associated with muscle strength in regional tissue mass. Known is also the larger osteogenic effect of locomotor activity during growth in comparison with that during adulthood.

Some negative behaviours (smoking, alcoholism): Smoking is a proven risk factor for bone metabolism. Women who smoke regularly are lower bone mass compared with nonsmokers. Smoking has been associated with lower levels of estrogens and early menopause. And it assumed that nicotine inhibits the function of osteoblasts.

The negative impact of large amounts of alcohol on the one hand due to a toxic effect on bone cells, the other-coming in from alcoholism nutrient deficiency, malabsorption syndrome and expressed hepatic impairment.

- ***Features of hormonal status:***

These are related to estrogen deficiency before and after the onset of menopause (amenorrhea, hypogonadism, menopause);

- ***Some chronic diseases or drug therapy:***

Endocrine, rheumatic, hematological, gastroenterological, nephrological and other diseases, organ transplantation, treatment with glucocorticoids.

Diagnosis of osteoporosis: The World Health Organization has established diagnostic criteria for osteoporosis and osteopenia, based on measurement of bone mineral density (g/cm^2) with DEXA (dual-energy-X-ray-absorbiometry) at the femoral neck - so-called "Gold standard".

The presence of osteoporosis is demonstrated by means of densitometry established indicator T-indeks - magnitude of the standard deviation (SD) of the normal average mass of bone in young healthy women in white race[1].

to -1 SD - standard

on - 1 SD up to - 2,5 SD - osteopenia

below - 2,5 SD without fractures - osteoporosis

below - 2,5 SD with fracture - advanced severe osteoporosis

Prevention of OP. Osteoporosis is a disease of old age, but it has its genesis in childhood and adolescence, and therefore primary prevention of OP is aimed at optimal skeletal growth and mineralization from an early age. It is accepted that one standard deviation (10%) higher PBM reduces the risk of osteoporotic fractures in elderly people by about 50%.

Secondary prevention is necessary for all women after menopause, elderly people of both sexes, and for people with chronic diseases, provoking reduction of bone mass.

Prevention of OP is complex and includes:

– A balanced diet, providing important for maintaining bone metabolism nutrients (minerals, vitamins, proteins); failing that, supplementation of the diet with preparations of calcium and vitamin D, or a combination supplements containing besides calcium and vitamin D, Mg, Zn, vitamins C, B_6.

– Exercise

– Correction of some behavioural risk factors: smoking cessation and reducing alcohol consumption.

[1] Men diagnostic use the same absolute values of bone density and T-index as in women.

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14 PROFESSIONAL TRAINING AND WORK OF PERSONS WITH DISABILITIES

In different countries around the world there is a different age limit for completing school students in disorders - in some countries it is 18 years in most 21 years. In many countries also permitted and attending additional class (i.e. extra class), thanks to which students with disabilities who are not yet ready to leave school, work hard to master the important skills for independent living and focus on the utilization of labour and professional skills and habits (Sweden, Finland, USA, etc.).

With the creation of the first educational institutions for children with disabilities, placed and early labour training and the fundamentals of vocational guidance. In the first school for the deaf opened in 1760 in Paris, in the school for the blind, founded in 1784 in Paris and in the school for children with mental disorders, also discovered in France in 1841, began to teach subjects like needlework, modeling, etc., which are directly related to the future professional development of persons with disabilities. So gradually began systematic preparation for future professional activity in children with disorders.

In 1984, Madeline Will (USA) developed a model for categorization of graduate school students with violations. They are divided into three main groups according to their needs at graduation and transition to adulthood:

1. Students who will no need of extra care and service after graduation and can alone successfully make the transition to adulthood and exercise profession - students with one disorder usually mild: for example, visually impaired, hearing impaired, students with mild intellectual disability, students with mild neuro-somatic disabilities.

2. Students who will require special care and attention for making the transition to adulthood and addiction professional activity for a period of time - students with combined disorders or with moderate and severe disability.

3. Students who will need protracted, perhaps permanent care and services for the transition to adulthood and can not be completely autonomous and independent in the performance of professional duties - students with multiple disabilities.

Appropriate vocational guidance includes detailed interview of person with violations. If the person is nonverbal or for other reasons can not only participate in the interview, then the necessary information is collected from parents or others who know him well - the class teacher, other teachers, neighbors, parents, etc.

Besides conducting the interview, the assessment should undertake a study of a number of documents and materials such as:

- school diaries, individual curriculum and program of person for the last few years.
- the medical file of the person - recent survey of vision, hearing, intelligence, general physical and neurological condition.
- psychological record of a person - is there documented behavioural problems and what therapy is conducted, how the person perceives their own violations, is there a realistic idea of their limitations, are there adequate expectations for the future.

Model list of questions to assess a person with disabilities

1. Tell me about yourself and your family.
2. Where are you going to live after graduation?
3. What are you going to do after graduation?
4. What job would you like to do?
5. How are you going to get a job?
6. What skills mastered in your school?
7. What skills have yet to learn?
8. What do you do best?
9. What are you doing worst?
10. Please describe how and to what extent do you see / hear / move, etc.
11. Have you worked before? Where? What have you done?
12. What are the obligations you have at home?
13. What are your goals for this year? And the future?
14. What help will you need to achieve your goals?
15. How would you describe yourself (use 5-10 adjectives that suit you)?
16. Which activities you like in school?

17. What activities outside school you like?

18. What do you do in your spare time?

19. Own you prefer to work or among other people?

20. What work do you prefer - inside a building or outdoors, in the open?

21. Which do you like more - to work with people, with data, with the animals or objects?

22. Describe how you imagine the perfect job for you.

14.1. VOCATIONAL REHABILITATION OF PERSONS WITH DISABILITIES

Appropriate vocational rehabilitation requires practice good professional training. At the same time it is important to know the possibilities for obtaining it. In modern society there at least seven options for obtaining professional qualification:

Special schools for children with disorders. This is the oldest form to master the profession both internationally and nationally aspect. For example, the creation of the first schools for deaf, blind and mentally retarded in Bulgaria respectively in 1898, 1905 and 1936 is advocated the absorption of various crafts. The school for the blind learn basketry and brush-making, later introduced the study of massage and tuning of pianos, in school for mentally retarded taught needlework. Today, besides the traditional crafts, to master the basics and of advanced professions, working with computers and modern special equipment.

Rehabilitation centers. They are intended primarily for persons where violations occur in old age. It provides both elementary rehabilitation, mastering living skills and vocational rehabilitation.

Specialized centers for vocational rehabilitation. This form of professional training enjoyed success in many countries - especially in the Nordic countries. It involves creating a special center for persons with certain types of disabilities or a combination thereof, where to teach different professions suitable for the type of disability. In Bulgaria these are social educational and professional institutions (SEPI) for persons with intellectual disability. In our country there are a total of 10 such institutions where people with intellectual disabilities master professions like tailoring, upholstery, cooking, painting, printing, furniture, women knitting, agricultural activities and more.

Mass vocational schools. This form is applicable for persons with visual, light neuro-somatic somewhat with hearing disorders. In her disabled do in ordinary vocational education and master the profession on a par with persons without disabilities. Suitable professional schools: music school and medical college to master the massage of persons with visual impairment, trade schools for people with hearing disorders, etc.

Through short university programs. This is a new form of professional training, popular in Western Europe and the USA. By university program aims acquiring knowledge and experience in a specific profession, such as computer technology and others.

Through vocational courses. These courses are created for people with a certain type of disability and have the task to teach students specific skills. Examples of such professional courses are: sewing, knitting, carpentry, piano tuning, repair of appliances, upholstery, etc. Usually these courses are organized by an organization of disabled people - for example, the Union of the Blind, Association of the Deaf, Union of Disabled People.

Through international programs. A number of educational and rehabilitation facilities around the world offer training to master a profession or qualification of persons with disorders, and exchange of students. Thanks to this form of vocational training disabled people can master entirely new profession or improve their skills in their profession known as familiarize themselves with modern trends in its exercise - for example, to gain an idea of the innovations in computer hard- and software for the blind and others.

14.2. LABOUR FOR PEOPLE WITH DISORDERS

In Bulgaria at the end of 2000 the number of persons with permanent disability (disabled) is 240,000. For Sofia only their number is more than 30 000 people. The basic legal documents regulating their rights and social benefits are: the Law on Protection, Rehabilitation and Social Integration of Disabled (as amended and supplemented by the 38th National Assembly in March 2001), the Code of compulsory social insurance from January 2000, the social assistance act and its implementing rules, the Regulation on expertise of efficiency.

Career development is a "Process by which the individual defines their professional values and carries significant professional activities. This process also includes the assessment of interest and the detection of potential "(Simpson F., 1986). It creates a feeling of satisfaction, allows free communication and not living in isolation, ensure financial stability and independence, creates self-esteem.

The main factors that determine employability of persons with disorders are:

Personal adjustment. To be a personal adjustment, the person must be able to observe good personal hygiene, dressing appropriately, have good manners and behaviour.

Social adjustment. It requires mastering the face of such skills will make him an effective member of a group or team.

Public adjustment. This factor implies adaptation of the person to the basic rules of society. For this purpose, it must be able to handle money, shopping, using public resources, etc.

Professional adjustment. The person to master all those work skills and habits that allow him to efficiently perform the professional activity.

Employability of people with disorders are a few:

Job's working place together with persons without violations. This means that the person is increasingly competitive and able to deal independently with various professional tasks as well as persons without violations. In support of this opportunity in different countries apply support measures in the face of two systems: a quota system and the volunteers. The quota system is an obligation of the employer to set aside a certain number of jobs for the disabled. This system has been successfully applied mainly in the 70s of XX c., but then began to decline. In Germany, for example the legislation provides each company with more than 16 work places to allocate 6% of persons with disabilities. The system of volunteers is more flexible. Her goal is to encourage employers naturally to employ persons with disabilities, without being obliged by law. For example, if an employer hire a disabled person, for one year his salary is paid by social funds. During this time invalid has the opportunity to demonstrate their professional skills and be approved. After the first year, if the employer decides to keep working person with violations, he had himself assumed the payment of his earnings.

Job's working place with persons without disturbances, but with help and support. This is an alternative form of work in a company, institution or workplace with persons without disabilities, but provide additional assistance from a specialist. This extra help can have different dimensions - for example, a one-time demonstration of the performance of a specific task or activity in the workplace, to continuously supervise and monitor the work. Specialists who can provide this extra help are usually: specialist for vocational training, social worker, special education teacher. There is a post "professional adviser" (job coach) in the US, Germany and others, whose task is to open new employment opportunities for persons with disabilities, to help them accustomed to tasks which perform in the workplace, discuss with them any problems, to mediate between the individual with disorders and employer.

Working in special enterprise or special environment for people with disorders. This system of vocational rehabilitation is the oldest historically - emerged in Europe in the late eighteenth century when to different schools for children with disorders (mostly blind and deaf) are created workshops (later created special autonomous workshops). Now in Bulgaria special enterprises (from 1952 for the blind) exist at the Union of Blind - i.e. "Success" and the Union of the Deaf - i.e. "Quiet work". In practice, the first special enterprises in the country occur around the middle of the twentieth century. They should be recruited only individuals with one disorder without additional disabilities.

Working in "enclave". This is a system in which a small number of persons with impaired work together in an ordinary working environment, among people without disabilities, are responsible for a certain part of the production process, eg., manufacture or assembly of certain detail. They work collaboratively with others with a similar condition, but get in continual contact with colleagues without disabilities. These enclaves are guided by a person of rehabilitation facility or institution for professional training, but could this be a person from enterprise with some training in special education.

Traveling work teams (groups). These are working groups of people with similar disorders who travel together and perform certain work under contract with various employers. Usually they include "professional adviser" or a social worker.

In a modern world for people with disorders should constantly seek new ways to professional appearance. The change of society towards persons with disabilities, as well as modern scientific-technological and information revolution, undertake in the search for new directions and strategies. Their implementation is necessary:

- **Studying the experience of different countries.**
- **Compliance with national traditions and possibilities of updating.**
- **Analysis of the status and trends and development of professions.**
- **Understand and use the experience of successfully realized in the profession persons with disabilities.**
- **Introduction of employers with professional capacity and realization of persons with disabilities.**
- **Promoting the professional capabilities of the disabled in society.**

Although often in the media are exported some

common issues related to persons with disabilities is very low emphasis on their ability or success professionally. This creates a false idea in society that disabled people are helpless, unable to work or vice versa - there is a lack of conversance about their potential. By consistently delivering in the media of the relevant information for the professional training of disabled people through visual demonstration of coping with the mastered their profession, can change incorrect perceptions of society for persons with disabilities.

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K.Nikolov

15 MANUFACTURING ENTERPRISES - ARCHITECTURAL BUILDING CONCEPTS AND REQUIREMENTS. VENTILATION.

15.1. ARCHITECTURAL AND PLANNING SOLUTIONS AND REQUIREMENTS

Creation of favorable working conditions at production enterprises is determined largely by the appropriate planning and architectural and structural design solutions. The last created material organized environment, the object of spatial resolution are linked in a single system factors in the production process and the conditions of work and rest, that are created for employees. Industrial architecture that is highly targeted technological certainty, observe and reflect the diversity in the formation of the production environment.

The design is based on mandatory construction-technical norms in line with hygiene standards and sanitary requirements and rules. Architectural and planning solutions in determining the location of the manufacturing enterprises of structure schemes and plans of the territories in the country in compliance with current regulations. The structure schemes providing arrangement of area, corresponding to socio-economic development guaranteeing environmental protection. The structure plans include: general - determining the predominant purpose and method of development of the different structural parts of the territories; and detailed - specifying spatial planning and development of settlements and lands, and settlement formations.

In determining the most appropriate **location** of production area reflect a number of factors: topography of the area, the direction of prevailing winds, so called "Wind rose", weather conditions, existing background concentrations of prevailing harmful substances.

It requires compliance with the permissible sanitary-protection zones (distances) of production or service facilities or sources of harm to the nearest housing, food, health, education, children and others objects, subject to health protection or to the border of the settlement regulation depending on the nature and capacity of the specific proceedings.

Do not allow the construction of industrial plants in: residential areas (except for small objects no pollute environment and at objects identified by detailed plans), water catchment areas, nature reserves and protected areas of dams, historical and archaeological monuments, resorts, areas around special objects.

Positioning itself industrial area is done by design aiming at appropriate **functional zoning** of the industrial area - such disposal of the main workshops, auxiliary, service, warehouse and administrative buildings, in which should not create conditions for harmful effects on workers of manufacturing process and the emergence of ecological failures to the natural environment.

By developing a general plan of enterprise now solve issues in planning the routes of the railways and internal factory roads, underground and above ground installations, the profile of the area and the overall development of the territory - landscaping using plants with sanitary-protective qualities and sustainable to harmful substances.

The buildings of the individual subobjects can be deployed separately, to block, to form a semi-enclosed courtyards or enclosed on four sides yards, while besides technical and economic feasibility of such decisions into account and hygiene, fire protection requirements and those in occupational safety.

Compliance with the construction-technical norms precludes adverse shading of two opposite buildings.

In functional and spatial architecture design of the Industry building itself and is looking implementing all hygiene requirements and conditions for safe operation in terms of:

1. Ensuring adequate **natural lighting** in accordance with the envisaged category of visual work.

Natural lighting in industrial buildings is carried by side (through windows) and upper (through glazed roof panels or glazing of different height roofing sheets).

Overhead lighting is only possible in one-storey buildings in the upper floors of multistory buildings **(Fig. 1)**.

Side lighting using windows with different design that depending on the nature of the industrial building is mounted on the front walls as targets

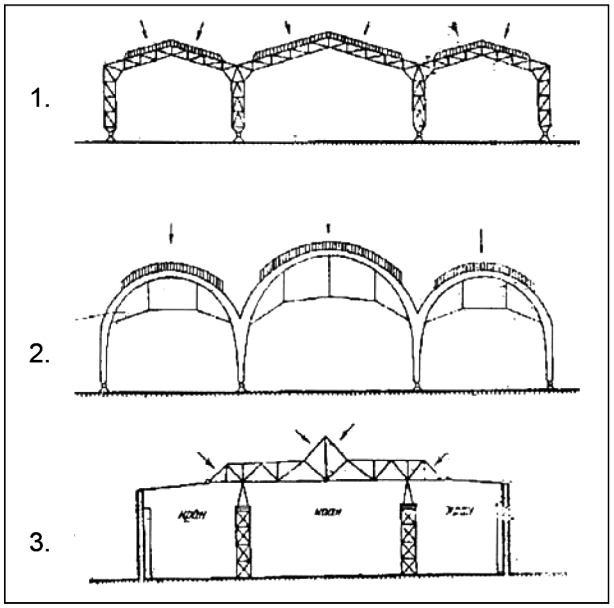

Fig. 1. Schemes of three-navicular-hall buildings with steel structures
1) transverse caterpillar lighting;
2) direct caterpillar lighting;
3) with ridge and attic lighting.

glazed surfaces, vertical or horizontal glazed strips, single holes arranged in rows or checkerboard. Their dimensions are selected according to specific architectural construction decision and the basis of existing regulations. The windows can be opened in different ways, such as open windows whole or in part. Widely use a glazing of glass bricks and glass profiled panels that besides giving diffused light, have good insulating properties.

In large-area buildings is arranged overhead lighting, which may be in the direction of the roof ridge, transverse to the ridge or in combination. Realized with the aid of fonari superstructures of various forms with respect to a certain width and height and inclination of the glass plates from 45 ° to 90 °. Envisaged so-called shedovi (saw-formed) roofs that give through vertical or inclined glazing uniformly good natural lighting **(Fig. 2)**.

The ceiling lights have great advantages and are widely applied in the construction of industrial buildings. It should not however be allowed direct entry of solar rays on work places, constituting a threat of overheating microclimate and intolerance for the body of the worker.

1. Create conditions for natural **ventilation** of production buuildings through appropriate structures of openable windows.

2. Selection of appropriate **construction systems and materials** for achieving hygiene standards for microclimate of work places, create optimal heated volumes and avoid air flows.

3. Anticipate architectural and construction activities for effective protection against **noise and vibration**: walls with the necessary sound insulation qualities, separation of areas with high levels of noise through the buffer stock and other areas, design of lightweight insulating partitions and screens, separation of soundproof cabins for remote control of technological processes.

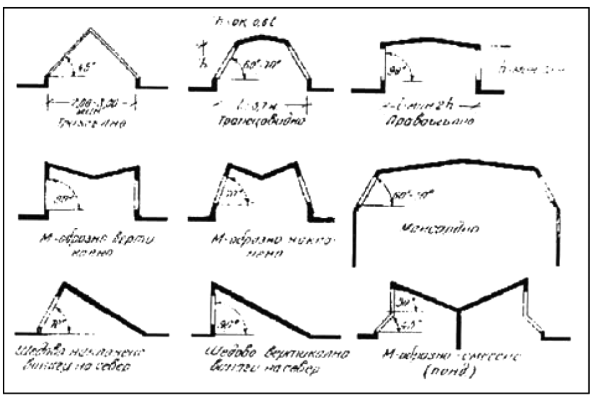

Fig. 2. Basic forms of upper lighting

Regulatory norms pose significant requirements for **construction products**. Construction products for use in construction are usable only if it does not endanger the safety and health of people, possess characteristics, that suitable the envisaged for them use in construction for reasonable working life, are properly designed and executed and meet the essential requirements for buildings. These requirements which may affect the characteristics of construction products are: mechanical resistance and stability, fire safety, safe operation, health and environmental protection, hygiene, noise protection, energy saving and heat insulation.

4. Ensuring legally necessary **premises for sanitary service** of workers.

Manufacturing buildings can be **one-story, multi-story, combined**.

The advantages of one-story buildings **(Fig. 3)** are: pass of the production process at one level, creating facilities for maintenance, repair and reorganizing; maximum load-bearing capacity of the floor; the possibility of creating lightweight structures for the building; providing good natural lighting, placement of lighting openings in the roof; convenient transportation and conditions for the rapid evacuation of personnel; possibilities for maximum spatial combining of the premises.

The disadvantages of the one-story buildings can be underlined: the need for larger construction areas within the industrial area, longer installation networks and communications, greater heat loss and more.

Multi-storey buildings are with smaller floor area, with more compactness of production facilities, with lower heat losses, better insulation and drainage of roof construction.

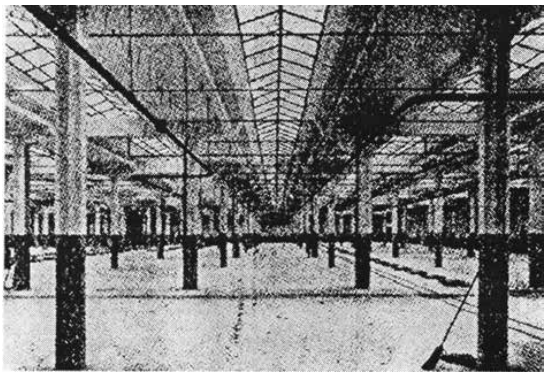

Fig. 3. Inner view of low one-story building

As their disadvantages are: greater difficulties to achieve the parameters of natural lighting, limited height of floors and installation of heavy machinery and equipment, a greater percentage of additional service area, greater difficulties in evacuation, supplying and repairs.

The one-storey buildings are designed and implemented if the assessment proves that the floor construction is contrary to the technology, unreasonably expensive construction works and contrary to hygiene and fire safety requirements.

The production buildings are designed in rectangular or square shape without unnecessary bends. Avoid designing of prominent building elements, canopies, window steps - especially in enterprises with dusty processes.

The architectural design on the **height** of production premises into account the dimensions of the machines that will be installed, at the required height for installation and repairs, heights required for crane and lifting equipment, requirements for safety at work and fire safety.

The floors in the production premises must be secured against slipping, are incombustible and current no conductivity (in explosive processes and using flammable liquids), are resistant to abrasion (movement of vehicles on them), are not damaged by aggressive substances (when work with acids, chemicals, oils, petroleum products, etc.).

Essential to create favourable working conditions is appropriate decision of interior furnishing of production baildings and exterior.

The interior determines the impact of compositional structure of the premises, the colour of various elements, lighting, visual communication (signs, directories), the distribution of mobile equipment. The coloring of the premises must comply with the positive impact, that should be on the psyche of the worker. The floors are stained in neutral tones and the columns and walls - in brighter.

Premises with cold mode of production are painted with warm, mostly yellow tones and in hot industries avoid staining in bright colors.

The coloring of machines and equipment, except in accord with the interior, should help to create safe working conditions - creating a contrast between control and working organs and the background on which projected.

According to statutory requirements, stained in certain colors technological pipelines, communications for heating, fixtures and more.

Architectural decisions relating to the **exterior of the industrial building,** as an additional treatment of the facades, surface treatment of fencing and construction elements, the color of the facades, provided lighting, signs and logos associated with aesthetic searches of the designer to create unity between the material nature of industrial architecture and aesthetic form. When designing the production project dropped self-demandable of in itself unnecessary and extraneous architectural forms and decorations, but works well thought out and rational composition, while aiming at the most appropriate and most economical solution.

Safe operation of objects depends on the properties and quality of **building materials**. Architectural and construction solutions must take into account the fact that the implementation of a number of building materials emit harmful substances, that are expressed most strongly in a specific process. Also, in case of fire some materials emit highly toxic gases, leading to a threat to health and life and rise to serious environmental pollution.

Construction materials as the most common classification can be divided into:

1. Natural stone (granite, rhyolite, syenite, marl, dolomite, limestone, marble, etc.), used in cladding, some floor coverings, aggregates for concrete , mosaic and more. In the application emit dust containing different percentages of free silica, sand, small fragments.

2. Ceramic building materials and products - bricks, tiles, shutters, cladding tiles, acid resistant and fireproof products, stoneware pipes, tiles, porcelain and more. Harmful emissions released during their application are: dust, pieces of undegraded waste, ionizing radiation (such as zirconium sand).

3. Materials of glass melts (flat glass, profiled glass, glass vials, glass fibres, gas-glass) and of molten rock (basalt, marl, slag pumice). Detached dust and undegraded pieces.

4. Inorganic binders - plaster, lime, cements, ashes of TEPP, inert additives, waste from asbestos production. Release dust containing silica, dust with toxic action, dust containing asbestos.

5. Concrete and solutions, asbestos cement products. Release dust containing silica, asbestos, glass and mineral fibres.

6. Metals - ferrous and non ferrous. Emit different hazards depending on their application - dust, metal aerosols.

7. Wood and wood construction materials-planks, flooring, doors, windows, beams and more. Set aside in their application dust, sawdust, vapour from paints, varnishes and others.

8. Synthetic building materials - resins, plastics, paints, hardeners, stabilizers and others. Separate vapors, aerosols, smoke, soot, gases with toxic, irritating and carcinogenic effects.

Continuous development and improvement of technology in the world determines the appearance of new and new construction, insulation, acoustic and decorative materials that are more widely used in the construction of manufacturing and service buildings.

Ministry of Health exercises **state sanitary control** through its specialized authorities. The preliminary health checks involving medical professionals - doctors, sanitary engineers and architects who give opinions on all relevant hygiene standards, building codes and regulations for prepared projects for construction, reconstruction and modernization of production buildings. Opinions shall be submitted to the parts. Preliminary health checks are carried out and in the construction phase of the project and ends with an opinion authorizing the use of the building in operation, which is provided by PIPCPH state acceptance committees and permission for using buildings in the Republic of Bulgaria.

15.2. VENTILATION

Aeration is a reliable and economical way of ventilation of industrial buildings. Used in buildings with higher heat load, because under these conditions it is reliable in all seasons. Used elevating power of heated air, which tends to the above aeration holes. For well organized aeration is required greater height of the building - exceeding 5 m and aeration holes are located on strictly defined places. The most commonly used two-way aeration when the air enters on both sides of the housing through the lower aeration openings (windows with a particular manner of opening and exits through the upper openings (fonari). Wide area buildings are inconvenient to use on aeration because in their inner part can not do enough fresh air.

Multiplicity of air changes in workplaces is determined according pollution and dimensions of the rooms.

Types of systems and ventilation:

- **Natural** - horizontal (windows and doors). It is more active as greater pressure difference and in temperature between two opposite walls. In all cases, natural ventilation is unstable and depends on the weather.
- **Vertical** - through the upper windows, using the elevating power of heated air.
- **Mechanical** - air supply and suction - two ventilation working together.
- **Air conditioning** - looking not only provide fresh air in the room, but maintaining microclimate parameters within certain limits - temperature and humidity in the room (these parameters vary according to the type of work). Certain procedures require dry air (installation of electrical apparatus), and another case moist (spinning, weaving).

Dust in the workplace strongly affects the quality of the environment. To avoid it is necessary to prevent facilities that emit dust, no-work without local exhaust ventilation. Regularly clean working environment and to prevent secondary dust in the air. This can only be achieved with well-organized maintenance and good condition of the machines and ventilation equipment, maintained by staff.

1. **Aspiration**. It is done using a local exhauster connected to the suction duct (under vacuum). We have to take into account the fact that the speed of suction rapidly decreases with distance from the hole. For example, a circular suction opening - off one diameter - speed drops 10 times. Where the place of separation of dust is mobile (eg mine where dust activity moves continuously), mounted mobile suckers associated with the intake air duct with flexible connection. If dust emission is at the level of the worktable it is appropriate to provide lower local suction (dust tends to settle due to gravity).

2. **Transportation**. Dust extraction in the workplace is transported by pipeline to the dust-catcher. There are two basic ways to avoid jam on pipelines transporting dust mixtures: while maintaining high-speed transport and while not allowing sharp corners, that support the layering of dust and jam.

3. **Dust-catcher**. Dust catchers are different types:

• ***Mechanical*** - gravity: ordinary settling chamber where the section is significantly increased and the speed of dust mixtures suddenly falls and the dust settles by gravity. Adverse is that these dust catchers are very large and inconvenient to use in most cases. To reduce their length are used mixed precipitators where dust mixtures moves in a zigzag and thus prolongs the path of the particles to can settle. Precipitated mainly larger particles.

• ***Filtering***. Filtering it through a filter fabric - cotton, paper or synthetic nonwovens. To avoid the accumulation (stratification) of dust on the filter

material, leading to a strong increase in resistance, applied the following means: increases strongly the surface of the filter material by making multiple folds or multiple sleeves that are shaken by periodic strikes on filters to unstick the accumulated dust. Perform and increasing the porosity of the material used by various artificial materials that are electrified (static electricity attracts dust particles).

• ***Centrifugal*** - used the high speed of suction fan, the spray tangents to the inner side of the cylinder which in its lower part becomes an inverted cone.

• ***Cyclone***. A cylinder mounted on an inverted cone. Dust mixtures enters in the upper part of the cylinder tangential with high-speed, rotated and its movement becomes helically, along the way dust particles lose their speed, by friction in the walls of the cone. They precipitated at the bottom of the inverted cone (there are discharged through a valve in the container for dust), and the cleans air changes its direction and is directed upward leaving the cyclone at the top. It is possible to increase the efficiency of these dust-catchers by reducing their diameter, while in same time not increasing its resistance. This is achieved by using a greater number of cyclones with smaller diameters, called multicyclones.

• ***Water dust-catchers.*** Dust mixtures are subjected to the action of water - or in the form of a liquid or in the form of a condensable vapor, such as resulting in wetting of the powder and create larger particles that are separated in the inner part of the apparatus in the form of a middling slime (silt).

• ***Ultrasound***. The issue of continuous ultrasonic waves from a single source support consolidation of particulates and they can significantly easier to capture the classic dust-catchers, such as dry cyclones or even settling chambers with partitions.

• ***Electric or electrostatic filters*** used for the first time of Cottrell in the US. The principle of dust collection in them is the passage of dust mixtures at moderate speed through a bundle of vertical pipes with a diameter of 20 to 30 cm and about 5 m long, through which passes a metal rod with a diameter of 2 to 3 mm, acting as a negative electrode. Pipes are a positive electrode and at a differential tension of 25 to 100,000 V., particulates are first ionized and then precipitate and adhere to the surface. In another type electrostatic, called wet, dust mixtures is suitably wetted at the inlet and after the sticking on the wall of the filter, it was washed with running water. This principle is very effective and it gets cleaning from the order of 99.9%, but is too expensive.

In **gas polluted** working environment aspiration is used in the same way to capture, as in the dust. As in dust the local aspiration combined with general exchange (of the whole room) ventilation. The exact design and implementation (for maximum efficiency) of ventilation, into account the conditions and characteristics of the technological process: the type, quantity and physico-chemical characteristics of the emitted gases and vapors, heat generation, etc.

What precautions should be taken before bring out the polluted air in the atmosphere? Incorrect usually sucked from the workplace or technological facility polluted air to throw directly out into the atmosphere without purification. Where are bring out into the atmosphere many harmful substances, such as hydrofluoric acid, sulfur dioxide, etc., should be undertake preliminary purification before throw out. This can be done for example by bubbling in water or a neutralizing solution. Since these purification of exhaust air are expensive, require consolidation of a group of gases in general or purifier such capture in which to use caught substances again in production. Therefore, in most of these cases the use of charcoal filter, in which obtained absorption of gases, and then they are recuperated by separation with steam.

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16 PERSONAL PROTECTIVE EQUIPMENT

Personal protective equipment (PPE) is used when risks can not be avoided or reduced sufficiently by means of collective protection or by means of methods and procedures for organizing the work.

The general requirements for PPE are:

- to provide protection against the risks in which they are applied, without themselves leading to an increase in any risk;
- be eligible at the appropriate workplace;
- comply with the ergonomic requirements and health of workers;
- match the user's dimensions, if necessary, after appropriate adjustment;
- where, due to more than one hazard, it is necessary to use more than one personal protective equipment, they must be compatible and continue to be effective against their respective hazards when used together.

PPE are divided into:

1. PPE for head protection.

This PPE group includes the following subgroups: helmets; hats; bonnets or under helmets; head cloth and kerchiefs.

These devices are designed to protect the head (without the eyes, ears and respiratory organs) from mechanical impacts - shock, contact with open electrical wires, unfavourable climatic effects - cold, rain and wind below +12°C, pollution with no-toxic and toxic powders, to protect hair from moving machines and mechanisms, solar radiation and other harmful radiation and hazardous thermal effects.

Of the above-mentioned PPE group, the helmets are the most important for maintaining health and safety for workers. They can prevent serious accidents at work (having a lethal finis) caused by the impact of falling objects and materials.

Classical in this regard is the INTRCAP MK 1A (weighing 320 g), which also protects against electric current with a voltage above 24V. **(Figure 1.1.)**

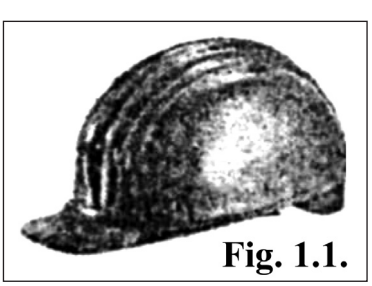

Fig. 1.1.

For special purposes there are other different helmets. To protect the head and face during the operation of fire departments, the helmet **(Figure 1.2)** is very good, protecting against: mechanical and thermal impacts; water and precipitation; aggressive substances; organic solvents, paints, varnishes, adhesives; oil, lubricants and fats.



Fig. 1.2.

2. PPE to protect hearing organs.

These PPE are divided into two sub-groups: internal antiphoning; external earphones and helmophones.

They are designed to protect against effects of noise exceeding 85dB (A).



Fig. 2.1.

The internal antiphonics are most often made of a soft, sound absorbing material that can be gradually expanded to take the form and size of the hearing channel **(Figure 2.1)**.

Internal antiphonics can reduce the noise to the order of 30-34 dB for the most dangerous, High noise rates. Some anti-phonics are also anti-allergic and are comfortable for people with narrow ear canals.

The external anti-phonics can: be fitted with helmets **(Figure 2.2)**; to receive and transmit speech with built-in microphones and headphones; to reduce noise with certain characteristics. External antiphonics are used at a noise intensity above 102-105 dB/A, and the helmphons at an intensity above 112-120 dB/A.

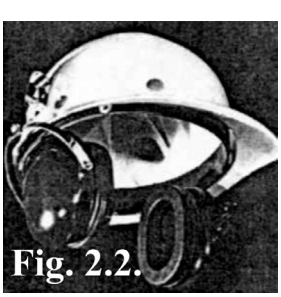

Fig. 2.2.

Antiphonics can not be used in cases where sound signals have to be received to ensure safe

work. In this case, you must use anti-phonics with a device for receiving the sound signals.

3. PPE for eye and face protection

Various types of spectacles and shields are used for this PPE group.

They are designed to protect against mechanical damage - blowing away particles, harmful radiation, spraying with aggressive toxic and hot liquids and irritating eyes powder. The main requirements for this PPE group are not to reduce the viewer's field of view, to avoid sweat, not to fall when performing different movements, to be able to combine with other PPE and in particular, to be able to use glasses , correcting vision.

In order to guarantee the protection qualities of glasses and shields, it is particularly important to have the materials from which the glass is made - the various types of light-filtering lenses and viewing screens that are resistant to blow, splashing with aggressive and hot liquids.



Fig. 3.1.

Such wide-field eyeglasses **(Figure 3.1)** protect against: a strike of objects and particles; non-toxic dust; splashing water; toxic solids and dust; toxic liquid substances and solutions, respectively splashes of acids, bases, solvents, paints, varnishes, adhesives, oil, petroleum products and mineral oils; toxic aerosols and gases - class 1-4.

The front protective helmet **(Figure 3.2)** protects against: sparks; splashes of molten metal, slag and broken particles; ultraviolet radiation; light rays with intense shine - over 200 nits.

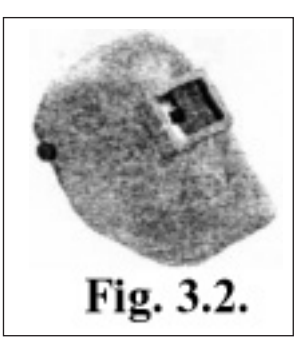

Fig. 3.2.

4. PPE for respiratory protection



Fig. 4.1.

These PPEs are of particular importance for the protection of the health and safety of workers. This PPE group consists of the following subgroups: mask whole; semi mask **(Figure 4.1)**; respiratory insulating apparatus **(Figure 4.2)**;breathing apparatus insulating with pure atmospheres; breathing apparatus insulating with compressed air supply; breathing apparatus isolating with an open system; breathing apparatus isolating with a closed system; space-suit and pneumo-suit **(Figure 4.3)**; filters designed to clean the air by retaining toxic substances.

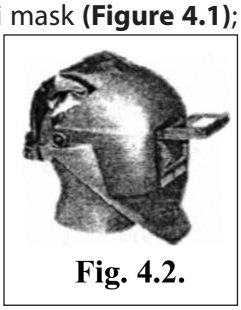

Fig. 4.2.

Particularly essential requirements for this type of PPE are: not to impede breathing, to diminish the field of view, not to smash the visual screen, to resist of impacts and aggressive substances. The body of the mask does not irritate the skin of the face, stick tightly and has good airproofness.



Fig. 4.3.

5. PPE to protect the upper limbs

This PPE group is designed to protect the hands, including the elbow joints from mechanical impacts, from dangerous chemical substances, from thermal and adverse climatic influences and from electrical currents.

Predominant in this group are protective gloves, which have a large assortment depending on their purpose, construction and the materials and technologies used.

Besides gloves for this group, sleeves and prophylactic wrist straps are included.

To ensure work safety, dielectric gloves, heat-resistant gloves and gloves protecting against aggressive and toxic substances are particularly important. For example, nitrile gloves "Soflex" protect against: blow away from objects and particles; piercing, cutting or scraping action; contaminated surfaces with non-toxic materials and contaminated surfaces with oil, petroleum products and mineral oils; non-mineral oils and fats.

Gloves for workers in slaughterhouses, sausages and butchers are also special **(Figure 5.1)**.



Fig. 5.2.

Protective leather cuffs protect against: piercing, cut or scratching; sparks, splashes of molten metal, etc. **(Figure 5.2)**.



Fig. 5.1.

6. PPE to protect the lower limbs

They are used to protect legs to the knee joint. Include the following subgroups: boots; half-shoes; sandals, slippers and prophylactic shoes; boots and galoshes; dielectric boots and galoshes.

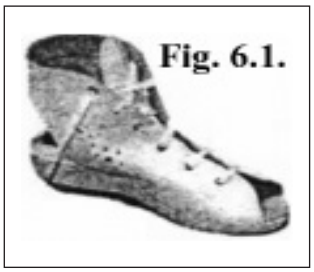
Fig. 6.1.

Personal lower leg guards are designed to protect against mechanical, thermal, climatic, strong aggressive and toxic substances, vibrations and harmful radiation. In most cases, personal lower leg protection should protect against the combined effects of several hazards and dangers. Very often used with slip-resistant soles.

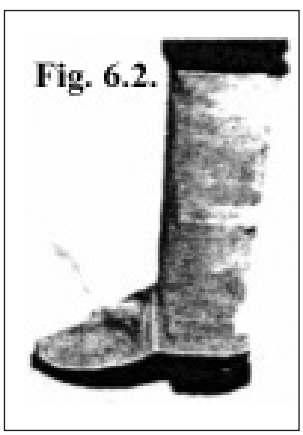
Fig. 6.2.

In a standing position, shoes with low or high leg are recommended **(Fig. 6.1)**. They also prevent slipping as well as convection heat from + 25 °C to + 37 °C.

To protect the legs from intense mechanical impacts, shoes with steel plates in the bombs can be used to withstand up to two tons of pressure. The shoes are lightweight and comfortable.

Boots protect against: slipping; contaminated surfaces with non-toxic substances; watering and rainfall; water splashes and wet surfaces; splashes and wet surfaces with acids and/or bases.

To protect against: piercing or pricking effects; sparks, splashes of molten metal, slag and hot particles are very practical in the case of leggings **(Fig. 6.2.)** or leather gussets.

Knee-pads is used for creep work (pavers, parquet floors, etc.). Against skidding in snowy and icy terrain, very useful work is done by special shoe cats. Their steel teeth have a height of 10 mm.

7. Special workwear and PPE for body and torso protection

This group of protective equipment has a wide range of applications. In most cases, special work wear is designed to protect against the simultaneous impact of several hazards and dangers.

The following subgroups are included in this group: fur coats and half-fur coats; costumes; jackets, jerkins and bodies; trousers, cami-knicks and half-aprons; shirts; underwear; cloaks, mantles and mackintoshes.

Subgroups are based on type of constructions. When combining different constructions and materials, special working garments with different protective qualities are made. To prevent of low air temperature (+12° to –20°C), static electricity and water splashes, wet surfaces are most often used with half-fur coats.

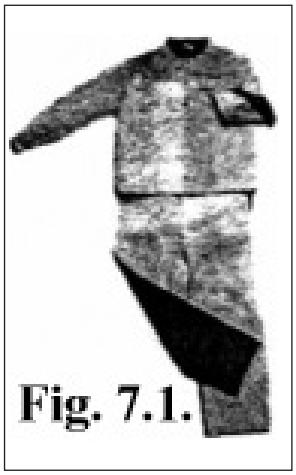
Fig. 7.1.

For welding works to protect against sparks and splashes of heated particles, welders' suits **(Fig. 7.1)** are recommended, which have a leather front, sleeves, back panels, front of the trousers and the saddle. When working near a fire, used costumes made of aluminized material impregnated with cotton cloth. For example, the suit "Inter safe fire suit" **(Fig. 7.2)** suit has a very good performance: it protects the body and face from sparks, molten metal splashes, slag, heated particles; thermal radiation up to 15x1000 W/m^2; heated surface above 100 ° C.


Fig. 7.2.

To protect against: toxic and aggressive substances, organic solvents and mineral oils, suits are recommended, some of which are completely air-proof.

At risk of splashes and dirty surfaces of acids, bases, solvents, paints, varnishes and adhesives, as well as from water and from contact with contaminated surfaces of non-toxic substances, use seamless, one-piece aprons with sewn ties for binding on the waist and for hanging on the neck.

When working in an area of severe radioactive contamination, workers must wear knitted underwear, socks, gloves and slippers under the other special workwear. All items of knitwear are cleaned (deactivated) after each use. They can not be used outside the work area.

8. Protective safeguarded equipment

These safeguarded equipment is designed to protect workers from potentially dangerous risk. This group includes the following protective means: dielectric carpets; anti-vibration mats; safety belts against falling from a height; cats for climbing; working seats in a hanging position; dielectric rods; protective support strips for attaching or lifting loads.

Due to the large variety of these safety devices, and


Fig. 8.1.

especially the great responsibility they incur, they must be provided with the necessary instructions for use, pre-training and training of the workers with them, quality certificates, shelf-life for preserving and operation, methodology for verification of safety features during operation.

For example, there are special requirements for the belts to fall from a height. The support in the event of a fall should be in the seat area (to protect the kidneys from tearing). In addition, a special braking device is also provided which absorbs part of the energy **(Figure 8.1)**.

9. PPE for skin protection - dermatological protective equipment

This group of protective devices are designed to protect against skin damage by chemical agents and harmful radiation. Dermatological protective agents are subdivided into the following subgroups: barrier creams; protective preparations; restorative preparations; disinfectants.

The right choice and use of protective creams, pastes and biological gloves leads to a reduction of professional dermatoses and limits the penetration of toxic substances into the body.

Depending on their solubility and character, these PPEs are divided into:

a) oil-no soluble (water-soluble) - they serve to protect the skin against mineral oils, fats, hydrocarbons and organic substances (solvents, varnishes, resins, etc.) free of water. Washing agents include water and soapy water emulsions. Marked with O/W:

b) water-insoluble (soluble in organic solvents) - serve to protect the skin against water and aqueous solutions of acids, bases, salts and other substances, as well as water-oil emulsions. Washing agents are organic solvents with subsequent skin lubrication with softening and lubricating creams. Marked with W/O;

c) silicone based protective substances. Silicones are organic substances resistant to acids, alkalis, water and high temperature. They are easily attacked by organic solvents (trichlorethylene, carbon tetrachloride) and petroleum derivatives (petrol). They have good coverage - they form a monomolecular film. Washing agents are active - binding substances and highly alkaline aqueous solutions. They are not recommended for use in the furniture industry, paint coatings and galvanotechnics as they can damage the quality of the product.

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17 MANUFACTURING AND PROFESSIONS - BASIC TECHNOLOGIES, HEALTH RISK, PREVENTION

D.Tsonevsky

17.1 AGRICULTURE

Agriculture is one of the main branches of the economy. The object of its production is the vegetable and animal raw materials needed mainly for the functioning of the food industry but also of many other branches of the economy.

To improve and optimize working conditions in modern agriculture, it is essential to meet the basic requirements of hygiene, physiology of labour and ergonomics. The introduction of new technologies and new equipment, the mechanization and automation of the basic labour-intensive processes, the optimization of the relations in the ergonomic system - man/machine/environment, in the agricultural production led to the creation of new professions - operators, mechanics, etc.

Major branches of agriculture are plant and livestock breeding. The broad range of agriculture includes sub-sectors such as grain production (grain and technical crops), vegetable growing, fruit growing, viticulture, etc. Livestock breeding includes sub-breeding cattle, sheep, pig, poultry farming. Each industry is characterized by general and specific labour-hygiene problems.

The general characteristics of all agricultural activities in the different sub-sectors of agriculture, distinguishing them from the working processes in the industry, are: 1. Higher physical workload and intensity of labour. The problem of the complete replacement of manual labour in key sectors of agriculture has not yet been resolved, operations connected with ploughing, sowing, harvesting of greenery crops are mechanized, other activities such as forage, special operations in viticulture, vegetable production, tobacco production are still not mechanized . In livestock farming, much of the labour is still heavily physically; 2. Particularities in the rhythm of work and the duration of the working day. Agricultural labour is characterized by irritability, both in terms of annual seasons and in the twenty-four-hour rhythm of some major activities. An intense load, for example, occurs during ploughing, sowing, harvesting, and in livestock - during the period of shearing, the sheep's birthing period. The duration of the working week and the use of the rest day during a harvest campaign is determined only by the weather conditions. The working day at the mechanic most often lasts for 11-15 hours, and in livestock breeding work begins at 3-4 in the morning and ends with small interruptions at 21-22 hours; 3. Frequent changes of operations - irrespective of the internal specialization in each sub-sector of agriculture, individual operations often change; 4. Agricultural activity is most often done outdoors under the influence of day and seasons-specific weather factors; 5. Biological factors. Direct contact with domestic animals, plants, insects poses a high health risk to professional, infectious, parasitic and allergic diseases among all categories of farm workers; 6. Chemical factors. The widespread use of chemical preparations such as pesticides, fertilizers, biostimulators, etc., creates conditions for acute, sub-chronic and chronic intoxications, for allergic diseases, to reduce the reactivity of the organism; 7. Factors related to labour mechanization. Here is added the adverse effects of factors such as noise, vibration, air pollution with harmful gases, contact with fuel-lubricants, overheating, non-ergonomic-work place and posture; 8. Other factors and peculiarities. The considerable remoteness of the workplace from the place of residence is adversely affected. Also, the aging of agricultural workers as well as female labour in unskilled labour operations associated with heavy physical labour is also significant.

17.1.1. LABOUR HYGIENE IN PLANT-GROWING

Labour hygiene in mechanics. The work of the mechanics - tractors, combine harvesters, mechan-

ics, agricultural machinery and tractors mechanics, is very diverse. It depends on the different stages of agricultural production.

The main machine in agricultural work is the tractor, which, combined with various attachments, is involved in a variety of operations in soil cultivation, sowing, cultivation of agricultural crops. The construction features, the differences in the type of fuel, the type of the chassis determine to the greatest extent the labour-hygienic conditions of work.

During operation, the tractor is subjected to a continuous impact of a set of unfavorable factors. Microclimatic conditions of operation depend on the presence or absence of a cabin, its constructive solution. The microclimatic conditions in the cabs of the machines used are significantly different from those of the atmospheric air. In the tests of different brands of tractors during summer, the temperature of the internal surfaces of the walls between 36 and 55 ° C and the air in it between 28 and 44 ° C, which exceeds the outside temperature by 5-14 ° C.

What matters for noise is the type of engines and the mode the cabs are mounted. Diesel engines generate higher-intensity noise than gasoline. The noise tests of the agricultural machines used in Bulgaria show that it is high frequency, with an intensity of 75 to 105 dB / A and hygienic norms exceeding 4-14 dB/A for different tractors, 6-12 dB/A in mowers, and with 5-10 dB/A in harvesters.

The vibrations of working with agricultural machines are mostly general - aperiodical with pushing nature. Meanwhile, periodic, local vibrations of constant character are also important in the operation of engines and gears. The vibrations measured on the seats of used tractors in the different activities exceed the hygienic norms for the mean frequencies from 2 to 21 times and for the high ones - up to 1.5 times.

The combined effect of noise, vibrations and their combination with forced posture leads to increased morbidity of the locomotory system, to angiospastic injuries, hypertension, angina pectoris, gastric motor disorder, vegetative dystonia, hearing and vestibular disorder.

Dust is one of the most significant unfavorable factors in the working environment of mechanics. Air pollution in the respiratory area depends on the nature of the work performed, the weather conditions and the soil humidity, the design of the machinery, the height and position of the seat, the sealing of the cabins, etc. The powder with which mechanizers are exposed is non-toxic, with an organic-mineral character and is finely dispersed. Mechanics complaints are particularly frequent during harvest when the air dust in the work area is highest.

The mechanics are exposed to the harmful effects of toxic chemical agents - pesticides, exhaust gases from engines (carbon monoxide and dioxide, nitrogen oxides, sulfur oxides, methane, aldehydes, 3-4 benzpyren, ammonia, hydrocarbons, mineral fertilizers, fuel lubricants The formation of toxic gases is related to the type of agricultural machines, the type of engines - petrol or diesel, the term of operation, their technical and operational characteristics, etc.

To limit the impact of the main harmful factors in the mechanics-noise, vibration, dustiness, adverse microclimate, etc., suitable solutions are made in the construction and operation of agricultural machines - provision of cabin with conditioning devices, sound insulation, vibration reduction measures. The ergonomic position of the seat, levers and other devices In the cabs also have their influence, and the organization of the working day, ensuring a correct working and rest regime, healthy eating are of great importance.

Labour hygiene in the field of grain-production. The work of the grain-producers is reduced to carrying out operations such as fertilizing the soil with natural and artificial fertilizers, loading and unloading, soil mowing, weeding, irrigation, grain decontamination, etc. Nowadays almost complete mechanization is carried out in the different types of soil treatment (plowing, bending, harrowing), sowing, fertilizing, irrigation, crop cultivation with pesticides, harvesting of grain crops. Lesser opportunities exist in some sub-sectors such as viticulture, fruit growing, vegetable growing, and the cultivation of certain technical crops.

In spite of mechanization in the field of grain-production, the problem of intensive physical work is still a problem, especially in manual operations. Their energy costs can be categorized as medium and heavy-duty occupations. In agriculture too often are the forced posture - inclined body, squatting, kneeling, etc. The physiological performance of the work indicates that it is carried out with some strain on the cardiovascular and nervous system and on the locomotory system. Grain farmers, especially in the summer, lose a lot of fluids, have a fast heartbeat.

During the work, the farmers are exposed to the constant influence of changing meteorological factors - outdoor work. Even within one season, the temperature often varies considerably, allowing for overheating or over cooling.

Pollution of air in various field works also fluctuates considerably. The main source of dust is the soil in its mechanized treatment, as its humidity has a major impact. The character of the operations is often associated with permanent irritation of the skin. During weeding, irrigation and other manip-

ulations skin microinjury, macerations, pyoderms, dermatitis can be produced. Failure to comply with hygiene requirements, lack of prior instruction, and handling of faulty machines and tools often lead to injuries. There is also a danger of biting from reptiles and insects, and from some transmissible infections (Crimea hemorrhagic fever, Haemorrhagic nephrosonephritis, Lyme Illness, etc.).

Farming activities are often also associated with a health risk from the use of pesticides. Failure to meet hygiene requirements during seed decontamination, spraying and dusting crops, failure to observe quarantine deadlines causes acute, sub-chronic and chronic intoxication.

Several specific features are also found in some sub-branches in the field of agriculture. In a study the parameters of the microclimate in the **greenhouse vegetable production** in Bulgaria show that during the hot season the air temperature varies from 25,6 to 38,5 ° C, the relative humidity - from 74 to 100% and the air velocity - from 0, 0 to 0.60 m/s, and during the cold period, respectively, of 14 to 27 ° C; from 61 to 96% and from 0.5 to 1 m/s. This shows that the microclimate by temperature factor during the cold period of the year is within the permissible norms, and in the warmer it is overheating, within the limits of the unfavorable and especially unfavorable conditions. The adverse effects of the discomfort microclimate are exacerbated by the significant physical load, the heavy load carrying (10 to 22 kg of single load), the high labour intensity and the forced posture.

Of the toxic-chemical factors, pesticides are also important here, using widely and in large quantities. It has been found that about 30-35 pesticides and several pesticide mixtures of PhOC, ChlOC, carbamates and dithiocarbamates are found to be used in greenhouses for vegetable crops. Significant is the high relative part (55-65%) of preparations with medium and high toxicity and their pronounced skin-absorption and irritation effect.

Fruit growing and viticulture are important sub-sectors in agriculture. Work here is also predominantly seasonal, outdoors, and the microclimate, as well as other factors in the work environment, are similar to those in the field of grain-production. The processes in fruit and vine-growing have specific mechanization - small tractors, machine spraying, etc. And are characterized by uneven rhythm and many manual operations. This leads to increased upper respiratory tract morbidity, to frequent conjunctivitis, dermatitis, allergic reactions. There is more frequent pyoderma and microtraumatism here.

Tobacco production also has a number of specific factors in the working environment. Tobacco growing is a complex process involving about 53 operations. The main stages are soil preparation, seed planting, seedling cultivation in greenhouse, plant planting, cultivation, harvesting, tobacco racking, drying. Along with meteorological and microclimatic unfavorable factors, seasonal workload, unfavorable working position, there are some specific dangers to tobacco drying. In the air of the dryer, concentrations of nicotine vapours of 0.94 to 8.1 mg/m^3, methyl alcohol - from 7.1 to 30 mg/m^3, aetheric oils - from 0.3 to 3 mg/m^3, ammonia - 10 mg/m^3, as well as traces of phenol, organic acids, CO and others.

The complex effects of working conditions here lead to diseases of hypertension, gastritis, cholecystitis, bronchitis, radiculitis, rheumatic polyarthritis, female genital diseases.

17.1.2. LABOUR HYGIENE IN LIVESTOCK BREEDING

In the different sub-sectors of animal husbandry, common unfavorable factors are: dustiness, air pollution with harmful gases, unfavorable microclimate, heavy physical labour.

The differences between the different livestock breeding sectors - sheep, cattle breeding, pig farming, fish farming, etc. are distinguished from the biological characteristics of the different livestock and the aims of their cultivation - meat production, milk production, etc.

The microclimate in livestock farms depends on the type, construction, and extent to which they meet modern requirements. A microclimate, mainly dependent on the weather conditions and the season, is established in the farms. Conditions for overcooling or overheating are formed therein.

In livestock farms, constantly acting are the harmful factors as of ammonia, CO_2, SH_2, mercaptans, aldehydes, ketones, and others, eliminated by animals organism. In the aeronautical environment of farms of an earlier type, for example, the concentration of ammonia has been found to reach 2.4-80 mg/m^3, carbon dioxide - 0.20.4%, hydrogen sulphide - from 8 to 30 mg/m^3 - significant exceedance of the MAC for these gases.

Major occupational hazards in livestock breeding are also some biological factors. The possibility of transmission of a number of zooanthropogenes such as brucellosis, anthrax, foot-and-mouth disease is high.

Dust is a significant adverse factor in the working environment. The degree of dustiness varies depending on the season, humidity, the nature of the food mix, etc. The powder is predominantly organic

in nature and can serve as a factor in the transmission of many causative agents of infectious and allergic diseases and mycoses.

Hygiene measures in livestock breeding should be directed to maximum mechanization and automation of the working processes, daily cleaning of the premises, daily provision of the breeders and carrying out periodic prophylactic examinations, good lighting and good natural and artificial ventilation of the premises, regular and effective veterinary control of the animals and organizing healthy eating for workers.

Illness and traumatism in agricultural workers. For the non-mechanized and partly mechanized agricultural labour, the diseases of the locomotor apparatus are characteristic. The spine, knees, shoulder joints are most commonly affected. The second place is chronic bronchitis and emphysema, followed by cardiovascular diseases, often with damage to venous vessels (varicose veins). Substantial place in the structure of morbidity also includes gynecological diseases related to the widely used female labour.

For mechanics, the structure of the morbidity is different. Diseases of the digestive system are the first place, followed by production traumatism, colds, diseases of the vegetative and peripheral nervous system. Among the breeders, first of all, is production traumatism, followed by colds, pulmonary chronic diseases, diseases of the locomotory system and various infectious diseases related to contact with animals.

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N. Stamova

17.2 MINING

17.2.1. MINING

Mining is carried out in two ways - open and underground.

The open method of extraction of ore and non-metalliferous minerals from an economic and hygienic point of view is more appropriate and progressive, but not in all cases it can be applied in practice. The open mines (quarries) in the mining industry generally represent stepped valleys reaching a depth of 100 to 400-700 m. Technological equipment is located outdoors on horizontal platforms.

The open-cast mining process consists of the following main operations: 1) drilling and blasting operations; 2) excavation and loading of the rock breed and ore in transport vehicles; 3) transportation of the ore to the enrichment plant, and the rock breed to the discharge; 4) road construction and road maintenance activities; 5) repairs.

The most common method for drilling-blasting operations in open-cast mining is drilling. Boreholes are filled with explosives, most often of the ammonite type. Filling with explosives is done manually or with special machines.

The excavation and loading of rock and ore is done by excavators or "mechanical shovel" excavators.

The transportation of the ore to the filling factories and to the rock mass to the discharge is most often done by dumpers with a lifting capacity of up to 200 t. The application of road transport has the advantage of avoiding heavy and labour intensive operations for the construction of a railway lines, but on the other hand the gases emitted by cars contain toxic substances.

In the underground mining process different systems for the ore deposit are applied. The system chosen must meet the mining and technical conditions and provide the most favourable and safe working conditions.

The underground mining process consists of the following production operations: 1) boring of blast holes or drilling; 2) filling them with explosives and blasting; 3) strengthen of mine workings[1]; 4) loading; 5) scraping and transporting weighed ore and rock mass; 6) auxiliary and repairs.

[1] Mine workings is each "empty" area worked in underground mines, generated after taking away weighed ore or rock mass.

Drilling of blast holes is most often done with vibrating tools - drill bits that work with compressed air. The most commonly used so-called rapid drill hammers (2700-3700 beats/min) of different weights: hand weights of about 20 kg and telescopes weighing 50 to 75 kg. Drilling, including self-propelled drilling machines is also applied, where mining conditions allow.

Explosives such as ammonites and dynamites, and detonators are used for the explosion. Explosives are placed in cylindrical cartridges. The correct choice of explosives determines to a great extent the safe operation and the amount of explosive gases generated by the explosion. The strengthen of mining work is necessary due to the presence of continuous rock pressure. The strengthening is made with wooden or metal frames, reinforced concrete and others.

The loading of weighed rock or ore is done manually and/or with pneumatic loading machines.

The transportation of the weighed rock and ore mass on the horizontal mining works is done by rail with electrowelds, and by the vertical shafts - by means of cells loaded with full/empty wagons.

Auxiliary activities provide ventilation ducts, plumbing, railways, equipment repairs, etc.

For the **conditions of the working environment in ore mining** is a characteristic complex of work place factors: specific microclimate, noise and vibrations, dust, toxic gases, absence of solar radiation in the underground mines, etc.

The microclimatic conditions in the opencast mines depend on the climate and the time of the geographic area where the mine is located. In the summer, continental climate regions can create adverse microclimatic conditions due to the combination of high air temperatures and intense insu lation with secondary reflection. Moreover, with the dehumidification of the mine the temperature rises every 100 m depth by 1-1,5 ° C and above, due to adiabatic heating, insulating and raising the temperature of the ore breeds. In evening, night and morning hours as a result of inversions, the air temperature in the lower places of the open pit is lower by 1-2 ° C than on the surface. Inverse air streams lead to the formation of fogs.

The air movement in the deep opencast mines becomes a complex aerodynamic scheme with low

ventilation efficiency and a sharp reduction in air velocity.

The relative humidity in bottom places of the open-cast mines compared to the open surface varies slightly, with its gradient in the diurnal range of 5-10%.

In the winter and transition seasons of the year the meteorological conditions in the open pit are characterized by low temperatures, often combined with strong wind and rainfall.

When assessing the microclimate in opencast mining, it must be borne in mind that some professional groups, such as sonders, bombers, roadmakers, repairers, are all throughout the shift or most of it outdoors. Another part - excavators, bulldozers, diggers and dumper drivers are in cabins and protected from external weather conditions, but if the cabins are not appropriately upgraded (without efficient thermal insulation), drivers is cooled to even freezing temperatures in winter, and summer - heating the air to temperatures 5 to 15°C higher than outside. The operation under the described microclimatic conditions is associated with a pronounced overtension of thermoregulation, abundant water loss and possible acute overheating of the body.

In underground mines, microclimatic conditions are mainly determined by the depth of mine workings and the efficiency of ventilation. In the main mining works (crossbeams, shroud, mining yard, galleries), there is considerable fluctuation in the air temperature, and in the stopes - it is relatively stable. In the vast majority of the Bulgarian underground mines the temperature in the main works is from 10 to 15°C, and in props, chambers and stopes - 20-25°C; In deep mines (over 500 m depth) can reach 30-34°C. The speed of air movement is also different - it can reach up to 5-6 m/s^{-1} and more in main mining works, and in deaf stopes - 0.1-0.3 m/s^{-1}. Relative humidity is very high - 85-95%, which depends primarily on the water content of the rock breed. It can be summed up that an unpleasant, uncomfortable microclimate is formed in the underground mining. The worker's consistent exposure to microclimatic conditions with the opposite effect on thermoregulation is important - in the stopes of overheating - and, in the event of a departure from them - significant cooling. Specialised sanitation standards for mine microclimate do not allow operation at temperatures above 32 ° C.

Noise and vibrations in mining are generated by the operation of machinery and equipment, especially for those using compressed air.

In the **open-cast mines**, sources of noise and vibrations are the different types and brands of drills, diggers, bulldozers, excavators, dumpers and others. The most characteristic features of the noise-vibration exposure in these cases are the following: transmission of vibrations to the worker through the floor, seat, levers and pedals; a combination of common with local vibrations; frequency variability of vibrations coupled with thrusts and in combination with noise. The exact characteristics of noise and vibration depend most on the type of machine, the operating conditions, the degree of wear of the mechanisms, the hardness of the breed, the design of the cabin, etc. (The modern models of these machines are designed to meet the requirements of international standards for noise and anti-vibration protection - noise and vibration levels usually do not exceed the permissible values). When operating machines in soft breeds, 4 to 16 times lower vibration intensities are measured on the control levers, 2 to 5 times on the seats, and 2 to 8 times on the floor.

In the case of heavy-duty dumpers, bulldozers and auto-graders, the level of general vibrations also depends greatly on the state of the roads, their speed of movement and their load. Vertical vibrations are most intense, with maximum values ranging from 1-4 Hz; when moving with a load the vibrations are weaker.

In the case of **underground mining**, the main sources of noise and vibrations are pneumatic hammers, drills, loaders, scrapers, etc. Highest noise levels are measured for drilling activities **(Table 1)**. The professional risk here is also conditioned by the high frequency nature of noise and exposure over most of the working time.

The local vibrations generated by the different types of drills typically exceed the hygienic norms - from 8 to 14 times for the average frequencies and

Process / Machine	Level to noise in dB/A	
	By S. Maksimov	By Altynbekov and all.
1. Pneumatic hammers	105-127	114-127
2. Drills	75-102	
3. Loaders – loading of ore – feeder ore	88-106 92-104 83-88	95-110 95-110 –
4. Scrapers	93-98	94-97
5. Electric carrier – in purifying wagons	85-87 –	– 108-110
6. Fans	92-105	70-75
7. Lifts	89-92	68-97

Tab. 1. Noise levels in different mining processes and machines

from 2.5 to 5 times for the highs. The contact time of the master-digger or his assistant with the vibrating punch takes about 25% of the working time. The nature and intensity of the vibrations depend on the pressure of compressed air in the net, the stiffness of the breed, the technical condition of the instrument. When assessing the occupational risk for the occurrence of a vibration disease, the potentiating effect of the static effort of the upper limbs (to maintain the instrument and the posture) as well as their cooling from the water used for the water drilling, must be taken into account.

Dust, the most widespread and dangerous production hazard in mines, is released in all manufacturing operations.

In the **opencast mines** the most intense dust source is blasting. As the blasting is carried out once a day (2-3 times a week) and during interruptions between shifts, until the start of the new work shift the dust dissipates and precipitates and at the start of work the dust corresponds to the average dust of the mine - 0.3 to 2 mg/m^3. When updepthing the mine, reduced natural ventilation helps to increase dustiness. Usually, in summer, dust is higher than in winter - then the ways in the mine are a serious source of dust.

Local dust contamination in digger cabins, bulldozers, excavators and auto-mowers is also dependent on freight, mining roads, wind, etc. **(Figure 1)**.

In the case of **underground mining**, drilling of explosive holes is the largest share of dust mining. When drilling 1 m of rock or ore mass, an average of about 150 g of finely dispersed dust is formed which is capable of causing dust in the stope up to 1600 mg/m^3. Before the introduction of the wet drilling, the average dust in the ore mining in Bulgaria was 500 mg/m^3, and after its introduction the dustiness decreased to 3-5 mg/m^3 **(Figure 2)**. Another highly dusting process is the explosion, but with effective ventilation equipment and other anti-dusting measures a concentration of 2 mg/m^3 is reached. Loading is also a powder-generating process, the most intense in the stope, the characteristic here being that fine fractions occur less frequently.

General and constant source of dusting in the underground mines is the settled dust in galleries and stopes that maintains a constant dusting background.

The silica content of mine dust is most often high. In many countries, incl. and in our country, for silico-dangerous are defined, mines with dust containing more than 10% free crystalline silica.

The dispersion of mining dust, whether in open or underground mining, is extremely high - dusts up to 5 μm in excess of 90%. The harder the breed, the more fine the powder is formed. Compressor air pressure or wearing of the drill bits also increase the finely divided powder fraction.

In addition to the dust, a significant amount of oily aerosol is excreted in the mining air during the operation of pneumatic hammers. Depending on the used lubricating oil, more or less carcinogenic substances can be found in this aerosol. In the air in the workplaces also particles of the mechanical impurities contained in the water used in the wet drilling and the various methods of irrigation, falled.

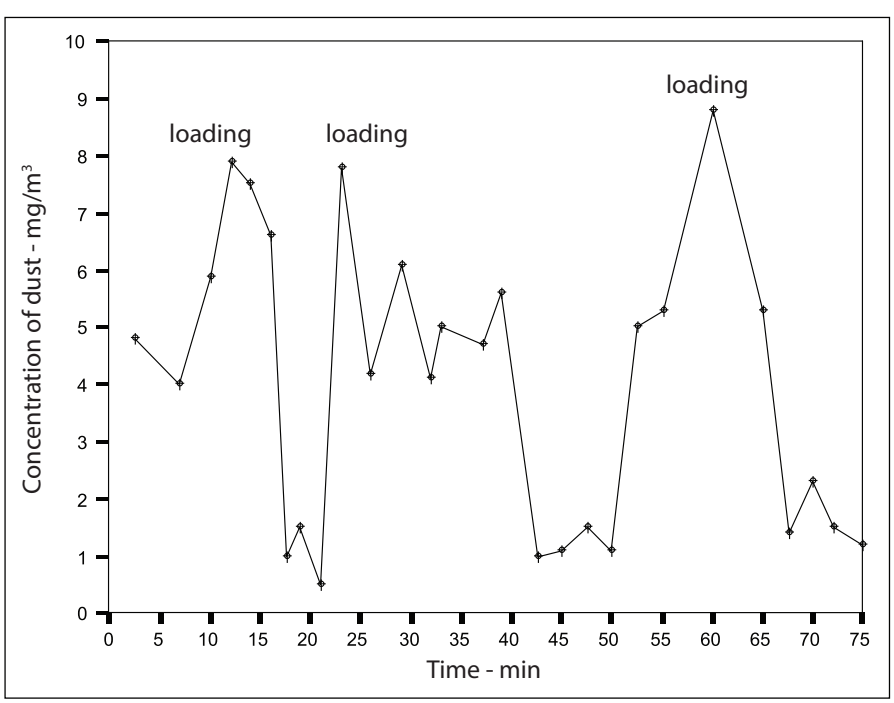

Fig. 1. Dynamics of the dust content of the excavator cabin in an open cast mine (S.V. Aleksiev, V.R. Usenko)

Toxic gases in mine workings can come from the ore that is being processed; as a result of the explosion; from road transport, from underground fires, rot processes, from compressor air, etc.

In open mining, the dust cloud raise at 100-150 m during blasting. Removing this cloud beyond the mine limits depends on the speed and direction of the wind, the size of the mine, etc. Normally, after 5-6 minutes, the concentrations of carbon monoxide and nitrogen oxides fall to the limit values. Higher and longer (up to 6-7 h) gaseous state can be expected with temperature inversions and windlessness. It is possible

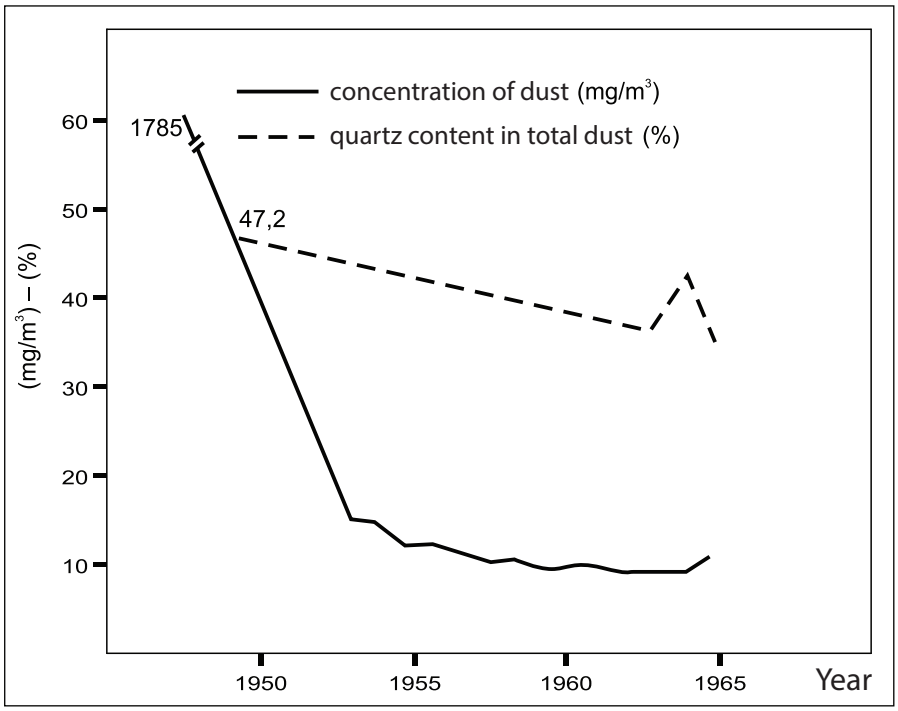

Fig. 2. Powder-hygienic characteristics of the Gorubso mines (by E. Petrova)

further extraction of gases from the demolished rock and ore mass.

Autotransport in the open cast mine constantly pollutes the air. Car exhaust gases are a complex mixture of toxic gases depending on the type of fuel, the type of engine, its operating mode, the degree of wear, etc. Their concentrations vary widely and are higher when the mine is deeper.

In the conditions of the underground mines, carbon monoxide and nitrogen gases are of hygienic importance and their quantity depends on the type of explosives used. With the modern organization of blasting mining at the end of the shift and subsequent two-hour or one-hour ventilation before the next shift, the blasting gases are usually not a serious hygiene problem. It is possible to accumulate larger quantities of these gases in the extracted material and to release them at the start of loading. Policyclic aromatic hydrocarbons resulting from the thermal destruction of mineral lubricating oils can also be found in the precipitated dust and as vapors.

In the polymetal mines (mainly in lead-zinc), the air of the working environment can also be contaminated with radon.

Mining **lighting** is one of the factors for safe and high-performance work. Stationary and portable lighting (acetylene or rechargeable lamps) is used, but it is often inadequate in intensity or with a high contrast object/background.

The labour process of the main professional groups in miners is varied. Some of the technological operations are mechanized, others are performed manually, and the relative share of manual labour, especially in underground mining, is high.

In the open cast mine, the main professions are: drilling, digger, bulldozer, digger, heavy truck driver, bomber, mine maintenance worker. Their main production activities are characterized by a moderate burden of labour, but in the case of repair and auxiliary work, the physical load, especially of the diggers, bulldozers and drills, is great. In all occupations, the static load on the musculoskeletal system is considerable. Significantly nervous-mental tension is experienced by the work of diggers, drills and bombers - the most pronounced it is for heavy-truck drivers.

In the underground mines the different types of activities are related to dynamic physical load, which does not differ significantly in the basic mining professions from the so-called "stope group" - master-digger, assistant master, driller, bomber, and cap (strengter). Almost all work operations (without drilling the blast holes) are performed by hand tools.

The static physical load in this group of miners is also expressed. The working posture is often compulsive, inconvenient, in a limited space with raised hands, and so on. Maintenance of the boring hammers is also associated with a static effort.

The work of miners requires constant attention, speed, dexterity, operational and long-term memory associated with visual tension. High labour risk requires responsibility for occupational safety, as well as discipline and emotional sustainability.

A major occupation in the underground mine is also the electric driver. His work is not associated with a heavy physical load, but requires a large and continuous visual load, good hearing, caution, speed and security in driving, resulting in pronounced nervous-sensory stress. Besides the professions that are directly connected with the implementation of the basic technological processes, workers from the so-called repair staff. Depending on the nature of the work performed, it may be associated with mild to high physical stress, with static effort, with significant but not continuous visual tension. Work requires accuracy, attention and speed, very good qualifications.

General and occupational morbidity. The most

common occupational diseases among miners are silicosis, vibrational disease, auditory nerve neuritis. Occupational toxicities are also acute poisoning with toxic gases, especially in emergency situations.

Silicosis is a typical pneumoconiosis for underground mining and, most importantly, for miners from the "stope-group". The estimated number of silicosis and silicotuberculosis patients in Bulgaria in 1990 was 5,200. About 90% of them have late forms. The dust-exposed workers in danger to silicosis are 23504 in the country in 1990. The problem of silicosis remains on a global scale.

There are peculiarities in the clinic of silicosis from the various mines. Workers in the gold mines during the "dry" drilling of the blast holes form "egg shell" changes in the hyloxic lymph nodes and "pea disease". Zinc mining silicosis occurs late after a long dustless or latency period of dust extinction, and is typically characterized by macular shades type "snowstorms", which are scarcely delimited. Silicosis in the copper mines has a favorable slow motion, low morbidity and a picture of mixed pneumoconiosis. In the iron mines, silicosis or siderosilicosis with a favorable run can be expected. Uranium mining causes progressive silicosis, arising as a result of high quartz concentrations in the air aerosol from the working environment and the potentiating effect of radon impact.

Pneumoconiosis in open-cast mines miners is less frequent. Pneumoconiosis in these cases is benign and progresses very slowly. In individual opencast mines, signs of chronic bronchitis with combined etiology (from dust, adverse weather conditions, toxic exhaust gases) can be found after work over 15 years.

Vibrational disease. In the underground mines, the contingent is the digger-makers and their assistants. The adverse effects of local vibrations generated by pneumatic hammers are potentiated by general and local cooling and static physical over tension of upper limbs. In the vertical drilling of blast holes, diggers are exposed not only to local but also to general vibrational action.

Vibrational disease incidence among workers in opencast mines is less frequent. Separate cases of vibrational pathology have been found in crawler excavators after 10-15 years of work experience, with symptoms typical of general vibrations, combined with angiospasm manifestations of the hands.

Drillers, diggers, bulldozers and heavy-duty truck drivers may also be at risk of developing a vibratory disease. The pathology of the nervous system may be accompanied by lumbalgia, lumbar-circulatory radiculitis, and diseases of the locomotory system - osteochondrosis and deforming spondylosis primarily of the cervical and lumbar spine.

Neuritis of the auditory nerve is most often found in diggers and their auxiliaries in underground mines, and in individual cases in the presence of underground repair personnel, machine-tool operators, and others.

In addition to the nature of work and social-living factors, the **incidence of temporary incapacity for work** (ITIW) in the ore extraction, also affects the age-gender structure - only men are employed in the main occupations, and their age is usually between 20 and 40 years.

In the case of underground mining, the structure of ITIW in miners is mainly formed by chronic diseases of the respiratory system, skin and subcutaneous tissue infections, diseases of the peripheral nervous system (cervical and lumbosacral radiculitis, the locomotory system (osteochondrosis, deforming spondyloarthrosis), from gastrointestinal tract diseases (acute gastritis, gastroenterocolitis) and from labour traumatism. For the open-cast mining, the structure of the ITIW is similar, but from the diseases of the respiratory system leading are acute infections of the upper respiratory tract, of gastrointestinal diseases are ulcerative disease and chronic gastritis. Hypertension is also high. Diseases of the locomotory system, peripheral nervous system and hypertonic disease have a higher relative share in those over 40, and younger workers are more likely to suffer from acute upper respiratory tract infections and gastrointestinal tract infections.

Prevention of work. In the opencast mines, it is important to ensure the hygienically acceptable values of the **microclimatic factors** in the cabins of mining machines and road transport. The walls and their floors must be lined with heat-insulating material. The best option is conditioning them.

For workers all year in the open environment, it is important to provide work-wear suitable for winter or summer, as well as winter warming premisses and for rest periods during the summer. It is also necessary to consume hot liquids in the winter and cool mineral water and fruit drinks in the summer.

In underground mines, the main precaution against the discomfort microclimate is working clothes: a wet-protected top coat and trousers, woolen underwear, socks and boots. Hot bath after work is mandatory.

The complex of measures to reduce **noise and vibrations** in mining and their impact includes organizational, technical and medical means. Organizational considerations include: observance of special rules for the operation and repair of machines, rational working regiment, extended annual leave, etc. The most important organizational measure is

to reduce the time of exposure through the interchangeability of workers from different professions. The technical means of reducing noise and vibrations from mining equipment should be taken as a rule by the producer companies, but only as an exception, by the users. Full elimination of the noise-vibration effect can be achieved by remote control of mining machines wherever is possible.

In the open-cast mines, the general principle of limiting vibration and noise is, above all, the vibration and sound insulation of the machinery cabs. In order to reduce the vibrations and impulses of the heavy-duty trucks it is important to have the roads in the open-cast mine with concrete or reinforced concrete slabs and the right choice of the speed of movement.

In underground mines to reduce the harmful impact of vibrations, the use of "self-driving" drilling machines, including automatic and remote control, will be very effective. Vibrocarretes and vibrating handles are used for the commonly used hammer drills. Vibrocarretes are designed to protect hands when drilling horizontal and sloping holes (they are spring shock absorbers and reduce vibration at all frequencies). In order to reduce the vibrations of the load machines, shock absorbers for the chassis are used.

Substantial noise reduction is achieved by soundproofing the engines of various mining machines, including and pneumatic hammers.

It is also mandatory to use individual anti-noise and vibration protection devices - anti-phones and impermeable and oil-resistant gloves with vibrating lining. In the complex of medical measures and means, apart from preliminary and periodical examinations, dispensary, labour readjustment, protective meals, physiotherapeutic measures - self-massage, gymnastics and hydroprocesses should not be overlooked.

In the open-cast mines the dust control is carried out in several directions: application of dust reduction methods, reduction of its intensity and removal of dust by natural or artificial ventilation. Dry dust removal or "wet drilling" is applied during drilling. If diggers are used - dewatering is applied. Covering the auto roads in the open cast mines with durable flooring - concrete or reinforced concrete slabs, wetting the roads with water (not less than 1 time per hour) also reduces the intensity of dusting.

Cabins for mining machinery and motor vehicles must be reliably secured by the dust (hermetic) and have air-purifying ventilation equipment. The optimal option is air conditioning.

To reduce dust in **underground mines**, a complex of anti-dusting methods and means is used at each stage of the mining technology. The main place is dedicated to the so-called "wet drilling" of explosive holes. The efficiency of dust extraction and retention of dust is increased by adding to the water the so-called wetters and foams.

In cases where mining conditions do not allow the use of water drilling, dry dust collection is used, which has a number of advantages over water drilling-capture and the finest particles of dust, as well as reduced humidity in the workplace and wetting of clothes, shoes and hands.

Removal dust in mining is also carried out by dispersing water (irrigation) at the dusting sites. Water cartridges, water balloons, water curtains are used. Dew is subject to the walls and ceiling of mine workings at the pre-blasting work, the gas dust cloud itself after blasting, the weighed rock mass after blasting and preloading, the dusty ventilation flow coming out of the debris and loading/unloading points in the mine.

Reducing concentrations of **toxic gas** from road transport in **open-cast mines** can be achieved by using catalysts. It is very important to keep engines in good technical condition (regular repairs, adjustments, etc.). In case of inversion and windlessness, if there is accumulation of toxic gases, work in the pit should be stopped.

In the underground mines the blasting must be done at the end of the working shift; between every two working shifts a mandatory ventilation time of 1-2 hours was established.

The main purpose of avoiding the formation of large quantities of explosive gases in the mining industry is the use of explosives having a correctly estimated oxygen balance allowing their full (no residue) burning.

Correctly conducted **ventilation** in **underground mines** is essential for: normalization of the mine microclimate, reduction of dust and toxic gases to the MAC. There are powerful stationary ventilation systems. The flow rate of the required air to ventilate the mine is calculated on the basis of the calculations of the intensity of the dust in the particular operating environment. It is not allowed to alternately ventilate the stopes with the same ventilation jet.

The air supplied by the ventilation system in the mine should be clean, which requires special attention to the complete dustless of the area around the shaft.

Much of the accidents and diseases in the mines are due to the non-use of personal protective equipment. They protect workers from discomforting microclimate, from dust, from noise, from electrical current, from moving parts of machines, from falling

rock scale/ore, etc. Personal protective equipment includes special work clothing and boots, gloves and helmet, as well as safety devices - brackets to protect the hands when cutting support materials, safety belts, glasses, dust masks, antiphonics, etc.

Medical-prophylactic measures. Due to the difficult conditions for working in the mining industry, those who want to work in this sector are subject to increased requirements. The contraindications for getting into work must be strictly adhered to and the results of periodic medical examinations analyzed.

Special sanatoriums for recreation and restoration of health have been created for mining workers. Profilactic professional nutrition are provided.

17.2.2. ORE PROCESSING AND DRESSING

Typically, a mine (open or underground) is also being built a flotation factory, where the initial processing of the ore starts. The technology includes the operations: ore grinding, screening, fine milling and dressing (flotation).

The ore is delivered to the bunker at the flotation factory with heavy-duty trucks. For crushing use jaw and cone crushers, for sieving - vibration screens. The transmission of the shredded product is carry by conveyor belts. Prior to the enrichment, the crushed ore was finely grind in drum mills in an aqueous medium. The resulting mixture is transported for flotation. Flotation is performed by chemical reagents in flotation machines (open-area chambers). In the enrichment of polymetal ores, "collective" (separation of all useful minerals from the "deserted" ore) and "selective" (separation of different minerals from each other) flotation is applied.

The concentrates obtained at the dressing are sent to the metallurgical plants for the extraction of pure metals.

When dressing ores in floation factories workers are exposed to an aerosol with complex composition, noise, vibration, discomfort microclimate. As the process progresses, the composition of the aerosol changes: in the crushing compartments it is predominantly dusty and the fine powder and quartz content in it most often exceeds the MAC several times. In the reagent and flotation compartments the vapors of xentogenat, hydrogen sulphide, sulfuric acid, etc., predominate, and their concentration in the working environment depends on the efficiency of the sanitary and technical facilities. Noise and general vibrations are most characteristic of crushing compartments. Near the crushers and vibration screens, the noise exceeds the allowable level by 2 to 8 dB/A on average, and the low and medium frequencies prevail in its spectrum. General vibrations also exceed the permissible norm.

Employees serving flotation factories have a different occupational risk. Those serving crushing compartments and conveyor belts do not have a fixed work places. Walking on the various machines, they are exposed to all the above-mentioned unfavorable factors. In the reagent and flotation compartments the operator type of work is predominant and the exposure is predominantly of the toxic chemical reagents.

The morbidity of the workers from the flotation factories has no specific features - cases of pneumoconiosis, chronic bronchitis, subtrophic and atrophic rhinitis and pharyngitis, diseases of the locomotory system and others.

The healing measures in the flotation factories should be aimed at: sealing and controlling aspiration of the dust sources, especially in the so-called "purings" (for site transfer, material puring), reduction heigh of puring, wetting of the ore, humid cleaning of the premises, mechanization of the processes for unloading of the reagents and preparation of the solutions, use of less toxic reagents, cladding of the machines and the walls of equipment with sound absorbing materials, vibro-insulating platforms, use of personal protective equipment, etc.

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17.3 FEROUS AND NON-FEROUS METALLURGY

17.3.1. FEROUS METALLURGY

Metallurgy includes the following main industries: agglomeration, blast-furnace, steel foundry, rolled-mil and ferro-alloy.

Agglomeration production provides the necessary high quality raw material for the metallurgical cycle. The technological process goes through: stock preparation (crushing and primary mixing), secondary mixing and caking with the end product - pieces of agglomerate and agglomeration to obtain a purified agglomerate.

The workers in the agglomeration production are exposed to a complex of dangers among which dust is first. Pollution varies widely between 20 and 200 mg/m^3. Most significant is the grinding of the stock, the concentrate, the coke, the limestone; in mixing drums; in the premises of the return cycle; in the places where the raw materials are processed from one equipment to another; in vibrating screens; when loading the agglomerate, etc. The main components of the powder are iron compounds (up to 65%), calcium (up to 20%), silicon dioxide (up to 12% - including free silica to 1-2%). For polymetallic ores the powder may contain other heavy metals - lead, manganese and others. 85 to 88% of the powder particles have dimensions up to 2 and 99% up to 5 μm.

The microclimate is adversely affected by intensive separation of infrared radiation (with values up to 2550 W/m^2) and convection heat, on many areas in production premises with heat sources. During the cold period of the year the production microclimate is overcooled with predominantly sub-normal temperatures and in places with strong air currents (up to 1.5 m/s).

Noise exceeds sanitary-hygienic norms (broadband with an intensity of up to 96 dB) only in places at agglo machines.

The working position is most often right - up to 35% of the shift time, sometimes a significant load, and the energy consumption is usually within the limits of heavy work.

As the leading professional nose is the production dust, the most common disease is bronchitis. It is characterized by a low run, is not accompanied by pronounced functional disturbances of external breathing, and is rarely complicated by an asthmatic component. The colds, as well as those of the peripheral nervous system and the locomotory system, have increased.

In the **blasting industry**, reductive melting of agglomerates, iron ores, fluxes and coke is achieved in order to produce cast iron. The process is continuous and is carried out in blast furnaces at a melting point of up to 1800°C. The addition of fluxes (usually limestone) helps to melt, and the bonding of lime with iron oxides leads to the formation of large amounts of slag. It swims over the cast flow and runs through the chutes - the slagflow, at the top of the furnace. From below, through a cast flow, pour cast iron into cast truck and transport to remake to steel or foundry pig iron.

The leading professional noxa is the extremely unfavorable microclimate. Conditions for overheating due to intense infrared radiation in the short wavelength of the spectrum, especially when the slag and cast iron are flowing (3500 to 7000 W/m^2) are created. In summer, the ambient air temperature reaches 35 ° C and in some cases up to 45 ° C. On the other hand, strong air currents (with air velocity above 5 m/s) and cold temperatures in winter (up to 1-2 ° C above the ambient air temperature) cause overcooling of workers with all health consequences.

Most of the workers (excluding those in the open-air ore yard) are exposed to excessive dust concentrations (similar in composition and dispersion to agglomeration production). A major danger is the release of blast furnace gas (mainly due to the increased CO content). Particularly endangered are workers at the colliery platforme, the hydroinsulators and the openings of the gas pipelines, the joints of the dust collectors with the raw gas collector and others. Toxic-chemical damages also have to be mentioned the pollution with sulfur dioxide, lead aerosols and others. The harmful impact of these noxae is enhanced by the synergistic action of the overheating microclimate.

The noise near blast furnaces is usually medium and high frequency with an intensity of 86-90 dB/A (although in some places it reaches too high values up to - 110 dB).

Many of the work operations are done manually and are associated with high energy consumption (at cast iron flowers and bucketmakers reaching

over 21 kJ/min). The labour of the flowers and gas-workers, where the energy consumption is below 20 kJ/min, is lighter. But at them, the job is associated with a certain high responsibility and control, and to the gas workers high qualification is necessary.

The most frequent health effects for workers include: danger of overheating or overcooling, increased colds, peripheral nervous system and locomotor system disease, trauma, severe burns, acute and chronic intoxications, bronchitis, etc.

Steel is produced by melting iron, mixed with iron waste or pure iron ores. This is done in three technological variants, defining the three types of workshops: marten, bessemer and thomas and electric steel foundry. Generally, in all three cases, too much infrared radiation and high air temperature are present. Sources of infrared radiation are the furnaces, the casting-shuttles, and the high temperature of the air contributes to the hot gases coming through the working openings in the furnaces. The technological cycle passes through three phases: preparing stock materials and filling the furnace, flushing and dropping the steel, spilling the steel.

In the stock compartments, the main disadvantage is the high dustiness of the air. The temperature regime is completely dependent on the weather, as these compartments are not heated and there are no heat sources. The speed of air movement varies, but the most characteristic are its high values - above 2 m/s. Workers here have a energy consumption of more than 21 kJ/min. Significantly loading the locomotorial apparatus from physical effort as well as from unfavorable working positions. The health effects are: bronchitis, increased colds and those of the locomotory system and the peripheral nervous system, and so on.

Mixer compartment workers are exposed to infrared radiation to a greater extent than those in blast shops, and summer air temperatures exceed 5 to 13°C outside air. During the winter season, however, subnormal and even negative temperatures are very common.

Toxicochemicals are: dusts at concentrations up to 70 mg/m^3 (with iron oxides but also free silica up to 10%), condensation metal aerosols (manganese, lead, etc.) with very high dispersion and concentrations above the MAC; blast furnace gas, CO_2, sulfur dioxide and others. (In the bessemer and thomas workshops, noxious gases in the air of the rooms may be released only when the convertors are blown).

Workers here are almost always upright, with a high intershift load of up to 80%, with more than half of this time performing manual labour with a energy output of over 21 kJ/min. Only in case of mixers in the converter the energy consumption can be within the range of 21 kJ/min.

The health consequences for workers are: the risk of overheating and overcooling; Increase of: cold morbidity, diseases of bone, peripheral nervous, digestive (ulcers, gastritis, colitis) and cardiovascular (hypertension, atherosclerosis) systems; high level of traumatism; severe burns; acute and chronic intoxications and others.

In the **rolled-roll production** a plastic deformation of metal products is made in order to obtain different rails, sheets, pipes and the like. Rolling is hot and much less cold (without heating the metal). In hot rolling, huge amounts of heat are released. Infrared radiation is most often in the range of 2100-3500 to 5600 W/m^2, Significantly above the MAV. The air temperature in the summer is 30-40°C (sometimes up to 50°C). In winter, however, only a few meters away from the furnaces and hot metal treatment, the temperature slightly differs from the outside. The speed of air movement is relatively high - up to 0.6 m/s and in some places even higher.

The noise is broadband, with low frequency prevalence, and is usually above the MAV - reaching 102 dB.

The powder consisting mainly of iron oxides is finely dispersed and its values are usually about 20 mg/m^3 (fluctuations are 0.2 to 70 mg/m^3). The toxicochemical factors - CO, sulfur gases, oil aerosols (although usually their concentrations exceed 3-4 times MAC) are of hygienic importance. In today's rolling production, the physical load is reduced, but it is still high (over 21 kJ/min) in a number of workplaces.

The unfavorable combination of injuries leads to: danger of overheating, heat stroke, overload of cardiovascular and respiratory systems, increase of cold morbidity, traumatism, burns, diseases of the locomotory system and of the peripheral nervous system, etc.

Ferro-alloy production provides necessary for steels casting iron alloys with manganese, silicon, chromium and others. The raw materials are ores, concentrates, converter slag, iron chips, coke, fluxes (limestone and dolomite). The technological cycle is continuous. It goes through pouring of the starting materials into the bunkers, filling the furnaces with electrode masses and stocks, melting, casting (usually three times in 8 hours). The main occupational hazards in the areas without heat release are: the unfavorable microclimate, entirely dependent on the weather conditions of the season and the high dustiness. The microclimate has high infrared radiation (400 to 2800 W/m^2) at workplaces near to thermal production sources. In the rest of the shop the microclimate is almost entirely under the influence

of atmospheric air. Dustiness exceeds MAC several times, and toxic gases, vapors and aerosols are usually at the limits of the MAC, although at times they may exceed 2 to 5 times. The work of conveyor drivers, dosers, operators of various control instruments has an energy consumption of 10.46 kJ/min, the dosers are within the average workload and the other workers are characterized by considerable physical load (especially in some technological operations).

The health effects are: the danger of overheating with infrared radiation - to heat stroke, increased colds, traumatism, diseases of the peripheral nerve and bone-muscular systems.

The main **hygienic measures** in the iron metallurgy are: proper organization of aeration by special roof structures - Pond type and openings for natural ventilation (usually in the upper parts of the walls); air showers on workplaces with intensive heat dissipation; water curtains - especially in front of the openings of the furnaces; reducing airflows with moving curtains - especially at the entrance/exit openings of the premises; sealing and encapsulation of intensive dust excretion facilities and processes; implementing automation and remote control wherever possible; regular wetting of the stock, including through special irrigation systems; provision of efficient general and local ventilation facilities; regular cleaning of the planted dust, and of windows and artificial light sources with special solutions according to a suitable formulation; introducing scientifically-based work and rest regimes with the provision of air-conditioned recreation rooms; mandatory use of carefully selected personal protective equipment (especially for dust, toxic aerosols and noise); providing appropriate workwear, underwear and shoes; provision of professional protective meals - Type A diet (with products rich in essential amino acids, vitamins C, B, A, milk, liquid foods and beverages, including low salt content mineral water); mandatory and regular conduct of preliminary and periodical medical examinations; dispensary of diseaseds and rehabilitation when needed, etc.

17.3.2. NON-FERROUS METALLURGY

After extraction of the ore containing non-ferrous metals (copper, lead, zinc, cadmium, etc.), it is enriched in special flotation plants to a concentrate from which the respective non-ferrous metals are extracted.

In non-ferrous metallurgy, the main production processes and deficiencies are similar to those in the iron and steel industry. A significant difference, which also affects the health consequences, is the additional presence of specific hazards typical of the respective production: copper, lead, nickel, zinc, cadmium, etc. This leads most often to acute and/or chronic intoxications characteristic of the particular non-ferrous metal yield.

Copper production. The raw material from which copper is produced is an enriched copper concentrate, which contains impurities such as sulfur (containing up to 40%), silicon, iron, zinc, aluminum, nickel, lead and the like.

The main stages of the process are: preparation of stock (dosing and mixing of components); frying the stock until it gets scum; melting and obtaining of copper matte; converter obtaining black copper; raffinate to obtain anode copper.

The stock frying is carried out at 800-850 ° C in different types of frying ovens, with a "boiling layer" furnace are advantage in technical and hygienic terms. Sulfur dioxide-containing gases separated there are discharged to form sulfuric acid.

The scum concentrate obtained after frying, consists of copper oxides, FeO and copper sulphide. The next operation is to melt the scum in an electric furnace at ~ 1500 ° C. When melting, two liquid phases are formed: copper matte - (an alloy of sulphides in which copper and other valuable components are concentrated) and slag (an alloy of oxides and silicates that does not contain copper in usable quantities).

The extraction of raw copper from melted copper matte is done in converters. Air or oxygen is supplied through the special openings of the converter, where the impurities burn or pass into the slag. The resulting black copper has a purity of 98-99%. Impurities are removed in subsequent technological operations - firing or electrolytic refining. The refining of black copper is carried out in a flame-reflecting furnace. The electrolytic refining of copper is carried out in the electrolysis workshop in special matrix baths with a high acid solution. Electrolytic refining of copper as a by-product produces copper sulphate - the most important technical salt of copper.

One of the leading disadvantages of the working environment of the predominant number of work places the **microclimate**. A number of technological processes (frying, melting, converting black copper) are accompanied by the release of large amounts of heat, a significant part of which is released as infrared radiation. Powerful sources of thermal radiation are heated furnaces, open flame, molten metal, copper matte and slag. The highest in the copper production is the temperatures of the workplaces when pouring the copper matte and slag. Heat radiation (mainly as infrared radiation) when handling molten metals is of considerable intensity - reaches 4900-8400 W/m^2. At the same time, the air temperature

is significantly lower in working places, standing at 5-10 m from the intensive heat sources, especially during the cold period of the year (temperatures below + 5 ° C are measured). Thus, the microclimate can be defined as **mosaic**. Typical for shops with intensive thermal power is the high airflow rate up to 2-2.5 m/s.

Some processes (eg. stocking of ore materials) are carried out in open halls and the microclimate in them is directly dependent on the macroclimate.

An exception to these microclimate features are the electrolytic refining workshops - they are constant and tend to overheat during both seasons of the year.

Noise exceeds hygienic norms of about 1/3 of workplaces (for furnaces, converters, compressors, etc.) and its intensity reaches 100 dB.

Copper mines are concerned with "dusty" production. In the initial stages of the process the **dust** is separated by crushing the starting materials and when the crushed material of the conveyor belts is poured. Most of the dust in the copper production is with the roasting and the electric casting. Smelting, conversion, roasting of slag and copper matte creates highly dispersed condensation aerosols (90-98% of the particulate is less than 1 μm). Dust formed in different technological stages contains mainly iron (15-27%), copper (10-13%), silica (36-53%), zinc (3-7%) - **Table. 1.**

The presence of stream air in the working environment causes dust to enter into naturally sedimentary dust, which increases dustiness and keeps it high also in technological operations where no intense dust separation is characteristic. Due to the high sulfur content of the raw materials, the main toxicochemical factor in the copper production is sulfur dioxide, the highest concentrations being found in the roasting of copper matte and the slag and in the blowing of the converter **(Table 1)**. Often the feedstocks contain other impurities besides copper, so compounds of arsenic, zinc and lead are found in the air in the working environment.

Adverse health effects for workers. The most common are upper respiratory tract diseases (and as a result of irritating action of sulfur gases), peripheral nervous system, digestive and cardiovascular system. There is a risk of overheating and heat stroke, as well as grave burns.

Production of lead. The raw materials for lead are sulphide lead ores containing the mineral galenit - PbS. Enriched lead concentrates, containing 40-70% lead are used.

The first technological operation is agglomerating roasting, where at 700-900 ° C the lead sulphides are converted to oxides to produce a burned, agglomerated material. Melting of the agglomerate is carried out in a stock furnace in a mixture with coke at a temperature of up to 1500 ° C, where lead oxides are reduced to metal. The stock casting produces: black lead containing copper, gold, silver and other impurities, copper matte (alloy of metal sulphides) and slag (a non-metal oxide alloy often containing a lot of zinc).

The purification of black lead is usually carried out by the so-called fire refining. Refining aims at removing impurities (copper, arsenic, zinc, silver, etc.) and increasing the lead content to levels that meet the technical standards. Refinery plants are housed in a separate building. The cleaning is carried out in refinery boilers at a temperature of 500-600 ° C. The high-quality refined lead spills into molds using special machines at a temperature of 450-500 ° C.

Tab. 1. Major technological processes, raw materials and pollutants in copper production *

Technological process	Raw materials	Air pollutants	Industrial waste
Receipt of copper concentrate	Copper ore, water, chemical reagents, sealants	Sulphur dioxide, dust containing arsenic, antimony, cadmium, lead, mercury and zinc	Flotation wastewater, waste containing limestone and quartz
Extracting honey	Copper concentrate, sulfuric acid		Heaps of concentrated acid solution
Melting	Copper concentrate, flux		A slurry containing the iron sulfide and silicon
Convert	Matte flux	Sulphur dioxide, dust containing arsenic, antimony, cadmium, lead, mercury and zinc	A slurry containing the iron sulfide and silicon
Electrolytic refining	Copper, sulfuric acid		A slurry containing impurities such as gold, silver, arsenic, antimony, cadmium, lead, mercury, nickel and others.

* In P. Roto, 1998

And in the production of lead the **microclimate** is one of the leading adverse factors of the work environment. The characteristic of microclimatic factor as a whole is similar to that of the copper production. The working places in the agglomeration furnaces have measured air temperatures of 34-36 ° C at a relative humidity of 75-88%; The thermal radiation of these workplaces is intense - 1700-2300 W/m^2. Similar are the thermal conditions of the melt of the agglomerate, and the intensity of thermal radiation in the refining plants is generally lowered.

The **noise** characteristic is similar to that of copper.

Most **dust** of lead production are released in the agglomeration (loading, roasting, stocking) and melting workshops. The dust dispersion is high - 90% of the dust particles are up to 5μm in size. High levels of lead and silica in dust require a strict system of prophylactic measures.

The main **toxico-chemical hazards** that determine the risk of occupational poisoning are lead aerosols. Their concentrations increase rapidly in the processes of pouring matte and slag. When refining from the exposed surfaces of the molten metal in the air of the working environment, besides lead aerosols, lead and nitrogen oxides, aerosols of zinc and other metals are released; overall concentrations of toxic substances in these plants are lower. In some cases, due to the fact that the outside air is also polluted with lead aerosols (in the case of a failure of the ventilation system filters), the air exhausted by it may be a source of aerosols for contamination of the working environment.

Sulfur dioxide is one of the main pollutants in the air and in lead production - mainly in agglomeration roasting and in batch kilns, but concentrations are significantly lower than copper production **(Table 2)**.

Adverse health effects for workers. Diseases of the digestive system are frequent (for the increased incidence of this pathology the role of lead exposure is considered proven), the nervous system and the upper respiratory tract. Severe forms of lead-like intoxication have recently been relatively rare, and light forms have been shown to be characteristic of biochemical abnormalities without clinical symptoms. The main reasons for chronic professional intoxications are the various disturbances of the technological processes and deficiencies of the ventilation equipment. Also, the toxic action of lead is enhanced by the impact of overheating microclimate and heavy physical work.

Zinc production. The zinc content in the polymetal ores is 2-12%, which is why they are enriched by the selective flotation method. The obtained zinc concentrate contains 40-70% zinc, as well as impurities - sulfur (containing up to 30-37%), lead, copper, iron, cadmium etc.

Zinc concentrates can be obtained by pyrometallurgical or hydrometallurgical method. In the pyrometallurgical process, the zinc is obtained by reduction with charcoal and retorts of pre-oxidized or roasted ore. In the hydrometallurgical method, the zinc is recovered from its sulphate solutions by electrolysis. In the world, both methods are almost equally applied.

In both methods, the processing of the concentrate starts with roasting. The process is carried out by blowing air through a layer of concentrates at a temperature of 1200-1300 ° C. When roasting, gases containing 4-6% sulfur dioxide are formed. In the pyrometallurgical process, the next process is the distillation of zinc at a temperature of the mixture of the roasting concentrate and the coke 1250-1350 ° C. Zinc is reduced and evaporated; its vapors in a mixture with carbon monoxide are passed to a condenser in which molten zinc forms at a temperature of 450-500 ° C. The zinc obtained by the distillation process contains 1 to 3% impurities (lead, iron, cadmium, copper, arsenic, etc.) and is therefore refined, and is 99.995% pure.

In the hydrometallurgical production of zinc, roasted concentrates are treated with hot sulfuric

Tab. 2. Major technological processes, raw materials and pollutants in lead production *

Technological process	Raw materials	Air pollutants	Industrial waste
Agglomeration roasting	Lead ore, iron, limestone flux, coke, soda, dust, pyrite, zinc	Sulfur dioxide, dust containing cadmium and lead	
Melting	Lead agglomerate, coke	Sulfur dioxide, dust containing lead and cadmium	Slag containing impurities such as zinc, iron, silicon and lime, industrial sewage
Electrolytic refining	Black, "raw" lead		

* In P. Roto, 1998

acid solutions. In addition to the zinc, other impurities pass through the solution, so the sulfate solution is purified by partial hydrolysis. The purified solution is subjected to electrolysis and the zinc is deposited on the cathode. The operating temperature is 30-36 ° C and, to maintain, the electrolyte is cooled. Leaves of cathode zinc are removed manually and melted into induction furnaces, after which the metal spills into molds.

In the frying of the zinc concentrate work places a warming **microclimate** is formed - during the warm season of the year, the air temperature reaches 28-34 ° C at a relative humidity of 44-61% and an air velocity of 0.4-1.7m/s. The heat radiation of these work places is 800-1700 W/m2. At work places at induction furnaces where cathode zinc is melted, the air temperature is 28-34 ° C at a relative humidity of 42-57% and a thermal radiation up to 1400 W/m^2. Electrolysis shops are characterized by the high relative humidity of the air - 80-98%.

Due to the fact that the processed stocks and the materials used in the technological process contain many chemical elements, a number of pollutants are included in the air in the working environment- zinc, copper, lead, arsenic, cadmium, sulfur compounds sulfuric acid, chlorine, etc. **(Table 3)**. Large concentrations of H_2SO_4, HCL, $ZnSO_4$, $ZnCl_2$, SO_2, as well as arsenic, lead, chlorine, manganese are measured in the respiratory area of electrolysis shops.

Some of the manufacturing operations (especially manual removal of leaves from cathode zinc) are associated with a significant heavy labour.

Adverse health effects for workers. Inhalation of zinc vapors in concentrations above 15 mg/m^3 may result in the so-called "founder fever". In humans, chronic zinc intoxications have not been established. There is evidence of an increased incidence of chromosomal abnormalities in the leukocytes of workers exposed to zinc.

The incidence of respiratory, digestive (gastritis and ulcer disease), cardiovascular and nervous system diseases is increased. The effects of production dust, sulfur compounds and acid vapors cause diseases of the nasopharynx (mucosal hyperaemia, rhinitis, perforation of the nasal septum). At workers from electrolysis shops are more likely notice chronic pharyngitis - a subatrophic form. Roasted and electrolysis shops have notice signs of diffuse pneumosclerosis.

Production of cadmium. The cadmium is derived from secondary products in the processing of zinc, lead-zinc and copper-zinc ores: copper-cadmium cake, dust of roasting and distilling furnaces, etc. The cadmium content in them is from 0.2-0.5% to 6-7%.

From these products the cadmium is extracted (sometimes after oxidative roasting) by treatment with dilute sulfuric acid. The resulting cadmium sponge like containing up to 80% cadmium, up to 20% zinc, as well as iron and copper, is subjected to oxidation by staying in a warm and humid room for 2-3 weeks, then cadmium is extracted with dilute sulfuric acid. In some cases, the cadmium sponge like is not oxidized but treated directly with 75% sulfuric acid. The cadmium precipitates from the cadmium sulfate solutions by electrolysis of cadmium cathodes. The cathode electrolyte cadmium, after drying, is melted in iron boilers under a layer of molten sodium hydroxide at a temperature of 330-350 ° C and is cast in the form of rods or moulds.

The peculiarities of the technological process of cadmium production determine the unfavorably acting **toxico-chemical noxae**. Highest concentrations of cadmium in the air in the working environment were measured during melting and pouring into the moulds. The most toxic are cadmium oxide and its inorganic compounds - nitrate, sulfite, chloride and others. Relatively less toxic are its insoluble compounds - tellurium, sulfur and others. In case of cadmium production in the air of the working environment, sulfur compounds also occur.

Adverse health effects for workers. Cadmium and its compounds have a polytropic action. Toxic effects of cadmium can be subjectively manifested

Tab. 3. Basic technological processes, raw materials and pollutants in zinc production *

Technological process	Raw materials	Air pollutants	Industrial waste
Roasting	Zinc ore, coke	Sulfur dioxide, dust containing zinc and lead	Industrial wastewater
Extraction of zinc	Roasted concentrate, sulfuric acid, limestone		Industrial wastewater containing sulfuric acid
Purification	Sulfuric solution, zinc dust		Wastewater containing sulfuric acid
Electrolysis	Cleaned sulfuric solution, colloidal additives		

* In P. Roto, 1998

by weakness, nasal bleeding, increased excitability and tiredness. Upon objective investigation, inflammatory changes of the upper respiratory tract and the bronchi, emphysema, hippo- to anosmia, hepatitis, bone pain and deforming osteoarthritis, decreased calcium content in the urine, etc.

Cadmium accumulates in the body and remains prolonged in the kidneys. Typical symptoms of prolonged exposure to low cadmium concentrations are bronchitis, emphysema and proteinuria due to renal tubular damage. Additionally, anemia, hypertension, functional liver disorders and bone damage develop.

Manufacture of non-ferrous metal alloys. Currently, production is carried out in two ways - from primary non-ferrous metals by melting or burning at high temperatures or from secondary metals by melting industrial waste. The technology of production in the application of one or the other ways in principle does not differ, which allows it to be treated uniformly from a hygienic point of view.

The production of non-ferrous alloys includes the following basic technological processes: preparation of stock, melting in reflective or electric furnaces at a temperature of 500-1800 ° C and spillage of the metal. Refining fluxes consisting of fluorine, chlorine, carbon and other components are used in the melting. The alloys obtained in the melting shops are introduced into the processing units in the form of balls, which melt in electro-induction furnaces and spill to produce certain details (foundry alloys) or undergo hot or cold mechanical machining.

Due to the presence of powerful heat sources, the **microclimate** and the production of non-ferrous alloys are generally characterized as overheating.

Sources of **noise** and **general vibrations** are packet-presses, sorting conveyors, electro-induction furnaces, spill machines.

A leading unfavourable factor in this type of production is the production **dust** containing substances with fibrogenic, toxic and carcinogenic effects. The main sources of dust are transport facilities, especially in the places where the raw materials are shipped from one equipment to another, dryers, melting furnaces, floating machines. The physicochemical properties of the powder are highly dispersed (disintegration or condensation) - dust particles up to 2 μm make up 65-70% of disintegrating aerosols and more than 95% of condensation. As a rule, dust particles contain oxides of the chemical elements in the alloy (aluminum, copper, zinc, silicon).

Often in the air of the working environment there are products of destruction of organic butyric substances (polycyclic aromatic hydrocarbons, acrolein, carbon monoxide).

Adverse health effects for workers. In the manufacture of non-ferrous alloys, the most common pathology is respiratory diseases (pharyngitis, nasopharyngitis and laryngitis, bronchitis, including asthmatic). The diseases of the musculoskeletal system (myofascites, radiculitis), of the digestive system (gastritis, gastroenteritis), of the skin and subcutaneous tissues, chronic intoxications are also frequent.

The main **hygienic and prophylactic measures** in the non-ferrous metallurgy can be summarized in several main directions. Anti-dusting and toxic substances include effective sealing of crushing, grinding, transporting and packing of concentrates, stocks, scums and agglomerates and of the mouth of the shaft furnace; efficient local gas extraction in the above-mentioned technological processes, as well as in the openings for the slags, matte and metal discharge and for the outflow chutes and the buckets for carrying them; full technological use of the released dust and gas waste products; the creation by mechanical ventilation of areas of clean air in the most polluted areas; efficient board ventilation of electrolytic baths and generalized ventilation of electrolysis shops; sealing and pneumatic and mechanical removal of powdery materials from the black lead refining pots; regular periodic wet cleaning of the floors and walls of the workshops and on the territory of the enterprises, and with industrial vacuums of the machines and equipment. In order to prevent the adverse impact of the production microclimate, it is necessary to effectively heat insulation and shield all heat sources (coking machines, furnaces, converters, etc.) and to provide a properly organized aeration. Fighting noise includes silencing of sources of intense noise (mills, screens, crushers, compressors, etc.) and in time compaction leakages of compressed gases.

Workers wear appropriate special workwear, gloves and shoes and personal protective equipment, to strictly adhere to personal hygiene requirements. For workers in non-ferrous metallurgy, it is necessary to provide professional food and drinking regime with appropriate mineral water - ex. contact with lead is provided with diet B and supplementation of chelators such as pectin, and for workers in contact with manganese, mercury, cadmium is provided diet B. For workers in contact with sulfur gases it is necessary to set mouth rinsing stations with 1% sodium bicarbonate solution and alkali inhalations. Pay attention to the professional selection of workers and periodic prophylactic examinations are carried out regularly.

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N. Gincheva

17.4 MECHANICAL ENGINEERING

The mechanical engineering is industry with a multiprofile activities. Includes heavy machine building - production of machines for power engineering, vehicles and equipment (automotive, shipbuilding, etc.), production of agricultural machinery, production of electric trucks and engine-driven trucks, electric hoists, metal cutting machines. The production of electric motors, accumulators, instrumentation, the production of computing and organizational equipment are also included in the mechanical engineering industry. Due to the heterogeneous production, the technological processes in machine-building enterprises are diverse. For the greater part of the heavy mechanical engineering, however, one-type production processes, machines and equipment are used that create similar labour-hygienic conditions. The main production stages carried out sequentially in the machine-building industry include: preparation of semi-finished products and their primary processing in foundry and forging-press industries; mechanical treatment, including the precision sizing of the product, mounting of the manufactured articles to obtain the machines and the equipment. The final stages are painting and metallic coating of individual parts or whole products to prevent corrosion and good commercial appearance of the finished product.

17.4.1. FOUNDRY PRODUCTION

Foundry production is a combination of technological processes for the production of molten metal castings in specially prepared forms. The traditional foundry cast materials are iron, steel, non-ferrous metals and their alloys.

Initially, drawings of the future product are made, and patterns (mostly wooden) corresponding to the outer shape and internal configuration of the finished product are made on the drawings of the construction. Casting forms represent the outer wall of the article. They also contain an axial - core portion defining the dimensions of the inner cavities of the article. The way of casting is determined according to the type of the molds and is carried out in two types: **casting in single (disintegrating) forms** - mostly molding or melting molds (from wax, coated with refractory composition) - precision casting; **casting in permanent, reusable forms** - metal cast (iron, steel) - metal mould or by refractory materials - with a capacity of several hundred to tens of thousands of castings. The most common casting method is in single mold casting molds - more than 2/3 of cast castings.

The main stages in the foundry production are: 1) Preparation of the molding soil and making shapes and hearts; 2) Burden preparation and melting of metal; 3) Pouring metal into shapes; 4) Demoulding, knocking and clearing (burnishing) deformation of castings **(Figure 1)**.

Working environment conditions.

The traditional method of casting in single-sided molds is the main source of unfavorable factors in the working environment.

The main hazardous and harmful factors of production are: high or low temperature and high speed of air movement, intense infrared radiation, separation of the working area of silicoso-dangerous dust and harmful chemical substances, presence of intense noise and vibrations, a large number of manual operations, danger of burning. All of these factors can be in different combinations and degrees of application in the different stages of the foundry production.

Microclimate. Foundry production is known as "hot" production - with significant heat load (high technological temperature and IR radiation). With massive heat input, only separate stages of the process are connected, while others run at normal and even low temperatures. The "hot" stages include melting and casting of metal, demoulding of castings, drying of mould and hearts, repair of a furnace. The rest, related to the preparation of casting earth, mold and heart shaping, the cleaning of castings refer to cold operations. Half of the heat emitted by a furnace (external oven temperature - 200 ° C) is IR radiated and the other is equal - by convection and combustion gases. Intense heat (regardless of air temperature) occurs during the filling of the mixer, the pots, during transport, when the metal is poured into the molds, when the slag is removed. Less radiation from castings. The air temperature in the foundries, in moderate climates, on hot days can reach the founder work places to 35-38 ° C and the infrared radiation at the moment of the molten metal dis-

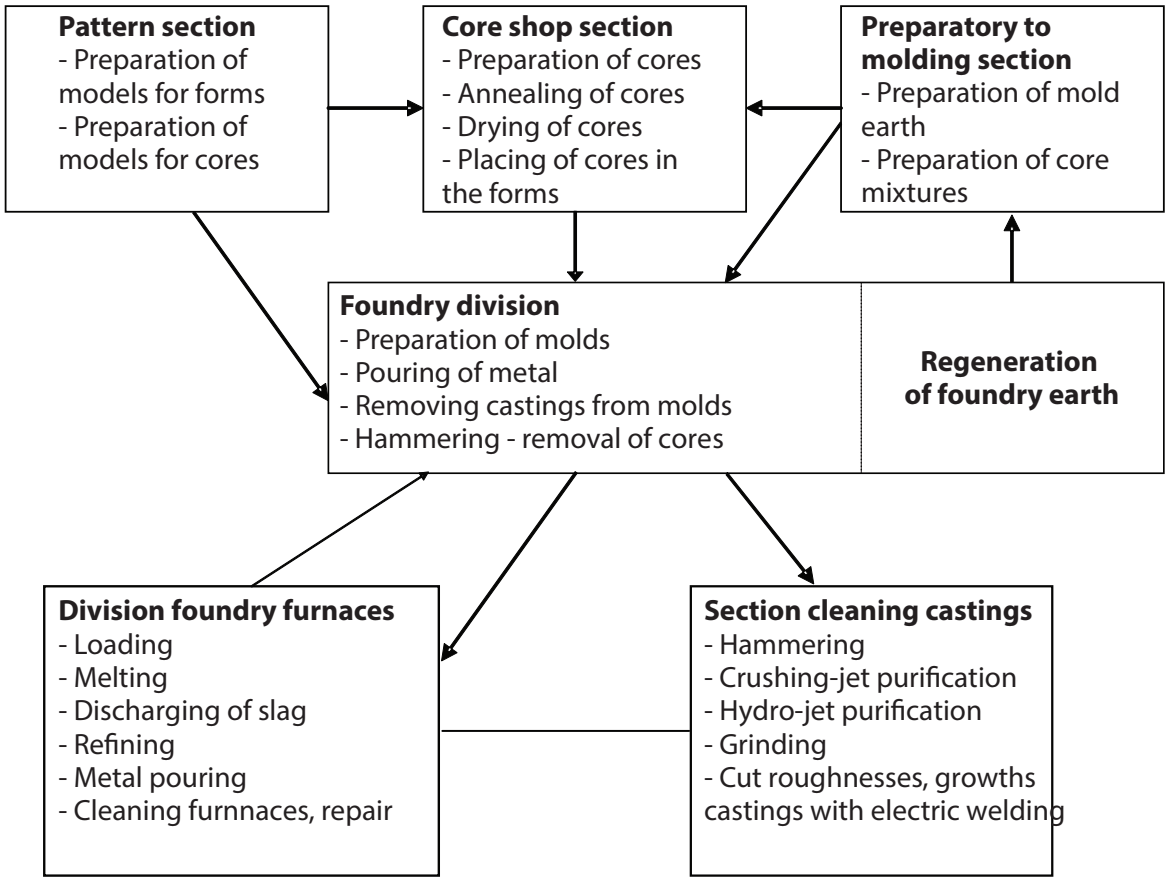

Fig. 1. Sequence of technological operations in a cast-iron workshop

charge - at 2 m distance - up to 6 kW/m^2, at 5 m - 4.3-3.3 kW/m^2. At the "casting dislodge" workplace, the temperature is 30-35 ° C. In casting processes with "in scattered casting", it is also possible that "cold" technological processes occur under overheating conditions. At individual workplaces and at different stages in foundry production, when located in a common hall, there are sharp differences in air temperature (especially during the cold season), which leads to the occurrence of significant air currents (dynamic microclimate).

Noise and vibration. In most of the processes in the foundry production, an intensive noise exceeding 85 dB/A is generated. Such features include the following: in the ground preparation - grinding, mixing and sifting of the molding earth and clay (coarse grain, ball mills, sieves); in molding - sealing of the molding earth in single casting molds using a vibro-stand, manual pneumatic rammer; when cleaning - by purification the casting of shaking grille, by shot-stream or by hydro-cleaning in chambers, in rotating drums, by grinding of stationary grinder aggregates or by manual pneumatic grinder. In all these operations noise levels of 90 to 110-120 dB/A peak values were measured. However, the background noise at the foundry is about 80 dB/A, with the noise being broad-spectrum. High frequency is in the case of grinders, medium frequency - for the molding machine, low frequency - for kollergangs. When compressing the molding earth with a molding machine, by vibration with dislodge grid generate general vibrations with a norms excess for frequencies above 8 Hz (8-63 Hz). With the impact of local vibrations, are molding operations using pneumatic rammering and the cleaning of piston castings with hand grinder are used to measure an over-integral vibration value for the 125-1000 Hz frequency range.

Dust. The specificity of the technological process in the foundry industry determines high dust exposure. All stages are related to the use (transport and processing) of powdery materials, and also by dust extraction operations: preparation and filling furnaces with burden, preparation of molding mixture and making of foundry molds and hearts, casting and cleaning of castings, repair fireproof in furnaces. For obtaining 1 t of casting, between 60 and 130 t of molding earth is processed. Powder concentrations in the air vary depending on the mechanization of production processes and the effectiveness of anti-dusting equipment. Concentration of total dust exceeds MAC in some productions up to 50 times. Highest concentrations of dust - up to tens of mg / m^3 are observed in the earthworks - in sand dryers,

soil mixers; in the foundry compartment - when the castings are dislodged, when cleansed with artificial grinders disks. In most cases small particles with a size of 3-5 μm (over 90%) predominate in the dust. The dust in the foundries is not homogeneous by chemical composition. Contains particles of sand, clay, carbon, iron oxide (released from purification casting of so-called scale) and many others components of the molding mixture. The adverse effect of the powder is mainly determined by its high dispersion and free crystalline SiO_2 content ranging from 2 to 30-50%. When heating the molding earth during casting (1500°C), crystalline modifications of SiO_2 may also occur - crystobalite and tridemite, which are believed to be higher in fibrogenicity. In some operations, other powders are released - the talc used to easily separate the model from the casting mold enters the working environment both when applying the model and releasing the model. Pure metal aerosols - iron, manganese, chromium, etc. are formed during the cleaning of the castings with oxygen or electric welding, with a size of less than 1 μm, also separating SiO_2. Wood dust is produced when making wooden models with hand-held mechanical or electro-tools.

Chemical compounds. The use of chemical substances and polymer materials in the preparation and processing of foundry molds, as well as the addition of various fluxes and alloys in the furnaces, the use of solid, liquid and gaseous fuels leads to the introduction of various chemical compounds into the air. These are: polycondensation metal aerosols (lead, manganese, copper, zinc, chromium, beryllium) and gases (carbon monoxide, sulfur dioxide, carbon dioxide); In the preparation and drying of foundry molds and hearts - phenol, formaldehyde, acrolein, ammonia, carbon monoxide, polycyclic aromatic hydrocarbons (benz (a) pyrene), furfuryl alcohol, diphenylmethyl diisocyanate, urea, sodium silicate, carbon dioxide; during casting - products of thermal and thermo- oxidation destruction of the supporters; in the destruction of oils - acrolein, carbon monoxide, etc.; in the destruction of polymers (self-hardening agents) - phenol, formaldehyde, isocyanates, ammonia, nitrogen dioxide, carbon monoxide, vapour of the resin catalysts - aromatic amines (based on pyridine). Of all the highest concentrations, carbon monoxide is defined - from 2 to 4 times above the MAC.

Working process of major professional groups.

The main activities in a foundry workshop are performed by the following professions: earth-preparator, formworker, heartmaker, burduner, founder, cleaner (crusher, grinder, electric-welder), operator of a shot-stream or hydro-cleaner. **Earth-preparator** (delivery of forming ingredients) is a profession with moderate physical stress and strain. This process in the large foundries is mechanized and partially automated while in the small ones it is carried out manually (with hand trucks) at a considerable distance (200-300 m) and in a forced working posture. The labour process takes place under the conditions of a cooling microclimate during the winter and transition seasons, over-noise, general vibrations and considerable dustiness. The work of the **formworker**, manually preparing the forms, with partial mechanization of the processes, is characterized by severe physical (dynamic and static) loading and moderate stress. The work is related to intermittent effects of intense noise, local or general vibrations (when sealing the form of a vibrator), often with a cooling microclimate. Chemical exposure is also possible if the drying of the molds is not done in furnaces but by drying the earth with methanol. Most of the operations are carried out in a forced working posture, blowing low to the floor. The **co-remaker** performs similar as form-worker operations, but all are executed manually. A large part of the operations are in a forced posture. It works under the conditions of a dynamic cooling microclimate and significant chemical exposure by the supporters. Labour is determined by moderate physical stress and tension. The **burdener** transports the burden (cast iron, foundry waste, old iron or rolled metal, blocks, steel, coke, lime, etc.) to a furnace, by hand, by hand- or electric truck - for small foundries, and trucks, wagons - for the big ones. Depending on the type of foundry and the mechanization of labour, the profession is characterized by moderate or severe physical stress and moderate tension. It works most often in the conditions of a dynamic microclimate, during the cold period - cooling (part of the work shift goes outdoors). The work is accompanied by the effects of intense noise and dustiness. The **founder (furnacer)** performs two types of operations: the first is related to the melting of the metal, the second one - to the repair of the furnace. The work is moderate, for some operations - with heavy physical stress and tension, in a forced posture. It works in a dynamic overheating microclimate environment, infrared and ultraviolet radiation, intense noise, local vibrations, dust with a crystal. SiO_2, chemical contaminants - CO, SO_2, polycondensation aerosols, and with a continuing danger of traumatism and burning. The **founder** is primarily concerned with casting the metal from the mixer into molds. In the small foundries the casting is done mainly by hand, and in the big ones - mechanized, with a crane. Work is associated with heavy physical stress and significant

tension. It runs under conditions of overheating microclimate - dynamic, high intensity of infrared radiation, noise, chemical pollutants emitted by thermal and thermo-oxidation destruction, upon contact of the molten metal with the form mixture - CO, CO_2, formaldehyde, acrolein, phenol, isocyanates, furfuril alcohol, sodium silicate. There is an increased risk of burning, traumatism. The **workers - cleaners**, in the case of non-mechanized dislodge of the molding earth from the castings with a hammer, a chopper, a lever, have a heavy physical load and considerable tension in a forced labour position. They work under high dust conditions with fine dust dispersion. It is possible to inhalate chemical products from the thermal decomposition of the resins, the oils included in the molding and core mixture. It works under the influence of pulse noise of considerable intensity. **In the case of cleaners-grinders**, dust exposure also includes metallic powder and grinder abrasive powder (usually aluminosilicate). A major adverse factor is the impact of intense constant noise and local vibrations. There is also an increased risk of traumatism and burns. Workers **cleaners serving the shaking grid** have exposure to general vibrations and intense noise with a discontinuous nature. Those **serving the chamber for shot-stream and hydro-cleaner and rotating drums** are mainly exposed to intense, intermittent noise. Their work is moderate physical stress and tension.

The **electric-welders** work is characterized by moderate physical stress and tension, often in a forced labour position. It works under the influence of ultraviolet and infrared radiation and exposure of chemical pollutants - weld metal aerosols with fine dispersion - less than 1 μm (iron, manganese, chromium) and gases (CO, CO_2, ozone, nitrogen oxides, hydrogen fluoride, etc.).

General and occupational morbidity.

Workers in the foundry production have a very high incidence of morbidity with temporary incapacity for work. High loss of work is primarily due to traumatism (domestic and labour). The greatest number of accidents at work were registered by the furnacers and the founders, followed by form-workers, cleaners and oxygen/electric-welders. The most common is eye trauma. Other high-intensity diseases include respiratory diseases (acute and chronic), muscular-skeletal system, peripheral nervous system, cardiovascular system. Of the occupational diseases with the highest relative share are the diseases of the musculoskeletal apparatus (epicondylites, myo-tendinosis, humero-scarapular periarthritis), PNC diseases (polyneuropathies), neuralgia of the auditory nerve, allergic diseases of the skin (contact dermatitis, eczema). There is a significant incidence of occupational diseases with dust etiology - chronic pulmonary bronchitis (often accompanied by emphysema) and slight forms of pneumofibrosis. They are mainly registered in form-workers and cleaners - grinders. Electric and oxygen-workers often experience pneumoconiosis - siderosis, as well as mixed pneumoconiosis. Less often in the foundry production there are people suffering from vibrational disease (vasospastic vibration syndrome), mainly in the case of workers with pneumatic tools (pneumotampers, pistols, grinders). The cases of "acute smog" (CO and SO_2) in founders and craners are single.

Prevention of work.

The fight against **dusting** involves: isolating high-dusting processes through rational planning of the building - separating the individual stages into separate rooms; sealing of dust sources; providing very good compaction of the cleaning chambers; maximum mechanization of dust-related processes; construction of local exhaust ventilation at all dust disposal sites; manual pneumatic tools to clean the castings no-frequently use or with built-in ventilation devices; weekly cleaning of the floor of the premises by removing the mold- and burning earth; use of wet methods for sand regeneration from the work off molding mixture; in castings cleansing operations use PPE - dust mask, dust-tight, working garment, closed to the neck (in the case of a shot-streaming outside the camera - inflatable protective clothing); periodic medical examinations with a focus on respiratory function research.

In order to reduce the **noise and the vibratory impact** in the foundry production, it is mainly the architectural and construction solution of the workshop, with isolation of the noisy machines and processes and of the vibration generating machines in separate rooms, in addition to specific operations, specific measures are applied. For example, when cleaning castings with removable grinders in non-stationary workplaces, movable screens with sound-absorbing walls are installed; systemic control of the technical condition of the hand tools - pneumotampers, grinders; good warming of the rooms in which it is working in contact with local vibrations. Application of PPE: against noise - antiphoning; against vibratory impact - vibrating gloves and anti-vibration shoes. Appropriate work regimen - 10-15 min intervals of staff in quiet rooms as well as periodic change of work operations. The medical prophylaxis of noise and vibration effects is related to professional medical selection and periodic medical examinations; prophylactic physiotherapeutic procedures in vibrations - hydroprocesses, massage, UV-irradiation; protective nutrition - diet A.

Reduction of the air pollution in the foundries

with harmful vapors and gases is accomplished by creating maximum sealing and effective artificial mechanical ventilation; replacing used chemical compounds and materials with less toxic and dangerous - e.g. replacement of organic supporters with less toxic inorganic - water glass, phosphatic, ferro-phosphatic. Mixing, forming, metal discharging, dismoulding and cleaning to provide local aspiration. Given the high risk of acute poisoning with CO_2, medical prophylaxis includes: provision of artificial respiration equipment as well as resuscitation equipment and training of operators for their application in case of emergency rescue work; work on dangerous areas only in pairs of workers. Compliance with medical contraindications for work on releasing chemicals and periodic medical control.

To improve the **microclimate** in the foundries, a number of measures are applied. **Radical and sanitary-technical** include: sealing of convective heat sources; for protection against IR during casting, pouring and cooling of the castings, shielding, good thermal insulation of the heated aggregates and transport lines, rapid removal of the heated production outside the production premises, ensuring efficient local and general exchanging ventilation, remote control, etc.

More important hygienic and medical-prophylactic measures are: furnishing of rest rooms with a normal microclimate; appropriate working and rest regime; providing a suitable drinking regime; organizing protective nutrition - diet A; use of special overalls for heat protection; eye protection from IR rays - use of special filter glasses; preliminary professional selection, observing the contraindications for accepting work in high temperature conditions and IR radiation.

17.4.2. FORGE-PRESS PRODUCTION

In the forging workshops, coarse articles of metal castings coming from the foundry or rolling mill are made. The technological process provides for plastic deformation of metal billets with or without forging in hot or cold state, under the impact of hammering machines or with single impact (presses, rollers). Hammering is performed only on hot metal, while pressing can also be applied to cold metal. Forge-press operations can be fully mechanized, but often executed manually. The sequential course of the process is: preparation of the metal billets (eg precut of a certain size), preheating, forging or pressing, cleaning of the article and control.

The heating of the metal billets is carried out in heating furnaces (flame - burners, electric furnaces, MF-MW induction). The loading/unloading of the metal billets is manually (with a metal lever, pincers) or mechanized - with a wire rod, a crane, a thruster/pusher, a roller conveyor, etc. Forging or pressing is preceded by the cleaning of the anvil and the forging die of dust (cast iron-iron oxides) most often with compressed air. The billet is placed manually or mechanically. Pneumatic and mechanical hammers, hydraulic, friction and mechanical drive presses are used.

Working environment conditions.

Microclimate. Forging production refers to "warm" production, convection heat output of more than 23 W/m^3. During hot days and with ineffective ventilation, air temperatures in the working area are measured at 40-43 ° C but more often at 34-36 ° C. Important is the high intensity of infrared radiation. For large forging units the intensity is 3.35-20.93 kJ.cm^{-2}.min^{-1}, with an impact duration of 50-85% of the work shift. Relative humidity in these industries is low - 15-50%. The airflow rate varies depending on the size of the workshops, fluctuating in the summer from 0.4 to 1.0 m/s, and during the cold period (in case of poor closing of doors) it can reach up to 6 m/s.

Noise and vibration. During work of heavy forging pneumatic hammers, mainly low-frequency noise and general vibrations are generated. The measured sound pressure values are between 95 and 115 dB/A, reaching 125 dB/A. For different types of press, the noise is lower in intensity - between 83 and 104 dB/A. The noise of the forge-press equipment is predominantly impacted, impulsive, with high peak values. Aerodynamic noise is generated when the compressed air discharge valves are operated at hammers and presses. The general vibrations generated during hammer/press work are above the norms for frequencies above 8 Hz.

Dust. In the forging works, the incoming dust in the working area is in small quantities (below the MAC). This is a metal powder of "scale" (iron oxides). The largest amounts are released when cleaning the anvil (blowing with compressed air).

Chemical compounds. In forge- press production, air in the working environment is polluted by chemical compounds from the combustion of various types of fuel in flame heaters. Carbon monoxide, carbon dioxide, sulfur dioxide, possibly acrolein, smoke, soot and other products of thermal and thermo-oxidation destruction of the fuel are released. Concentrations are usually below the MAC, at the MAC level or 1.5-2 times the MAC.

Work process of the main professional groups. The main professions are a hammerman and a press operator. The work of both professional groups is similar. In the low-level mechanization of press in-

dustries, most operations are performed manually. The weight of the billets is from 1 kg to 50-100 kg. The hammerman is under the influence of general vibrations transformed in local by adherence to tool, which is above the norms. The execution of the basic operations in low-level workshops is also associated with a high static load on the musculoskeletal system. During service of heating furnaces, hammers, presses, workers are subjected simultaneously to high air temperature and radiant heat. It is possible to increase the body temperature, and the amount of the separated sweat during an 8-hour shift is between 1.5 and 5 l. Depending on the mechanization and the type of forging-press production, the energy consumption of the workers is between 12.56-25.12 kJ/min. The physical load is ranked at the limit of moderate to severe. Nervous-mental stress is also high due to continuous work with a high risk of mechanical injuries and burns.

General and occupational morbidity. The incapacity with temporary disability is very high. In its structure, with the highest proportion being colds of the respiratory system, diseases of the skeletal-muscular and peripheral nervous system, traumatism (most often burns), diseases of the gastrointestinal tract (enteritis) and of the cardiovascular system. Occupational diseases with high incidence are overload of diseases of skeletal-muscular system and peripheral nervous system (vegetative polyneuropathy), occupational hearing disabilities.

Prevention of work. These include: a rational architectural and building solution of the production buildings and the deployment of the equipment; choice of technological process, equipment and fuels with minimal detriment; mechanization and automation of production processes; organization of effective ventilation; choice of suitable PPE. For example, one-storey buildings with large area and cubic capacity, with one-sided and one-way installation of the equipment (avoiding machine saturation), are built for the protection against overheating microclimate in forge- presses production. Aeration of production facilities is organized. Replace hot pressing with cold. To reduce heat generation, rational heat protection is built by thermal insulation of the walls and openings of the furnaces. Apply water cooling of the casing and the shelters of the furnaces and water curtains in front of them. Local mechanical suction ventilation - umbrellas, above the furnaces is built. Air showering is carried out by stationary and freely movable facilities. To improve the microclimate in the crane cabinets, thermoinsulation protection of the floor, walls, as well as the placement of local air conditioners is provided. Of the hygienic-medical measures more important here are: providing a room for rest with an optimal microclimate; rational nutrition and drink regime; work and rest regime. Workers must be provided with means of personal protection. Adherence to medical contraindications for work in conditions of overheating, intense noise and vibration, and periodic medical control.

17.4.3. MECHANICAL MACHINING OF METALS

Mechanical machining of all kinds of mechanical machining of metal - turning, sharpening, milling, drilling, threading, grinding. Machine tools are used, which, depending on the operations performed, are mainly divided into "cutting blades" (lathes, mills, drills) and "abrasive" (grinding machines, polishing machines, sharpening machines). Nowadays, modern methods of cold processing - electrochemical, plasma technology, electro-erosion treatment are also used in mechanical engineering. The raw material (metal) used is metal preforms from foundry and forging presses or cut metal (metal blocks of metallurgy). The abrasive disks used in this manufacture most often consist of corundum (Al_2O_3) or carborundum (SiC), with a ceramic binder (including quartz sand) containing free crystalline SiO_2 to 2-3.5%. When working on both types of equipment, due to high speeds and mechanical friction, heat is intensively formed. This requires the use of lubricating coolant (LC) for cooling the cutting tool and the metal.

New processes of cold metal processing are applied less frequently and only for special productions. **The chemico-mechanical method** is based on the use of a solution of acids, hydrogen peroxide and triethanolamine (in chambers); **the electrochemical method** is based on the anodic dissolution in metal chambers of an electrolyte (sodium chloride solution and sodium nitrite solution); **the electro-erosion treatment** is based on the occurrence of electrical discharges between the electrode and the workpiece in a liquid dielectric medium (oil or a mixture of kerosene oil), wherein at the discharge point the temperature is up to 5000-10,000°C.

LCs are complex multi-component compositions consisting of various chemical compounds. They usually contain a basic ingredient - 60-90% (oils, polymers, surfactants, electrolytes) and additional additives (in low%) - emulsifiers, binders, anti-corrosion and antioxidants, antimicrobials, bacteriostats, Compositions (LC) are classified as: 1) oils; 2) emulsifying; 3) synthetics; 4) semi-synthetics; 5) aqueous solutions of electrolytes.

Additives include chemical compounds of various classes: sulfur, chlorine and phosphorus com-

pounds, amines, alcohols, inorganic salts.

With prolonged use of LCs in machines (often from 1-6 months), they are a good medium for the development of bacteria and fungi - oily aerosols damaged 50-100%.

Working environment conditions.

The microclimate in the mechanical workshops is most often within the limits of the norms and is not a hygienic problem. Typically, it has lower airflow speeds - to static. In the large modern workshops equipped with automatic lines, with metal-cutting machines with a program-numeric device with display, the rooms can be air-conditioned.

Processes of machining parts of different machine tools are associated with **noise** generation. Usually the noise is medium and high frequency. Some models of lathes with a digital programmer, drills, dental machines, screwdriver machines generate noise under norms or an excess of 1-2 dB/A. High-frequency noise with an intensity of 95-98 dB/A generates large grinding machines, up to 92 dB/A - dental machines with maximum sound power in the 250-2000 Hz range. The noise in the mechanical workshops is of continuous nature, with constant, all-shift impact.

No **dust**, but ships of different sizes are produced for cutting machines. Airbrush-type machines have been measured for powder aerosols ranging from several $mg.m^{-3}$ to 45-50 $mg.m^{-3}$. The powder is a finely dispersed aerosol with a mixed mineral-metallic composition (20-40% - metal, 60 to 80% - mineral). Contains in free quantities free crystalline SiO_2 - from 0.9% to 9.6%. The metallic component of the powder depends on the type of metal to be treated, most commonly iron or iron oxide, aluminum, copper, etc. Some chromium, nickel, titanium, beryllium are also released in the treatment of some alloyed steels with the metallic powder.

Chemical substances enter the working environment from finely dispersed LCs - steam-aerosol mixtures (95% of the aerosol is less than 5 μm). With prolonged use of LC, their starting composition may change due to metal particulate contamination (catalytic action), products of thermal oxidation destruction, evaporation of some components and partial microbiological decomposition. High toxic and dangerous products are formed, for example, In the operation of electro- impulse and electro-erosion machines. The released oily aerosols are in concentrations of 15 to 40 $mg.m^{-3}$, soot - up to 22 $mg.m^{-3}$, CO - up to 30 $mg.m^{-3}$, iron aerosol and iron oxides - up to 6 $mg.m^{-3}$, 3,4-BP - up to 17 $mg.m^{-3}$ (the latter is the result of heating the oil to thousands ˚C).

Depending on the chemical composition, the conditions of operation (storage), the metal treatment regime and the LC regeneration, the microclimate characteristics, the number of **microorganisms** in the LC can reach significant levels - for example, in oil - 10^2-10^5 in ml, emulsion - 10^4-10^8 in ml, synthetic - 10^4-10^7 in ml, the number of pathogenic microbes moving from 10 to 10^3 in ml. Without inclusion in the composition of the LC of bactericidal additives, the number of saprophytes increases greatly. Trichosporon, Graphium, Fuzarium, Aspergillus are defined as microscopic fungi.

Labour process of the main professions. The main professions in the mechanical workshops are turner, milling, grinding, hobel machines operators, operators of machines with program-numerical control and others. Their work is similar, they work under almost the same labour conditions. Manual machine servicing operations are light and medium in physical loading but with significant psycho-sensory stress. When servicing the machines, the work is connected with lifting and transporting small weights. Large parts and articles are placed on machines or mechanically carried - with cranes, hoists, manipulators, electric trucks. Modern metal cutting machines in the mass production of the same type of parts are semi-automatic and automatic machines, aggregate machines with full or almost complete mechanization of operations, including the machined part moving with automatic start and stopping of the machines. Operator functions are limited to visual control on the display or on the control panel. The commitment of the main professional groups for servicing the machine tools is significant - from 60 to 82% of the working time. When servicing the universal hand held lathes, it is even greater - 98%. The working posture is standing, very often compulsion. Machine controls (buttons, levers, pedals) are often non-ergonomics. The work done requires constant high attention activity and good memory, the ability to concentrate. It is associated with a very heavy workload of the visual analyzer, especially in the case of hand-held machines. Very good coordination of the movements, high qualification and quick reactions are needed.

General and occupational morbidity. The persistence of the upper respiratory tract of the finedisperse vapor-gas-aerosol mixture of LC (mostly irritant additives, bacterial aerosols and thermooxidative destruction products) disturbs the body's protective barriers. This creates conditions for the development of infectious processes in the **respiratory system**. In the structure of the morbidity with a high proportion of them, there are ACUAP, acute and chronic bronchitis, pneumonia. High frequency are spastic bronchitis, allergic rhinitis and sinusitis. They can also be directly associated with sensitization

caused by the direct action of bacterial and chemical allergens from the LC, as well as by the chromium, nickel, and cobalt contained in the powder aerosol.

Based on the resorption effect of certain components of LC (saturated and unsaturated hydrocarbons, amino-nitro compounds, etc.), **vegetative-vascular type reactions** (asthenic syndrome, angiospasms, hypertensive reactions) have been reported in the workers.

However, the most commonly reported diseases in workers are **cutaneous** - from the effects of LC on the skin, especially mineral oils and emulsions. The chemical constituents of LC with local irritant action and contact allergens (chlorine-containing compounds, surfactants, ethanolamine, phenylenediamines, benzylamines, aldehydes, chromium, nickel, etc.) further damage the skin and cause irritative and allergic dermatitis. Also important for the development of skin diseases is microtractility (from chips) at work. Bacterial allergens are referred to as a major link for the development of hyperkeratosis, oil papillomas, chronic pigmentation of the skin. Microscopic funguses from the LC also play a role in allergic reactions. The most commonly diagnosed skin diseases in workers are: oil folliculitis and "oil acne"; allergic dermatitis; photodermatists; hyperkeratoses; papillomas (precanceroses); permanent pigmentation; dermatitis and onychomycosis.

Production traumatism - mostly ocular - and hand wrists (finger injuries) from chips, cutting tools, etc. is also a great honor.

Potentially for workers there is a **carcinogenic risk**. This is predominantly in the case of skin contact and inhalation of LC containing higher viscosity oils; oils with more heavy aromatic hydrocarbons (3,4-BP); combined use of nitrites, nitrates, aliphatic and aromatic amines in LC (formation of N-nitrosoamines); containing chromium, nickel, cobalt.

Prevention of work.

Sanitary- technical and technological means: complete isolation/shielding of the working part of the equipment and provision of local aspiration for removal of the separable vapor-gas-aerosol mixture of LC. Securing a block (shutdown of the machine and LC delivery) when lifting the hiding facilities; Installation of chip refractories and chip-shifters, fences, shields, screens. Mechanized collection and removal of chips; Introducing electropark or electro-impulse sharpening of the cutting tool to avoid eye trauma; equipment of mechanical workshops with modern automatic assemblies.

Sanitary-hygiene: replacement of highly toxic and dangerous LCs and additives with less toxic and dangerous for health; hygienic standardization of the LC based on the toxicological characteristics of the individual ingredients; sanitary- technical instructions for the safe storage, transport and preparation of LC working solutions as well as instructions for their use, cleaning and regeneration.

Personal hygiene and PPE: use of special work clothing, which prevents the penetration of the LC into the skin; regular washing and processing of working clothes; bathing after work or after wetting; use of special detergents and protective creams; safety glasses when working on non-insulated and non-screened machines; respirators and semi-masks for abrasive machine operators. **To reduce the noise impact:** machine denoising; sound insulation; sound absorbing screens and barriers; technical maintenance; use of anti-phonics; work and rest regime. **To reduce the weight and tension of labour:** machinery mechanization; ergonomic deployment of machinery controls and visual inspection; rooms for short-term rest; natural, general and local artificial lighting, according to norms.

17.4.4. ASSEMBLY PRODUCTION

In the assembly works, the main technological process is to collect, connect, assemble individual parts and details until a finished product (machine, aggregate, etc.) is obtained. Auxiliary (finishing) operations are: coating of metallic coatings, paints, varnishes, lubricants for anti-corrosion protection and good aesthetic appearance of the product. The last step is a trial operation and a suitable packaging of the product.

Assembly in serial production is most often done by part flow (conveyor) methods. The assembly of the individual units and the final assembly (fitting, joining, assembling) is done by hand tools - drills, screws, hydraulic presses for bending, welding machines, etc. For the transport of heavier and bigger parts is used mechanized intra-shop transport - chain conveyors, transporters, telphers, bridge cranes, electric trucks and others.

Specific technological processes in the assembly production, which carry a higher professional risk are: welding, metal coating and painting of the products.

Welding. In the most frequently used thermal methods it is done by joining the metal parts by means of localized heating and melting. There are other more modern methods - the so-called "cold welding" (eg. ultrasonic welding). The thermal welding methods include: arc, gas, electron beam, laser and plasma welding. Thermo-mechanical: electrical contact (spot) welding, high-frequency welding (contact and induction), vacuum diffusion welding, can also be referred to these methods. The most important are arc, gas and contact welding.

The electric arc welding uses the volt arc (6000 °C). It works with special devices (manually, maybe robots) powered by direct or alternating current. Melting (steel) and non-melting (tungsten) electrodes are used. For the quality of the welds, coated melting electrodes are used. It contains substances necessary for the sustainable combustion of the arc to create gas and slag protection of the metal from the air and also substances with alloying function to improve the quality of the molten metal. The coating composition includes: alloying agents - ferroalloys (ferro-manganese, ferrochromium, ferrosilicon, ferrotitanium); gaseous materials (marble, magnesite, etc.); slag-forming materials (fluorine compounds, carbonates); arc stabilizers (silicates, quartz, spat, nefelin). In order to isolate the arc and molten metal from the air, gas protection is provided - supplying inert gas (argon, nitrogen, helium, carbon dioxide) to the welding zone.

In **contact point welding**, the parts are joined in separate points by pressing against the electrodes (non-melting). Depending on the type of welded metals, the contact portion of the electrical current becomes plastic or melts and, as a result of the compression, the metals are joined together.

Oxygen or air and gaseous fuel (most commonly acetylene, but also hydrogen, propane, a propane-butane mixture) are used for **gas welding**. They are fed into the welding torch and mixed before they are ignited in the nozzle. The high temperature (above 450 ° C) melts the metal surfaces at the points where they are to be joined. When using a weld metal rod (a lower melting point metal), the bonded metals only heat (do not melt).

Metal cover. The most commonly applied protective metal coatings are nickel, chromium, zinc, copper, cadmium. The following metal-coating methods are used with great industrial application: galvanoplasty, metallisation, dipping of metal products in a molten metal bath, industrial tinning. The electroplating method is most often used. On it, a thin layer of a protective metal is deposited on the surface of the article, emitting an electrolyte solution under the influence of a constant electric current. This process is carried out in special galvanic baths filled with aqueous solutions of acid salts (nickel sulphate, copper sulphate, zinc sulphate) or complex alcaline salts (cyanic compounds) of zinc, copper, cadmium, aluminum, and silver. In advance the surface of the product is cleaned mechanically, chemically and chemico- mechanically. Crushing jet purification, grinding, polishing, ultrasonic scouring, etching using strong inorganic acids (hydrochloric, nitric, sulfuric) with subsequent neutralization are used. They are chemically degreased with organic solvents (gasoline, trichlorethylene), alkaline solutions, detergents.

In case of electrolysis, a breakdown of the process mode (increase in voltage, current strength, electrolyte concentration and temperature) can cause a violent release of hydrogen (gas bubbles), entraining part of the electrolyte solution and products formed in result of chemical reactions in the bath. It is also possible to remove hydrogen cyanide by mixing alkaline electrolyte with cyanide compounds and strong acids.

Painting and varnishing. It is carried out in a certain sequence, including: **surface preparation** - mechanical cleaning (from roughs, rust, oxides) with hand or mechanized tools (metal brushes, grinder, crushing jet, etc.) and chemical cleaning (degreasing with alkaline solutions, organic solvents, detergents or staining with inorganic acid solutions); **priming** - casting of lead or zinc-containing primers dissolved in organic solvents (hand-brushed, jet or pneumatic in chambers); **filling** - smoothing the surfaces (filling the roughness with nitrocellulose or oil kits); **grinding** - mechanical or manual; **painting/varnishing** - painting and varnishing, most often pneumatically by spraying with extruders (in 70%) or electrostatically; **drying** in hot air circulation chambers, induction or infrared dryers.

Working environment conditions. In the course of assembly, they are specific for each stage. When **connecting, assembly** of the parts, the leading production factor is the noise-vibrating (using manual instrumentation, intra-department transportation). The impact is periodic. The noise is high frequency with the highest intensity when working with a nut-runner and drilling of parts (95-100 dB/A), in manual grinding - (88 dB/A). When grinding, drilling is about 80 dB/A, and when moving conveyors - between 80-82 dB/A. In handheld instruments, local vibrations exceed the norms in the range of 32-250 Hz, with peak values at 32 and 63 Hz. The working in welding, the metal coating and the paint and varnish conditions are disadvantageous and dangerous. In all three operations, labour-hygiene problems are mainly the result of airborne intake of metallic aerosols and gases and vapors of chemical compounds. In **the arc welding**, the weldable aerosol (condensed metal vapor) released is from the electrode coatings and from the molten metal of the welded surfaces. It is highly dispersed - up to 98% of the particles have a diameter less than 1 μm (0.01-0.6). Contains iron oxides and impurities - manganese compounds, hexavalent chromium, nickel, vanadium, molybdenum and others. The aerosol concentrations in the range of 5 to 12 $mg.m^{-3}$ have been measured in open-air welding, and in

Tab. 1. Characterization of the chemical factor of the working environment in arc welding

Type	Content of the compounds	
	emission products	concentration in mg.m $^{-3}$
External * arc welding	carbon dioxide	2000-6000
	carbon monoxide	10-40
	nitrous oxide	2.5-4.0
	nitrogen dioxide	0.5-3.0
	manganese aerosols	0.1-0.2
	iron aerosols	1.2-2.2
	lead aerosols	0.02-0.2
External ** welding electrodes "Vejen"	carbon dioxide	6000-8000
	carbon monoxide	3-5.0
	nitrous oxide	0.5-1.5
	nitrogen dioxide	0.3-0.5
	manganese aerosols	0.3-0.8
	iron aerosols	3.0-12.0
Indoors ** welding in boiler	carbon dioxide	46000-55000
	carbon monoxide	120-180.0
	nitrous oxide	12-16.0
	nitrogen dioxide	3-6.0
	manganese aerosols	0.6-0.7
	iron aerosols	0.2-1.2
	lead aerosols	0.05-0.06

* By D. Tsvetkov, 1990 - CT 72a of the Center for Hygiene
** By Tsvetkov D., 1990 - CT 143of the Center of Hygiene

closed and semi-closed spaces ranging from 100 to 250 mg.m^{-3}. Extreme hazards and toxicities are the vapors and gases generated by thermal decomposition of materials (fluorine compounds) or as a result of the photochemical action of ultraviolet radiation on air gases (carbon monoxide, nitrous pentoxide, nitrogen dioxide, nitrogen oxide, ozone) . Concentrations of these materials are by external welding around the MAC or up to 1.5 times the MAC, whereas in case of semiautomatic welding, the excess is repeated several times - up to ten times **(Table 1)**. In the welding works there is a combined effect of chemical exposure and the effects of radiant energy - ultraviolet, infrared, as well as rays from the visible area. The intensity of the infrared radiation varies according to different technical parameters and the mass of the heated metal and varies within the range of 100 to 2450 W.m^2. For ultraviolet radiation a total spectral density is given at a distance of 1 m from the welding area - 0.4-162 W.m^2.

The working conditions of the **gas welding** are similar to those of the electric arc welding. Here, however, there is less intensity of radiant energy, and the air pollution with carbon monoxide and nitrogens is more pronounced. Due to the production of acetylene from carbide, which is sometimes contaminated with phosphorus and arsenic, the high toxical gases phosphorus - and arsenic hydrogen can form.

Depending on the stage of the **metal-coating** process in the air of the working environment, various chemical compounds are introduced - mainly aerosols of hydrochloric, nitric, sulfuric, phosphoric acid, sodium, chromium, nickel, cyanide, etc. **(Table 2)**.

In **lacquer-painting work,** the preparation of the metal surface - the mechanical cleaning, is associated with the removal of dust (roughly and fine-grained) and rust removal - iron oxides, and in case of dry grinding after priming and plating - lead compounds. Fine grinding also produces iron oxide, however, in insignificant concentrations - 0.25-0.7 mg.m^{-3}, while in crude grinding - the concentrations are high - 8.0-9.7 - 13.1 mg.m^{-3}. Lead concentrations - from 0.02 to 0.45 mg.m^{-3} are reported for pneumatic application of lead-based primers. In the case of spray painting solutions in an aspiration chamber, organic solvents were found in concentrations of around the MAC or slightly exceeded: amylacetate (190-215 mg.m^{-3}), acetone (200-250 mg.m^{-3}), turpentine (180-300 mg.m^{-3}), gasoline (75-150 mg.m^{-3}), butanol (600 mg.m^{-3}), toluene (83-275 mg.m^{-3}), styrene (12-15 mg.m^{-3}). Larger concentrations are measured when drying dyed articles and ineffective ventilation.

Labour process in the main professions. The main professions in the assembly workshops are: fitters, locksmiths, welders, spraying-painters, galvanotechnics. In the labour characteristic of all the groups mentioned, a large part has manual operations performed with different, most often pneumatic tools. Fitters collect, assemble, or fit individual

Tab. 2 *. Characterization of the chemical factor of the working environment with metal coating

Stage	Content of compounds	
	released substances	concentration in mg.m^{-3}
Preparation of metal surface chemical cleaning	chlorine hydrochloric acid sulfuric acid sulfur trioxide sulfur dioxide nitric acid nitrogen oxides	0.5-1.0 0.3-0.5; 5-10.0 3.0-3.8 4.0-7.0 10.0-15.0 2.0-3.0 2.0-3.0
Degreasing: - Org. solvents - a mixture of sodium hydroxide, sodium carbonate and sodium phosphate	gasoline hydrocarbons sodium hydroxide phosphates sodium fluoride	200.0-600.0 22-32.0; 4-6.0 traces – 0.25; 0.1-0.5; 1-1.5 0.7-3.0
Coating with gold	cyanides	0.01-0.02
Chroming	chromic anhydride	0.03-0.09; 0.07-0.15
Zink plating	zink	10.0-15.0

* By K. Kochemidova, 1990

parts and details, make the necessary holes, screw, rivet, reoiled, perform technical tests. All these operations are performed in a sitting or standing position, but often also in a forced position. Occasionally, weighing up to 50 and over 50 kg is required. The physical load of the fitters is characterized as medium-heavy, the work is free-rhythm. However, conveyor assembly is another widely used method, characterized by: rhythm, fragmentation and uniformity of operations, with repeated repetitions leading to motor and psycho-sensory monotony. The work of the fitters requires high attention, good visual memory, spatial orientation, responsibility. Occupational risk is related to the possibility of accidents at work. The work of **electric welders** is characterized by standing and very often with a forced working position requiring a static position of the arms and shoulder girdle. Often these operations are for 40-80% of the shift. Work is also associated with physical load - lifting/moving parts (characterized as medium heavy). The visual tension is significant. High attention, good memory, accuracy, high responsibility is required. It is associated with motor monotony with free rhythm of labour. Occupational risk is also determined by the possibility of electrotrauma. **Galvanotechnics** in modern metal coating departments, with complete mechanization and automation lines, perform the function of operators with predominantly nerve-sensing and nerve-mental load. In the old galvanic units the work is connected with many manual operations. The work is done in a standing position, in a free rhythm and is connected with continuous movement. When loading/unloading the parts, the work is also associated with a physical load - manual carrying a load up to 25-50 kg (medium weight). Both technologies require very good qualification and memory, high attention, moderate visual tension. Occupational risk is determined by the possibility of acute intoxications - in accidents (eg. with cyanides), by remote effects using electrolytes with an allergenic and carcinogenic effect (chromium, nickel). Electrotraumas are also possible. The work of the **extruder-painter** depends on the type of technology used, the degree of mechanization and the automation of the process. In non-mechanized dyeing processes, manual operations are in the course of the entire production cycle. The working posture is standing, in some cases forced, often with long static positions of the body. Production operations are associated with a significant load on small arm muscles (multiple repetitive monotonous movements with a small volume) and static shoulder girdle load. The work is related to the physical load in carrying materials and details and is of medium weight. Nervous-sensory stress is moderate - constant attention is required, significant visual analyzer tension, accuracy, good qualification. Respiratory and skin contact with organic solvents determines the high professional risk of acute and chronic intoxications due to spraying of lacquer-painter solutions.

General and occupational morbidity. High-frequency at **fitters** are the diseases of overt6ension of the locomotory system, of the PNS, which dominate the structure of occupational morbidity. Less frequent cases of vibrational disease with local vibrations are reported. In **electrical welders**, the structure of occupational morbidity is mainly formed by chronic respiratory diseases (pneumoconiosis of the type of siderosis or sidero-silicosis and chronic

bronchitis), from overtension of the locomotory system, PNS, cases of chronic manganese intoxication (manganoconiosis). Prolonged exposure to infrared and ultraviolet radiation may lead to cataract development. Typical acute occupational disease is photoophthalmia (ophthalmia electrica). UV-burns of unprotected skin have also been observed. The acute professional diseases is also related to metal fever when welding articles containing copper, zinc, and magnesium. In case of gas welding, acute and chronic poisoning with nitrogen oxides, phosphine and arsine (in the preparation of acetylene) is possible. High frequency in **galvano-technics** includes diseases of the irritant effect on mucous membranes and the skin of excreted vapors from acids and bases: upper respiratory catarrh (acute and chronic) - rhinitis, pharyngitis, laryngitis, bronchitis; conjunctivitis, tooth enamel destruction. Contact with chromium salts, especially chromium trioxide, also shows significant damage to the UAP and especially to the nose mucosa (to necrosis of the nasal mucosa, ulcers, perforation of the cartilage of the nasal septum - currently the pathology is rarely found). Chronic bronchitis, chronic pneumonia, asthmatic bronchitis are common here. Skin diseases (eczema, dermatitis) are also a major frequency of the action of chromium, nickel, dicyanoacetate and contact with organic solvents. By labour traumatism with high frequency is chemical burns with acids and bases. In case of accidents and ineffective ventilation acute intoxications with the used electrolytes and organic solvents are possible, and in galvanization - metal fever.

In **spayer-painters** the cases of high-frequency there are diseases of over tension of the LS, of the PNS. Less commonly, acute and chronic intoxications with lead (lead minium), diluents and organic solvents (inhalation and skin contact) occur. The contact with dust (metallic, polymeric) emitted by mechanical cleaning, dry grinding, polishing is the cause of chronic bronchitis. In the structure of occupational morbidity, the diseases of the skin (dermatitis, eczema) are also a result of contact with diluents and organic solvents.

Prevention of work. The healing measures for the installation itself concern the use of pneumatic tools and the conveyor working method. It is recommended to use: nut-drivers whose noise parameters do not exceed the norms; technical control and regular technical support; using tools equipped with vibration absorbing elements at the handle. In the conveyor assembly, to avoid monotony, it is required: to combined operations - to be able to change and to manipulate several operations per day; considering the speed of movement of the conveyor belt with the capabilities of the average worker; changing the belt speed according to the change of capacity; providing ergonomic working furniture; work and rest regime, etc.

Arc welding: implementation of automatic welding processes (robots); standardization of welding materials - reduced to the content of toxic components in the coating of electrodes; welding under local aspiration protection with a capture efficiency of not less than 90% for stationary devices and 75% for mobile; stationary work places be equipped with screens to protect against radiant energy; for stationary welding of small and medium parts, with the use of protective gases to provide aspiration cabins; to reduce the reflection of UV radiation, to paint the walls of the rooms with zinc oxide include paint; use of PPE - protective masks with filters or hose gas masks with air supply at welding in closed or semi-closed compartments; use of safety shields, helmets and glasses with protective light filter lenses; provision of special workwear, gloves and shoes to avoid burns and electrotraumas; provision of onboard, inflow-suction, local ventilation for the removal of released chemicals and additional flow of general exchange ventilation for galvanic baths; use in galvanic bath compartments and chemical cleaning of acid resistant floor coverings and wall linings; replacement of organic solvents as degreasers with synthetic detergents; Improvement of the technology with exclusion and replacement of highly toxic metal salts in the composition of electrolytes with less toxic; creation of automated flow lines with remote control; providing special workwear - rubber boots, aprons and gloves, glasses and filtering gas masks. Using protective and greased skin creams.

Lacquer-painting work: standardization of the composition of varnishes and paints; exclusion of toxic components (chlorinated hydrocarbons, benzene, methanol, etc.); replacing the tin lead with iron; replacing organic solvents based on aromatic hydrocarbons with less toxic and dangerous; replacement of organic solvents as degreasers with synthetic detergents; performing pneumatic (spray) painting in pressurized chambers with powerful local suction and general inflow-suction ventilation; providing continuous mechanized and automated flow lines for cleaning, degreasing, washing and drying the details; use of modern methods of applying the painting solutions (electrostatic painting, airless painting method, etc.); in the case of pneumatic painting, provision of effective insulating PPE, with mechanical injection of fresh air, special work clothes, gloves, a panoramic glass mask or hermetic glasses; protective and greasy skin creams.

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N. Gincheva

17.5 CHEMICAL INDUSTRY

The production of chemical industry can be incorporated into three major groups: **products of basic inorganic and organic synthesis; semi-finished products** derived from basic chemical products; **finished** chemical products obtained from the processing of semi-products. This production is obtained in the main and some other specific industries of the chemical industry **(Table 1)**.

Despite the wide variety of chemical industries, **technological processes** have similar operations (stages) performed with relatively uniform equipment. The main operations include: **Preparatory** - milling, sieving, separation, dosing, mixing and transport of solid starting materials and pumping, vacuum piping and filling of the reaction vessels with liquid components; **Flowing of basic chemical processes** - oxidation, reduction, chlorination, sulphurisation, nitration, amination, electrochemical processes, etc.; **Separation of chemical compounds** - distillation, filtration, centrifugation, extraction, crystallization, rectification, etc.; **Final operations** - drying, milling, prepacking and packaging (in bags/sacks and filling of drums, tanks, etc.), storage; **Additional operations** - making of technological samples, replacement of catalysts, cleaning of filters, planned and emergency repair works, etc.

The most commonly used **equipment** is: disintegrators, mills, crushers, screens, vibrators, separators, dryers, towers, reactors, furnaces, receivers, heat exchangers, baths, centrifuges, filters, crystallizers, packing and filling aggregates; transport of powdered ingredients - augers, elevators and liquid ones - with pumps, compressors and others.

Depending on the type of process, the required equipment can be installed in a closed building (window, windowless) and can also be installed outdoors on open ground (atmospheric installations) on the roof of one-story buildings. Large-clearance appliances - columns, receivers, heat exchangers, reactors, etc., apparatuses operating at normal and high pressure, under vacuum, high and low temperatures, and more are usually installed outside the buildings. Outdoor equipment is linked to a remote control and control system from the control room (periodic monitoring of the separate apparatus by the servicing staff). When highly toxic chemicals are used in the process, the hazardous steps are taken in separate isolated production facilities.

There are three main types of technological pro-

Tab. 1. Chemical industry - production and products

PRODUCTION of basic chemical industries	**PRODUCTION of other chemical industries**
1) Main products of inorganic synthesis - acids, bases, salts, gases	1) Production of oil refining
2) Main products of organic synthesis - raw materials for the preparation of plastics, synthetic resins, fibers, rubbers, alcohols, solvents and raw materials for detergents, dyes and pigments	2) Products of petroleum and coal - bitumen, asphalt, lubricants and additives, synthetic oils
3) Fertilizers and pesticides	3) Car tires and other rubber technical goods, incl. shoes
4) Plastics, synthetic resins, synthetic rubber, cellulose and synthetic fibers	4) Products from plastic
5) Pharmaceutical	5) Chem. substances used as additives, emulsifiers, stabilizers, antioxidants, flavorings, colorings, preservatives in food products, softeners and sterilizing agents for water
6) Paints, lacquers, enamels	6) Chem. substances - corrosion inhibitors, for the treatment of paper, wood, leather, fire-extinguishing agents and the like.
7) Soaps, detergents, cosmetics, perfumes	
8) Various chemical products - explosives, adhesives, inks, photographic films and photopreparations	

cesses in chemical production: periodic; continuous; closed cycle production (waste-free technological process). In the **periodic process** there is no complete mechanization and automation of production. Many of the production operations are manual and labour-intensive - charging and emptying the apparatus with liquid or powdered materials (raw materials, reaction mass, intermediate and end products), introducing catalysts, cleaning appliances. The frequent opening of the equipment creates an opportunity for chemicals to enter the air, besides inhalation and skin contact of workers with chemical damages. With the periodicity of technological processes characterize the production of pharmaceutical preparations, of plastics products in low-production industries.

The continuous production process and the newer **"closed-cycle-nowaste technology"** are beneficial in terms of their technological and hygienic characteristics. They allow for mechanization and automation of production, with remote control and control, which reduces residence in the working environment (only periodic control). In addition, maintaining the persistence of the process parameters (temperature, pressure) preserves the hermetic of the apparatus and reduces the risk of chemicals being released. Continuous process and non-waste technologies are used to deal with highly toxic and dangerous chemicals - ex. production of phosphorus compounds, isocyanates. Besides the mentioned types of technological processes of hygienic importance are their characteristics at the level of the maintained **temperature (low and high temperature), pressure (high, low pressure, in vacuum), the inclusion of catalysts**, etc. At high pressure, there is a risk of chemical compounds being released from leaks, accidents, explosions and fire. Catalytic processes also carry an increased risk of manual production operations - opening of the chemical equipment for charging/removing the catalyst. Modern and hygienic conformation are chemical processes, running at low pressure or in vacuum, at low temperature, automated (continuous) production.

Working environment conditions.

A leading unfavorable production factor is the **chemical** one. Air pollution is also in the production premises and the open-air factory. The surface of the equipment, the vehicles, the ventilation equipment, the walls, the floor covering, the clothes and exposed parts of the workers' skin are also polluted. The separation of chemicals depends on the leakage of the equipment and communications - emergency or when performing certain manual operations - eg. concentrations up to tens of times above the MAC are recorded under planned repairs in the production of acids and bases (inorganic synthesis); In organic synthesis - when cleaning the evaporator tubes of a reaction mixture (production of methylmercaptopropionic aldehyde); in dismountling and cleaning of filters (manufacture of PAN-fibers).

In most chemical processes, depending on the operations performed, even in the same room, changes in concentrations are observed over time in the work shift. This type of impact also favors the absence of permanent workplaces, the movement of service personnel at different levels of equipment. Different concentrations of chemical pollutants are also recorded in the different shifts in processes with mismatch of the shift cycle with the technological one - ex. emptying and cleaning of the reactors, cleaning of the filters takes place only in a fixed shift. Relatively constant content of chemicals is observed in the production processes with a stable technological process, uniform equipment and permanent work places - ex. plastics processing.

The chemical factor is multi-component and with a different aggregate state. Most commonly, in the air of the working environment, a vapor-gas-aerosol mixture, comprising starting, intermediate and end products, as well as substances formed by interactions within the mixture or the result of thermal or thermooxidation destruction, is introduced. The chemical composition of the mixtures may remain relatively constant and may change. In the air of the working environment, besides chemical substances with general-toxic action, substances with remote effects can also be introduced - mutagens, carcinogens, teratogens, etc. (e.g., paints for dyes, tar, soot, PEH, chloroprene synth. rubber, etc.). Depending on the density of the vapors and the specific weight of the individual chemicals, they are unevenly distributed in the space.

The air in the working environment is polluted with **toxic dust**. It is most often used in the initial and final stages of the technological process - preparatory and final operations (raw material processing and drying, prepacking). Concentrations are determined tens of times in excess of the MAC. Dust dispersion is usually high - particles with dimensions less than 5 μm predominate. The possibility of adsorption of gases and vapours on the dust aerosols, which alters the toxicity, is of hygienic importance.

Physical factors, expressed and common to all chemical industries, are the unfavorable weather conditions and the noise-vibration factor. **The microclimate** in high temperature process or exothermic processes (lime, sulfuric acid, carbide, phosphorus, etc.) is characterized by air temperatures above 30°C (reaching 35-36°C during the warm period). Some manufacturing operations - filtering, centri-

fuging, precipitation, flushing, etc., are associated with significant emission of water vapours and increased room humidity. In open installations, the thermal state is determined by the conditions typical of the geographical area.

The noise-vibration factor is expressed in almost all workplaces, and in the operating rooms. The main sources are the technological equipment - mills, disintegrators, crushers, elevators, tumblers, vibrators, centrifuges, compressors, pumps. Generated mechanical and shock noise has a high intensity of up to 100 dB/A norms. Fluid and aerodynamic noise, exceeding 4 to 10 dB/A, is generated in the flow of liquids and gases in high-speed pipelines, operation of furnace burners and of coolers.

Labour process in the main professions. From the technology staff, the main occupation is an **apparatus-worker** (operator) having controlling functions. The workplace is in control rooms, next to the control and operating panels. **An assistant - operator**, in addition to control functions of control measuring instruments (MI), is involved about 60-80% of the time of active working time of the chemical apparatuses.

In the major process chemical industries with continuous process, the main activity of the operator is continuous monitoring of the CMI, which requires rapid visual perception, understanding and processing of the information. The work is with a slight physical load but with very high nervous-sensory and nervous-mental tension. In a stable technological process and the operation of the equipment, their work is also markedly monotonous. High qualification is required. The work of the assistant apparatuses is more physical (medium weight work). The working posture is predominantly standing and forced, less sitting. The work is also associated with nervous-sensory tension with high responsibility. Rapid reactions, good qualification and coordination of movements are required. They worked under unfavorable weather conditions (outdoors, overheating), in environments with significant chemical pollution and noise-vibration effects.

General and occupational morbidity. Leading groups in the structure of occupational morbidity in chemical industry workers are: diseases of the musculoskeletal system by over tension; occupational pulmonary diseases; allergic and skin-allergic diseases; hearing impairment. The relative part of diseases from chemical damages is low due to the mechanization and automation of technological processes. Acute and chronic intoxications are rare - acute intoxications occur mainly in emergency situations and in repairs. The most frequent are the uncharacteristic forms of chronic intoxications in the influence of low concentrations of compounds with non-specific symptoms. However, there is also a symptom that may be associated with damage to certain organs and systems (critical). Liver symptoms, for example, are detected by the use of aliphatic halogenated hydrocarbons (mono- and dichloromethane, carbon tetrachloride, vinyl chloride, etc.), aromatic halogenated hydrocarbons (chlorinated naphthalenes), pesticides, and others. Symptoms from the kidneys occur with the effects of cadmium, mercury, lead, arsenic, bismuth, carbon tetrachloride, ethylene glycol, acetic acid; haematological abnormalities are observed when exposure to benzene and benzene derivatives, chlor-organic pesticides, exposure to arsenic, arsine, lead, copper, aromatic amino and nitro compounds; neurasthenic syndrome and psychotic syndromes occur when aliphatic and aromatic hydrocarbons and their derivatives (acetone, benzene, styrene, phenol, vinyl chloride, etc.), carbon monoxide and the like are present. Several chemical noxiousnesses cause the development of certain reactions by the CVS - hypotonic and hypertonic reactions, IHD. In the production and processing of certain plastics (polystyrene) and synthetic fibers (polyamide - capron), female workers found abnormalities in ovarian function and in the course of pregnancy and childbirth. At the highest frequency these deviations are in the production of polystyrene and polyamide fibers - caprons articles. The chemical industry with high professional risk is connected with chemical carcinogens - arsenic and compounds, benzene, benzidine, vinyl chloride, chloromethyl, cyclic amines and others.

Prevention of work. Concerning the leading unfavourable factor - chemical, prophylactic measures include: choice of closed-cycle processes and use of waste-free technologies; use of low pressure or vacuum process processes; replacement of dry methods for the processing of solid or powdered starting materials with wet; excluding or limiting the content of highly toxic harmful substances in the raw materials and finished products; hygienic standardization (certificate) of the raw materials and the final product; appropriate planning of the territory and production facilities according to the technological process and maximum isolation of the dangerous processes; provision of artificial inflow-suction (general) mechanical ventilation. Mounting of local aspirators. Equipment lock when shutting down the aspiration. Emergency ventilation device - general, with multiplicity of 8-10; instruction for performing repairs and emergency situations; Use of PPE to protect respiratory organs (helmets, respirators, insulating apparatus, etc.), to protect eyes, exposed areas of the skin, hand protection (eyeglasses, shields, gloves, boots

and special workwear); provision of protective and professional nutrition depending on the chemical substances used; preliminary and periodic medical examinations; organizing a gas-life-gard service for a first-aid and taking urgent measures.

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N. Stamova

17.6 TEXTILE INDUSTRY. MANUFACTURE OF COTTON FABRICS

According to the type of processed raw materials (cotton, wool, silk, flax, etc.) and the type of fabrics obtained, textile production has several branches. Here, the issues of labour hygiene in one of the main proceedings - cotton textiles, due to its versatility, will be addressed, pointing out the differences typical of other branches.

The two main production stages in textile production have been adopted - spinning and weaving are combined as "mechanical treatment of textile fibers". The other stages - bleaching, painting, finishing and final processing of fabrics make up the "mechanic-chemical".

Technology of fiber machining

The "**spinning"** stage consists of the following technological processes:

- breaking and knocking of cotton in batter sections, by sorting-batter aggregates;
- dragging and scraping the cotton of the cotton-combining machines machines and shaping it in the form of a fluffy tape;
- drawing the fluffy tape into thinner strips by means of cross drifts;
- extra retraction and initial tightening of the cotton strip by flyer machines;
- final pulling and tightening of the cotton strip of spinning looms (rings).

The yarn produced is wound on spools which are referred to the next weaving stage of the same establishment or are dispatched to other weaving industries.

The **"weaving"** stage includes the technological processes:

- rewinding the yarn by means of winding machines;
- warping, i.e. composition the base and winding it onto a special shaft;
- sizing the base in sizing baths with a solution of starch, soaps, fats and antiseptics, drying in a drying drum and rewinding on a special drum;
- threading each thread from the base into the corresponding remise hole and the corresponding reed plate;
- preparation of weld by rewinding the yarn on special spools;
- attaching the shaft to the base on the weaving loom, loading the shuttle with a weld and putting the loom into operation.

The finished cloth is removed from the loom and transferred to a special compartment for grading, removing some defects, or scrapping defective sections and sizing. It is then sent for mechano-chemical processing.

The mechano-chemical treatment of woven fabrics is a more complex and varied technology. It takes place in the so-called **finishing workshops** - wet and dry. It consists of the following technological processes:

- sewing the finished fabrics to form a cloth tape with a length of 2000-4000 m;
- singeing the cloth tape in singeing machines (heavy metal surfaces are heated) to remove the fibers;
- desizing (by soaking in bathtubs with water and starch removal preparations applied to the substrate before weaving);
- boiling of textile in hot sodium hydroxide solutions;
- bleaching with chlorine preparations and subsequent washing of the fabric with slightly acidified water (boiling and bleaching can be carried out as a continuous process in closed aggregates);
- painting and stamping of fabrics; The painting is carried out in different containers and aggregates (in hot or cold solutions) and stamping on special forming machines. Different paints and pigments, as well as auxiliary reagents (acids, bases, salts, soaps, etc.) are used. After painting, the fabrics are flushed (hot and cold flushing) with water and dried in tumble dryers. After stamping, the fabrics are re-dried (according to their purpose and used paints are used for "maturing" in special machines at high temperature, others go for washing and flushing);
- the final treatment of the fabrics consists of finishing, drying, smoothing, etc. Finishers are adhesive solutions of starch, dextrin, plasticizers, etc., which give the necessary elasticity, dampness, nice appearance, etc. of fabrics.

Finished fabrics are sized, graded, rolled up and stored.

Working environment conditions.

In the **mechanical machining of the fibers** the combination of the technological requirements for

performing quality spinning and weaving with the technical characteristics of spinning machines and weaving looms form specific working conditions.

The microclimate in the spinning and weaving halls depends primarily on the requirements of the technology. As a result of the high friction between the moving parts of the machines, static electricity arises, which charges the unprocessed fibers. In order to reduce it in the "spinning" in the rooms should be maintained higher - the optimal from the technological point of view humidity 55-70%, and in the "weaving" - 55 and 80%, according to the type of fiber. Another characteristic peculiarity from the technology and technical characteristics of the machinery and the production facilities are the formation and release of a considerable amount of heat, which leads to the formation of high air temperatures, especially during the hot seasons of the year. The low air velocities do not may play a significant role in relieving the thermoregulation of workers under these conditions **(Table 1)**.

The microclimate factor in spinning and weaving halls is static.

Noise is one of the main disadvantages of mechanical technology in textiles. The sound levels measured at the workrooms of the various textile factories depend on the number of machines installed, their technical characteristics and condition, the way of laying, etc., and almost always exceed the permissible hygienic norm. In a comparative aspect, the noise in the weaving halls is higher than the spinning noise. **(Table 2).** By nature, the noise is broadband - in spinning is more common medium and high frequency, and in weaving - mostly high frequency.

Silent looms generate lower sound levels - by 7-8 dB, but due to their large number in the rooms (workshops), they can not make a significant improvement in the overall noise background.

The vibrations (general) are created during the operation of the textile machines and are transmitted to the floor. Vertical vibrations are predominant, their frequency is mostly low - 13-24 Hz, and the good fitting and foundation of the machines reduces them within hygienic norms.

Machine	Level of noise in dB/A
Devils	87
Cotton combining machines	84
Cotton strips	90
Gross cuts	89
Ring machines	89
Tighteners	92
Spoolers	86
Warpers	85
Looms "Yantra"	100
Automatic looms "Covo"	101
Looms "Shyohner"	101
Looms "AT-101"	101
Looms „AT-125“	99
Looms „Vinkenson“	100
Looms „Stema“	96
Looms „Rosher“	95
Looms „Pikanol	95
Jacquard looms	94

Tab. 2. Noise characteristic of different textile machines (by S. Maximov)

Dust is also one of the main disadvantages in mechanical processing. In cotton and linen textile plants, higher dust concentrations are produced, whereas in wool and silk textiles they are lower. There have been observations that the use of synthetic materials in the textile industry also reduces the amount of dust aerosols.

The earlier the mechanical processing of the fibrous raw materials is found, the greater the dustiness is found in the work rooms - the highest dust concentrations are found in the deviling, breaking, dragging and scraping of the fibers (in batter and cotton-combining sections), gradually decreasing in spinning and weaving. The amount of dust gener-

Tab. 1. Measured mean values of microclimatic components in the spinning and weaving process

Room	Cold seasons			Warm seasons		
Stage "spinning"	T °C	Relative humidity %	V m/s	T °C	Relative humidity %	V m/s
1.Bater sections	21,5-25,6	50-70	0,07-0,42	21,0-28,5	50-80	0,02-0,72
2.Carding sections	21,0-28,0	30-50	0,04-0,20	22,0-32,4	50-80	0,02-0,70
3.Spining halls	16,0-29,3	30-75	0,04-0,32	20,0-32,8	55-80	0,07-0,46
Stage "weaving"						
4.Warping sections	16,6-23,5	35-60	0,01-0,20	22,0-28,0	40-80	0,03-0,15
5.Sizing sections	15,6-23,6	32-88	0,03-0,20	28,4-32,0	49-70	0,12-0,30
6.Weaving halls	20,0-25,0	50-70	0,07-0,35	20,0-35,0	54-75	0,60-1,20

ated also depends on the quality and the impurity of the raw material, the nature of the treatment, the technical characteristics of the machinery, the humidity, the way of cleaning the machinery and the premises, the availability and the efficiency of the ventilation equipment.

The textile powder is polydisperse - with an average particle size of 10-60 μm and falling into the rough dispersion powders. Textile powder particles less than 3 μm in length are not found, and particles longer than 5000 μm are classified as textile fibers (not in the "dust" category).

With the modern furnishings of textile enterprises with advanced textile machines, the dust concentrations are within the permissible norms - deviations are only possible in the batter and cotton-combining compartments due to the possible presence of silica.

There is also a significant **bacterial and mycotic air pollution** in textile factories linked to the amount of dust. In sorting premises eg. 2000 colonies/m^3 of air were found, in the batter - 1.5 times less, in spinning and weaving halls - 1300-4500 colonies/m^3; in most cases, it is about spore-forming and Gram-negative rods, fungus - Penicillium, Aspergillus, Mucor and others. The microbial insensitivity to flax processing is greater than that of cotton and wool processing.

In mechanical and chemical processing (finishing workshops), the microclimate is most often formed by unfavorable meteorological components. At workplaces near the singeing machines, hot dyeing, drying machines and drums, maturing machines, dressing and ironing of fabrics, etc., a lot of heat is released in connection with the specific technology. On the other hand, the use of many wet processes (desizing, boiling, bleaching, painting, continuous washing, flushing and drying of tissues) in open vessels and apparatus also creates high relative humidity resulting in overheating microclimate, especially during warm annual seasons. In cold seasons, due to lower temperatures, up to 100% relative humidity and condensation of water vapor in the work rooms, and the formation of a cooling microclimate can also occur.

The noise of machinery and equipment in wet and finishing workshops is not as intense as compared to noise in spinning and weaving halls. The sound levels of the more noisy machines (drying and washing machines, stamping machines, raised presses), are at 78-85 dB, and the exposure is shorter.

Toxic chemicals are most important to the working conditions of the mechano-chemical treatment. The carbon dioxide, carbon dioxide, and some aldehydes are released during the singeing of the fabrics. The desizing of the fabrics is associated with the fermentation of the starch and the unpleasant tooth odor (indole, skatole, mercaptans).

In bleaching pollution of the working environment with chlorine. Dyeing and stamping of fabrics is associated with many and varied chemical substances.

The cotton textile is still used in the black aniline painting Air pollution with aniline vapours can occur during painting, drying and maturing Of the processed fabrics (the black aniline dyeing has recently been limited).

Sulfur dye painting has been spread, which is related to the release of hydrogen sulphide in air during their preparation. Now days indigo- and indantren dyes find large application. They should also be reduced in advance to water-soluble leuko- compounds, but this is done with sodium hydrosulfide, whereby it is possible to remove sulfur dioxide in a minimal amount.

Very often used, so-called cold ("ice") dyeing with diazo- dyes, a process associated with the release of nitrogen oxides, both in the preparation of the solution and in the treatment of the fabric. The release of nitrogen oxides, but in smaller quantities is also characteristic of the use of indigo- dyes. In the mechanization and sealing of the painting apparatus and the presence of a suction ventilation installation, nitrogen oxides are typically from 4 to 9 mg/m^3.

Stamping the fabric is carried out most often with the same dyes mentioned above.

The use of acids in different types of painting is accompanied by the release of their aerosols. The most commonly used are acetic and sulfuric acids.

The use of finishers containing formaldehyde resins or other chemical compounds leads to the removal of formaldehyde, phenol and other toxic vapors and gases, both during dressing and in the subsequent technological operations - drying and ironing. This is especially true for the woollen textile.

Labour process in the main professions.

The spinning and weaving fabrication operations are highly mechanized and therefore the work of textile workers does not require any particular physical effort - energy expenditure averages up to 10 kJ/min, but work is hight intensity during work shift. A typical feature of textile production is its feminization.

The main and most prominent are the professional groups of **carders, spinners and weavers** engaged in the mechanical processing of fibers.

Carders serve the cotton-combining machines by placing and stopping the machines, monitoring their work, controlling the proper collection and arrangement of the cardering belt in special contain-

ers (pots). Their work takes place in the free standing position of the body and in free rhythm. No heavy weight is required, the load on the visual analyzer is moderate and the work done does not place significant demands on the attention, the memory and the qualifications of the workers.

The spinners carry out manipulations to: change the terminals and load the spinning looms; spinning; timely and qualitative binding and spinning of the breaking threads; cleaning the rollers and the entire machine from nap and others. One spinner usually serves several spinning looms. The working position of the body is free, standing or slightly bowed. Continuous walking around the machines is necessary, resulting in an average of 6-8 km for one shift. The operation of the spinners is also associated with a large number of hand movements (an average of 1000 movements per hour) with significant repeatability (more than 300 times per shift) and accuracy and speed is required. Working movements are usually performed in the maximum working area, accompanied by static effort of the body. Continuous attention and very good vision are required, resulting in significant nervous-sensory intension. The mode of work has been shifted, including night shifts.

The working process of the weavers has the same characteristics as the spinners. In this case, the search for the breaking threads of the base is also associated with the bending of the body to a squat. Serving several weaving looms requires high demands on coordinated activity of hand muscles and the visual analyzer.

The main professions in the **finishing workshops** at the mechanical and chemical processing, are those of bleaches, painters, stampers, painting workers, finishing workers, and others. The work of these occupational groups is associated with mild to moderate physical workload, standing posture, with the possibility of micro-pauses and unregulated breaks and significantly less expressed intensity compared to the workers involved in the machining. Sometimes it is necessary to carry loads, but using auxiliary equipment. In many cases, the work is carried out with a large number of unvaried working movements of the upper limbs.

The occupational activity of the above-mentioned professions often requires constant attention, continuous participation in vision; it is also associated with considerable responsibility for quality and implies good qualifications. All this results in an average nervous sensory strain.

General and occupational morbidity.

Unfavorable production microclimate, intensive noise, dustiness, contact with dyes and other toxic substances, standing position without seating in most occupations, load and overload of upper limbs, body and legs, high labour intensity, nervous sensory stress, shift mode, and more, determine occupational risk both for the occurrence of occupational illnesses and for high morbidity with temporary disability.

The long-time work in the case of intense high noise has led to varying degrees of professional deafness in spinners and weavers.

Exposure to high dust with textile dust is associated with the emergence of so-called byssinosis. There are published data showing that 52% of workers in the batter and cotton-combining premises after 10 years of service experience signs of byssinosis, and 10% - advanced byssinosis with loss of working capacity. In addition to chronic bronchitis, byssinosis is accompanied by emphysema due to long-term inhalation and accumulation of textile dust, sometimes also asthma or asthmatic states - so-called hypersensitivity pneumonitis, hypersensitivity to cotton, and other allergens in the textile powder aerosol.

The contact of the workers from the workshops with the toxic substances can lead to the occurrence of acute and chronic intoxications, professional eczema and other allergic diseases, neoplasms and others. The overload of the locomotor system is a prerequisite for the occurrence of professional myositis, tendomyositis, tendovaginitis, radiculitis, periostitis and arthritis of the joints, with deformation of the fingers, with formation of a flat foot, etc.

ITD among textile workers is characterized by high intensity indicators. In its structure, colds, female genital organs and pregnancy complications, occupational accidents, gastrointestinal and cardiovascular diseases, peripheral nervous system diseases, neuroses, and diseases of the locomotory system are the highest relative part.

During the different years (periods) there are some minor shifts in the ranking of significance in the above mentioned groups of diseases, but they are obviously the problem pathology in the textile production and to a great extent their expression is related to the specific working conditions, the nature of the labour process and the feminisation of professions.

Prevention of work.

Microclimate. Improvement in spinning and weaving workshops can be achieved by providing air conditioning (climatic instalation). The substantial healing of the microclimate in the finishing workshops can be accomplished by encapsulation and thermal insulation of machines and aggregates - heat sources; well-designed and maintained aeration of the working premises; air dusk of the worst work places and more. The limitation of released water vapor and high relative humidity is achieved by

closing all open vessels and apparatus; installation of ventilation equipment for continuous suction and discharge of water vapor. During the cold seasons to avoid condensation and work places misting, it is necessary to introduce heated air up to 40-45 ° C in the underfloor space.

Provision and use of appropriate work clothes and drinking regime, and execution of hardening procedures.

Noise. Design of non-shuttle and micro-shuttle looms; replacement of mechanical spindles with electric or pneumatic spindle-free spinning can reduce the noise level by 8-10 to 20 dB. Isolation of noisy machines by placing rubber pads, springs or other suitable materials between the machine and the place of its foundation is also important for reducing vibration. A modern method, which is widely used, is the treatment of textile machines with sound-absorbing varnishes.

Noise reduction is also achieved by sound acoustic treatment of the walls and ceilings of the premises or by sound absorbing screens - this can reduce the sound level by 8-10 dB.

It is imperative to use internal antiphonies and establish a proper work and rest regime.

Dust. There are modern closed aggregates, which ensure continuity of processes and pneumatic transmission of the raw material for defibering, carding and cardering. The periodic cleaning of the machines must be carried out pneumatically with industrial vacuum cleaners or with constructionally to machines separate suction and self-cleaning devices. Moisturizing and lubricating processed materials also reduces dust generation.

The removal of dust in textile plants by ventilation equipment is hampered by the specificities of the textile powder aerosol. When processing waste or low-grade raw material varieties, this should be done in insulated rooms with encapsulated machines provided with local aspiration. Certain raw materials also require pre-disinfection.

The use of anti-dusty personal respirators is required with high dusting and for a short time.

The removal or reduction of **toxic substances** takes place in several directions: encapsulation of the machines; mechanization of production operations; pressurization and mechanization of the process of preparation of the solutions; use of dyeing aggregates with continuous action; installation of efficient aspiration facilities; replacing chlorine as a bleaching agent with new preparations; use of less toxic dyes and finishes.

Prevention of **over-stress and fatigue** requires a number of measures to ensure a more rational physiological organization of work, such as: for each occupation, a proper working and rest regime; to apply training (make easier in working) exercises to limbs; relaxing procedures during scheduled breaks and more. Also: ergonomic machines, equipments and working furniture; natural and artificial lighting that meets the requirements of the respective professions.

Protecting the skin and eyes from toxic and aggressive chemicals requires the use of reliable **personal protection** with special workwear and shoes, safety gloves and eyeglasses. Wearing protective caps or headscarves is necessary to prevent hair from falling between rotating machine parts. The dynamic- standing posture requires working shoes with orthopedic feet.

Medical-prophylactic measures include: preliminary and periodic medical examinations with contraindications to work; dispensaries; labour rehabilitation; climate and balneotherapy; protective professional nutrition and more.

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17.7 CONSTRUCTION AND PRODUCTION OF BUILDING MATERIALS

17.7.1. CONSTRUCTION

The technology of construction works can be grouped in three main stages:

I. Works on a zero cycle (underground part of the site)

II. Elevation of the overhead part of the site

III. Finishing operations.

The first stage includes: **a) earthworks** - clearing of the site for construction, digging, consolidation of soils (through cementation, silicification, bitumination, emulsification), drilling, blasting and caissonworks; **b) construction of underground communications facilities** - water supply and sewerage systems (welding of steel, heating and gluing of PVC pipes, their laying in the prepared trenches), heating installations (installation, welding of metal pipes, anticorrosive and heat insulation works); electrical installations (withdrawal of power cables and their laying in the trench); **c) drainage and building of the foundations of the site** - the foundations are made of concrete, which is pouring mechanically or manually into pre-prepared formwork; reinforcement columns are planted in the foundations and concrete sealing is performed by vibrating with needle vibrators.

Most of the I-stage activities are mechanized. Excavation and transport-technology machines are used - excavators, bulldozers, scrapers, grooves and others. Sometimes (underground, high-altitude, rocky soils, etc.) a part of earth-excavating, drilling and blasting operations are carried out by hand-held electric and pneumatic vibratory tools.

The second stage depends on the type of the site, the way of construction and the building materials. It is built mainly with ready-made items and bricks.

In the modern **industrial construction** is done the assembly of ready-made constructions and elements (reinforced concrete, steel). They are fastened by welding and subsequent cement coating at the joint site. The assembly works can be carried out from the foundations up to tens and hundreds of meters high, using cranes, helicopters, welding machines and hand-operated tools (electric perforators, welding pistols, pneumatic spanners, drills, etc.).

In the case of **monolithic construction**, the construction of the site includes the arrangement of bricks (clay, silicate) in horizontal rows, their connection and attachment with building mortars made of cement, lime, sand, gravel and water. The monolithic concrete and reinforced concrete structures (slabs, beams, columns) are made using cranes, concrete pumps, vibrating equipment for sealing concrete, and in small objects mainly hand tools - as electric- and air tampers.

The second stage includes carpentry, roofing, anti-corrosion work, hydro-, heat- and sound insulation work.

Carpentry works include: 1. installation of roof and floor wooden structures; 2. installation of joinery - frames, doors, windows; 3. installation of parquet, paneling, railings. Timber building is delivered to sites pre-treated to protect wood with antiseptics, insecticides, fungicides, anti-pyrans. Also, various adhesives, such as tartal, casein, dextrin, nitrocellulose or newer-, urea formaldehyde, melamine formaldehyde, resorcinol formaldehyde resin, epoxy resin, natural latex, neoprene adhesives, polyamide, polyester, polyvinyl acetate, perchlorvinyl, cyanoacrylate. Finishing operations in carpentry are polishing, varnishing, staining and painting. The wood is coated with liquid film-forming substances - non-pigmented (lacquers - polyester, polyurethane based on amino formaldehyde resins, polyvinyl chloride, perchlorvinyl etc.) and boiled oil - polymer of vegetable, dried oils - flaxed, hemped, tunged) and pigment (paints, enamels). Dyes and enamels contain inorganic pigments (compounds of zinc, titanium, aluminum, iron, chromium, lead, cadmium, manganese) and fillers (chalk, bentonite, kaolin, kieselguhr, aerosil, barite, mica, talc, asbestos) in stable suspension with organic solvents. In varnishes, lacquers and paints, the most commonly used organic solvents and diluents are toluene, xylene, cyclohexane, methylethylketone, acetone, ethylbutyl-amyl acetate, turpentine and the like. For painting, mostly non-mechanized hand tools, as well as electric and pneumatic tools, are used.

Roofing includes various technological operations and materials depending on the type of roof. For **flat roofs** the main operation is waterproofing. It is made with rolled bituminous materials (tarpa-

pers). These materials are bonded with cold (bitumen, dissolved in gasoline) or with hot bitumen and tar adhesives (coal tar). By another technology, the gluing is done by direct heating of the roll materials with propane-butane burners on the prepared roof surface. Recently, non-rolled waterproofing materials based on polystyrene and other polymers with organic solvents as solvent - naphtha and xylene have also been used. In this case priming with polymer-cement and polymer-concrete compositions. The surface is first dried and then absorbed with organosilicate compositions, furan and vinyl polymer compounds. When building **sloped roofs**, the operations include the installation of a wooden structure, piling of roof tiles and basalt tiles, installation of gutters, etc.

Anticorrosion works include painting and lacquering of metal structures and impregnation with polymer compositions and lining of reinforced concrete surfaces. For **metal facilities**, rust cleaning is performed first, - manually, mechanically - by sandblasting, or thermally - with oxygen-acetylene burners or gasoline lamps. Another method is to apply a rust convertor (sulfuric or phosphoric acid). Then degreasing with organic solvents and finally applying the anti-corrosion coating. It includes priming with oil, alkyd or nitrocellulose primers, painting (minium, iron oxide coatings, etc.) or lacquering (epoxy, polyvinyl chloride, polyacrylic lacquers). **Ferro-concrete surfaces** are cleaned, puttyed and grinded, followed by priming and application of polymer coatings. In some cases, the anticorrosive treatment involves rocking (limestone, marble, granite, ceramics, terracotta) that are prepared by cutting, and grinding.

Thermo- and sound insulation is performed with: 1. Porous bulk grain materials (perlite, ceramsite, vermiculite, etc.) - mainly for thermo - and sound insulation of floors; 2. fibrous materials (glass and mineral wool) - for thermo-insulation of pipelines; 3. use of prefabricated monolithic structures made of aerated concrete, foam concrete (for thermal insulation of roofs), foam plastics (foamed polystyrene-styropor, polypropylene, polyurethane foam, foamed phenoplastics, etc.).

The third, final stage includes the following basic finishing operations: 1. installation of internal water and sewerage systems, electrical installations, district heating; 2. production of floor coverings; 3. external and internal finishing-painting works.

The installation of internal water and sewerage systems is related to the drilling of holes in masonry and concrete, the preparation (cutting and bonding) of the pipes and their fixing in the walls, heating and gluing of PVC pipes. In the district heating, the welding of the pipes with electrical, anticorrosive and thermoinsulation works, installation of radiators. Basic operations during the installation of the electrical installations are electric cables, installation of distribution boxes, switchboards, sockets, switches. The technology of making floor coverings depends on the type of flooring. On wooden flooring - see carpentry; in mosaic, ceramic, terracotta coatings - see anti-corrosion work; in the case of monolithic polymer coatings (polymer-concrete, polymer solutions, self-leveling coatings based on epoxy resins), the priming and coating of the polymer coating in basic and leveling layer, grinding and varnishing, are prepared after preparation of the starting materials. In the interior and exterior painting works are carried out after the preparation of surfaces - cleaning with electric brushes, coating and flattening, plastering, priming and painting.

The third stage, as well as the second, is less mechanized than the first. Some operations are performed with electric and pneumatic tools.

Working environment conditions

The working conditions of construction workers are difficult to characterize in general due to the heterogeneous technological processes and the impact of a number of specific factors such as:

- frequent change of work places - variability in the working time of a given place from single days to months, with frequent movements of workers within or outside the country; often - remoteness of sites from settlements;
- differences in construction methods;
- non-permanent working conditions due to outdoor or unfinished, non-isolated premises, in cabins of transport or transport technology, often uninsulated, insufficient or unheated; daily show influence sunshine intensity, wind direction and velocity, humidity and rainfall, daytime and seasonal temperature fluctuations, and in some cases-altitude;
- working at different heights and having mechanisms for moving heavy loads at different heights;
- there is a risk of contact with hazardous materials used in the past (crocidolite asbestos, lead-containing anticorrosive coatings, etc.) for collapsing and repairing buildings.

The main adverse factors in the construction are: **weather conditions**, **dust** (from rock and material, sand and soils, lime, cement, gypsum, concrete, asbestos, ceramics, polymer materials), **toxic gases and vapors** (welding gases and aerosols , explosive gases, exhaust pipes, bitumen, tar, peaks, resins, adhesives, varnishes and paints), **noise and vibrations** (from transport and transport technology, construction hand-held machines - electrical and pneumatic).

In **the first stage**, in the execution of the earthworks, the main workplace with a complex influence of the production factors is the cabin of the excavation and transport-technological machines. The cabin air temperature in the summer reaches 37°C, in the winter - 4-12°C (up to - 4°C). The humidity depends on the season, rainfall, region, altitude. The concentration of dust depends mainly on the degree of sealing of the cabins, but also the state of the site's area, the roads, the amount of rainfall. The dust usually consists of quartz particles and sand, the silicate and free silica content varies depending on the region. Fuel combustion gases (carbon monoxide, nitrogen oxides, aldehydes - acrolein and formaldehyde, hydrocarbons, soot, sulfur dioxide, oil-based lubricants) are also found in the cabin air. The composition of the gases varies depending on the type of engine, power and technical condition, operating conditions, fuel type, etc. When working on transport and transport technology machines, noise and vibrations are generated, with noise being more intense in low and medium frequencies. The vibrations are general, with significant amplitude, with impact and by local at controls. In manual earthworks (such as drilling, blasting), exposure to dust and unfavorable microclimate is leading, depending on the season. Hand-held power tools and pneumatic tools have noise-vibration effects (local vibrations). When the soils are strengthened there is exposure to dust and vapors of chemical compounds: in cementation - cement, lime, slag and chemical additives; for with sodium-silicate solution silicification and accelerators - calcium and sodium chloride powder, aluminum sulphate, sodium silicofluoride, phosphoric and sulfuric acid vapours; in bituminization - polycyclic aromatic hydrocarbons, hydrogen chloride; when resinized with carbide resins - formaldehyde, phenol, furfuryl. There is also a skin contact with these substances. In the case of **caissonworks** (tunneling, construction in or under water basins, laying the foundations of bridge constructions), the leading unfavorable factor is the increased atmospheric pressure. The air in the caissons can be contaminated with dust and a mixture of chemicals entering by the compressed air as well as soil (methane, hydrogen sulphide), explosives (carbon monoxide and dioxide, nitrogen oxides) and welding gases. A number of technological operations (eg. pneumatic tools) generate intensive medium and high frequency noise. When building underground communications, working conditions are determined by the meteorological factor (depending on season), dust (soil particles, building materials), noise-vibration factor (hand-held electric tools, construction machines), chemical pollutants in some specific operations (eg. on heating and bonding of PVC pipes - products of thermal and thermo- oxidation destruction of PVC - carbon monoxide, vinyl chloride, lead or cadmium aerosols, aldehydes, etc., welding of pipes - welding gases, aerosols of metals and their compounds). When constructing the foundations hygienically significant occupational injuries are: the meteorological factor - depending on the cooling or overheating season, with radiation cooling from the surrounding surfaces; during installation of the fixture by welding - gas-aerosol mixture; when compacting the concrete mixture - generating intensive noise (90-115 dB) and impact of general and local vibrations (amplitude 0.1-0.9 mm, frequency - 45-105 Hz); removal of dust (concrete, cement, soil particles).

In the **installation works**, the hygienic importance of the meteorological factor depends on the climate of the region and the season, as well as on the height at which it is being built. When using the hand-held power tools generate noise and local vibrations; intense noise is also generated when working on bridge cranes, helicopters and loading elevators. In the case of **monolithic construction**, the meteorological factor is also leading, and additionally with great hygienic importance is the cooling from the long, direct contact with the cold surfaces of the building materials and tools. In the air of the working environment dust of cement, lime, gypsum, sand, etc., also containing free silica, is released. There is also the possibility of prolonged skin contact with highly alkaline building solutions (lime-cement).

When carrying out the **carpentry**, the meteorological factor is again the most unfavorable factor. When using mechanized hand tools, intermittent noise and local vibrations are generated. In operations such as planing, grinding, circling in the air comes rough and fine wood dust (including exotic wood species), which also supports various chemical substances used in the treatment of wood (creosote and anthracene oil vapours, compounds of copper, zinc, mercury, chromium, chlorinated naphthalenes, chlorinated methane and ethane, nitro compounds). When assembling wooden parts using polymeric adhesives, vapours of organic solvents and outgoing monomers (toluene, butyl alcohol, formaldehyde, epichlorhydrin, etc.) are released. In the polishing, varnishing and painting of wooden surfaces the leading production factor is the chemical - the vapors are released from organic solvents. Hazardous are two-component varnishes, which use high-toxicity substances such as isocyanates - from polyurethane varnishes, ethylenediamine - from epoxy varnishes (catalyst in epoxy resin). In the aforementioned carpentry there is a risk of fire and

explosion due to the easy flammability of wood and adhesives, paints and boiled oil.

In the **waterproofing works**, the most important industrial noxiousnesses are dust and chemical contaminants. Powder is intensively removed during the preparation of the substrate - rough or fine depending on the technology - concrete, cement, sand (containing free silica, chromium). Chemical contaminants are released during priming (depending on the nature of the primer) and when applying waterproofing materials. With the use of cement-sand primer, workers are in contact with alkaline building mortars, and when applying heated bituminous - polymerics and asphaltics primer emited polycyclic aromatic hydrocarbons, initial monomers - furfuryl, acetone, furfur-acetonic monomers, vinyl chloride, thermal products of destruction - formaldehyde and other aldehydes, higher alcohols, chlorinated hydrocarbons etc., from the hardeners - vapours of sulfuric and phosphoric acid. In the hot fixation of rolled bituminous materials with bitumen and tar adhesives, the main pollutants are polycyclic aromatic hydrocarbons, carbon monoxide, sulfur and nitrogen oxides, and in the case of cold gluing - petrol hydrocarbons. Using nonrolled materials naphthane hydrocarbons and xylene, emited. The unfavorable meteorological factor (depending on the season) is also important. The use of sandblasters, vibrators and others instruments is associated with exposure to intense noise and local vibration. In the **anti-corrosion works** for protection of reinforced concrete structures, the production deficiencies are analogous to those indicated in the waterproofing works. Only in the preparation of dry-stone lining dust is released in higher concentrations and with a higher content of free crystalline silicon dioxide. In the case of sandblasting of rust to protect metal structures, dust containing iron oxide, particles of sand with a high content of free crystalline silica are released. In the chemical cleaning and degreasing of the surfaces, the vapors are released from the chemicals described in the technology. **Hydro- and noise insulating** works are mainly associated with exposures of foamed concrete dust, dust of glass and mineral wool, chrysotile asbestos and others. There is also a skin contact with fibrous materials and alkaline solvent mixtures containing chromium, nickel, cobalt.

The **finishing operations** are carried out in the case of unfavorable, seasonal fluctuations in the meteorological factor and with the impact of specific noxiousnesses depending on the type of operation. When laying the WC installations, the leading factor is the dust and the heating of PVC tubes and welding and soldering of metal pipes - the chemical. The production of the flooring is related to the generation of intense noise, local vibrations and dust (wood, mineral with free silica content, cement). When applying polymer flooring and painting, the leading factor is the chemical.

Labour process in the main professions

In modern-day construction, much of the technological operations are carried out mechanically, but there are also many that are done manually. The relative share of manual work is high in small sites, as well as in difficult accessible areas - in high mountain areas, water basins and others.

For most occupational groups, the work process is characterized by medium physical work - for example, the work of the drivers of the transport-technology building machines and the other means of transport. The working posture is mainly seated, excluding maintenance-related for repair time, as well as for assembly and disassembly of machinery when the posture is standing, forced, with a nonphysiological position of the body. When driving machines due to the often non-ergonomical layout of the control levers and pedals, the sitting position is also characterized by a considerable static load and forced position of the body. Driving is also associated with constant observation and control, and work is characterized by nervous sensory and neuro-emotional intension because of the need to make immediate decisions in complex situations.

The work of other workers (engaged in insulation, anti-corrosion and carpentry) is characterized by alternating operations. There is a significant physical stress in lifting and carrying weights, with a wide use of manual labour and a static load to maintain the often forced posture, which for a time changes with standing, bending to the knees, with support often on an inclined surface. The work of plasterers and painters is more monotonous, characterized by a great monotony of movements requiring physical effort. The working posture is standing, very often - forced - a strong slope downwards with folding at the waist, on the knees, or vice versa - lifting of the fingers, stretching up arms over the shoulders and stretching back and upward head, and pulling the neck, sometimes at high altitude . The inconvenient working posture is associated with a significant static load, most commonly in the neck and local, upper limb, loads. The masonry work is considered the most labour-intensive in building. Along with the inconvenient, forced working posture - a curved, leaning forward body, work mouvments is also a problem. They are monotonous, with a lot of monotony, repeated many times, with significant load on the locomotory system and separate muscle groups on the hands (mostly on the wrists). For one shift, about 4000 uniform movements are performed with the

right hand and a load is moved to 6 tonnes (bricks, blocks, mortar, etc.), including on height.

General and occupational morbidity

The incidence rate of temporary incapacity for building workers is determined by the working conditions of the different occupational groups, their age-sex composition, the sanitary-living conditions, the organization of nutrition and often the remoteness of the construction sites by healthcare services. Typical of this industry is seasonal dependence - much higher morbidity during the cold and transition seasons of the year.

In **the structure of the ITD**, most intensive are diseases of the upper respiratory tract and the lungs, diseases of the locomotory system (LS) and the peripheral nervous system (PNS) - radiculitis, polyneuritis, skin and subcutaneous tissue (contact dermatitis, eczema, photodermatitis), diseases of the gastrointestinal tract. A very high frequency and weight in construction is traumatism.

Occupational diseases are the most common among construction workers, such as those of the LS and PNS, pulmonary aetiology (pneumoconiosis, bronchitis), occupational intoxications, vibrational disease, occupational hearing impairment, caisson disease (underwater). In some professions (concrete, bricklayers, plasterers, painters, carpenters), allergic diseases of the respiratory system and skin (contact with cement, wood dust from exotic trees, resins, oils, organic solvents, etc.) are high. Intoxications with organic solvents (for painters, parquet workers), lead (for anticorrosive coating workers), metal aerosols and gases (for electric welders and fitters) have been reported. Pneumoconioses suffer from isolationists (asbestosis), stoneworkers and sandblasting (silicosis). Cases of vibrational disease have been reported in concretors drivers of heavy-duty construction machines, crane drivers, grinders, and pneumatic machines. Hearing damage (reduction to loss) occurs in carpenters, bombers, concreters, and craneists. Risk factors for the occurrence of carcinogenic diseases are workers in contact with asbestos and derivatives, zeolite, chromate, nickel, coal, bitumen, asphalts, peaks, as well as workers with ultraviolet impact.

Prevention of work

In order to prevent the impact of unfavorable **weather conditions**, it is necessary during the cold period to take short breaks for warming in specially equipped rooms, offering hot drinks or stopping outdoor work at extremely low temperatures. When working in high temperatures, a special working regime is required with longer interruptions in the hottest hours of the day and shift of workday earlier in the morning and later in the evening and provision of a drinking regime according to the operative regulations.

Removal of the harmful effects of **dust and toxic substances** can be accomplished by applying progressive technological lines, sealing the equipment for dimensioning, dosing and preparing concrete, mortars, adhesives, paints, and providing effective local ventilation. In order to reduce exposure to asbestos dust, it is necessary to pre-spray water and to place warning signs and inscriptions for increased asbestos concentration, when destroying asbestos-cement constructions. To reduce exposure to quartz dust, it is recommended to work with wet materials, spraying water in the work area, organizing effective local ventilation with removable suction devices, sand replacing in sand blasting with slag or metal ball. Replacement of highly toxic by weakly toxic substances helps to reduce exposure to harmful chemical compounds - ex. replacing the organic solvents with water emulsion, inactivation of the chromates contained in the cement and its solutions by addition of 0.1-0.2% iron sulphate and the like.

To reduce heavy manual labour with **physical load**, it is recommended to mechanize heavy and labour-intensive operations, especially for lifting and carrying loads. For workers with a high occupational risk (exposure to silicozogenic and carcinogenic dusts and chemical compounds) it is necessary to carry out prolonged prospective **medical surveillance** in order to identify possible health effects after a latency period. Workers should be aware of the immediate occupational risk by providing information leaflets, incl. and instructions for safe operation and basic information on first aid in traumas, acute intoxications, etc.

The most effective for building workers is personal protection with **appropriate work clothes and personal protective equipment (PPE)**, especially for high-risk work. When exposed to silicone, asbestos and others mineral powders, respiratory protection is recommended with dust masks, retaining particles 2-5 μm and finer ingredients but with the least resistance to inhalation; when working with sandblasters - insulated air-carrying helmets; When exposed to lime dust - a protective mask combined with safety glasses. Protection of the body from dust is carried out with anti-dusting suits, made of dense cotton fabrics, the protection of the hands - with tarpaulin gloves; in case of skin contact with cement dust or lime, protective pastes and creams under the gloves are recommended, when exposed to glass fibers - whole body protective dress, hands, safety glasses or facial anti-dust helmets. To protect the respiratory organs from gases, vapors and aerosols

with exposure to paints and varnishes, half masks with suitable filters are recommended, and for body protection - suitable cotton cloths reinforced in front with a coated cloth. In the case of coal tar and peat, we recommend workwear of impermeable material covering the whole body; when welding in confined spaces - gas masks with a suitable filter or insulating breathing apparatus. The PPE and workwear must always be fitted with crash helmets, seat belts, etc.

17.7.2. PRODUCTION OF BUILDING MATERIALS

Building materials are several groups: 1) natural a) compact materials (stones, rock fragments); (b) bulk materials (sand, gravel); c) inorganic binders (cement, lime, gypsum); 2) concrete and building mortars; 3) ceramic materials and articles (bricks, tiles, etc.); 4) thermal insulation and acoustic materials and products (mineral wool, perlite, glass fiber, styrofoam); 5) organic binders and articles (fiberglass, epoxy resin, linoleum, etc.); 6) roofing and waterproofing materials (ruberoid, asphalt concrete, etc.); 7) metallic materials (fittings, iron structures); 8) wood materials (beams, parquet, woodwork, etc.); 9) building materials of polymers (profiles, mill work, cladding, PVC pipes and other polymers); 10) lacquer-painting materials.

Cement production. It comes in two technologies - dry and wet. **Raw material** for production are limestone, marl, clay and corrective mineral additives (tras, zeolite, kieselguhr, gypsum, blast slag, ash from TEPP, etc.). Depending on inputs, cement with different qualities is produced. The raw materials are crushed, sieved, mixed, dried and baked. When burning as fuel, fuel oil, gas or coal (powdered) is used. In the **wet method**, fine grinding (crushing) is done in water to form a charge in the kind of an aqueous suspension - the so-called slime. In the **dry process**, the charge is in the form of a fine dry powder. The next step for both methods is to bake the slime/charge at 1300-1400°C in vertical or rotating inclined furnaces to form solid lumps - clinker. After removing the furnace, the clinker is subjected to rapid cooling. The clinker is maturing in a storage room, after which the mineral supplements are added, and they are grinded and sieved with them. The resulting cement is stored in large concrete silos. The **equipment** used in the course of the process includes: crushers and mills (hammer, roller, jaw, cone, rotating ball mills), crushers, shaft or rotating inclined furnaces, silos with compressor mixers, automatic filling machines. Contemporary cement plants are highly mechanized and automated production with remote control and management.

The leading unfavorable production factor in cement production is the raw material **dust** and of the finished product. Dust concentrations fluctuate widely, up to tens of mg/m^3, depending on the degree of mechanization and process automation, and on the effectiveness of ventilation. The material composition of the powder comprises from 20 to 80% total silica, with an average of about 2-3% being of free crystalline silica. The amount of crystalline SiO_2 depends on the different types of starting materials and additives used and the cement brand. The powder composition also includes small amounts of calcium oxide, magnesium oxide, bicarbonate and alumina, manganese and barium oxide. Also, hexavalent water-soluble chromium in concentrations between 0.004 and 0.008 mg/m^3. The dust is mostly finely dispersed - 70-98% of the particles are less than 5 μm.

The microclimate in the different stages of the technological process has different characteristics. The milling of the raw materials works at acceptable levels (during the winter season with a cooling character), while during drying and baking of the raw material to clinker, the temperatures are high and during the warm period can reach 40-42 ° C. For the overheating microclimate, the impact of infrared radiation (measured at 3780-5040 kJ.m^{-1}.h^{-1}) is measured by heated outdoor surfaces of kilns, drying drums. It works outdoors during all seasons.

Noise and vibration. In the crushing, grinding, sifting and transport of raw materials and finished product processes, the used technological equipment generates medium and high frequency noise with a significant intensity of 90-115 dB/A. When operating the burners of rotating kilns, the measured sound levels are between 110 and 120 dB/A. Two to three times of the norms were exceeded by general vibrations when working on ball mills and pelletizing trays.

Chemical pollutants. When baking the raw materials to the clinker it is possible to produce CO, CO_2, SO_2 hydrocarbons from the fuel and their entry into the working environment (around the furnaces).

Work process. From the technological staff, the main professions in the cement industry are: a miller, a furnacer and a cement filling machine. The number of auxiliary staff - fitters, locksmiths and maintaining transport and sanitary facilities - is also significant. Technological staff works at a slight physical load, mostly in a standing position about three-quarters of the shift, with a free rhythm of labour. Work is predominantly of control and managerial nature, but it is related to risk and responsibility. Manual operations mainly involve periodic sampling. The work of cement fillers has a great physical load. Contempo-

rary cement production with a high degree of automation leads to high nervous sensory and nervous-mental intension.

Morbidity of workers. Occupational morbidity is mainly associated with the impact of cement dust. Cementary pneumoconiosis is benign and progresses very slowly. Single cases of silicosis in cement production were recorded in milling and belt conveyance workers. A particular manifestation of the effect of cement dust is the so-called "Cement pneumonia" - fibrosing bronchitis, bronchiolitis and obturative emphysema. Chronic bronchitis with dust etiology is common. Relatively high incidence is also bronchial asthma and allergic rhinitis that are associated with the effects of hexavalent chromium. Diseases of the respiratory system are also high in the structure of morbidity with temporary disability. There is also a high incidence of professional dermatoses - "cement eczema" (micro-impurities of chromium, perhaps cobalt and nickel), and other skin diseases - nail-around erosions and skin infections (furuncles, abscesses, panartiums). Other occupational diseases include eye diseases - conjunctivitis, occupational deafness and stomach and duodenal ulcerative disease.

Prophylactic measures are aimed at: **reducing dust generation** by encapsulating and sealing the equipment and dust-free cleaning of production facilities; **reducing the intensity of radiant heat** by installing heat screens, thermal insulation of the equipment and control of production processes by insulated (closed) control panels; **improvement of microclimatic conditions** by means of general mechanical ventilation; **reducing the hard labour** through mechanization and automation of technological processes; medico-preventive measures - preliminary and periodic medical examinations, PPE, protective-professional diet and drinking regime (at overheating).

Manufacture of ceramic products (bricks, tiles). Ceramic products are a product of pure clay or its mineral admixtures. The most common minerals in clay are kaolinite and montmorillonite, which are contained in small amounts of mica, feldspar, calcium carbonate and iron oxides. Mineral supplements include: quartz sands, diatomite; burning materials - coal dust or sawdust; also feldspar, perlite, tuff, carbonates.

The main stages of production are: extraction of raw materials; preparation of the raw materials; Molding or pressing of bricks and roof tiles; drying; burning; storage and expedition.

The extraction of raw materials is most often in open quarries and mechanized (excavators, bulldozers, etc.). The preparation of the raw materials includes mainly clay processing operations (crushing, sifting, battering, wetting, mixing and kneading). **The production of bricks** is done in two basic ways: 1) **plastic (wet)** - crushing the clay with rollers; adding water to a blender; subsequent rolling of the mixture; passing the clay through a horizontal grinder to a homogeneous plastic molding table. The bricks are molded most often mechanically (also manually with a template), making the clay tape (obtained in belt presses) with subsequent passage through automatic or semi-automatic apparatus, cutting it to undrying bricks. The bricks before burning are dried in adjustable furnaces (tunnel dryers, etc.) or outdoors. 2) **semi-dry and dry** - after the addition of admixtures to clays, preparation of clay powder by drying and grinding, artificial humidification and rolling of semi-dry material, screening and feeding of mechanical presses, in high-pressure steel molds. Bricks made by this method are baked without pre-drying. Bricks are baked in ring and tunnel kilns for 36-48 hours at 900-1000 ° C. The fuel used is solid (coal) and gaseous. At present, electricity kilns is used - band and multichannel.

In the **production of tiles**, the plastic method is mainly applied. The quality requirements for clay are high. Molding, drying and baking are analogous to the production of bricks. It is baked at a higher temperature - 1000-1100 ° C.

Working conditions. In the manufacture of construction ceramic products the main adverse factors of the working environment are meteorological and dust. The chemical factor is also important for the "burning" process. In the extraction and preparation of the raw materials, the noise-vibrating factor can be added to the leading unfavorable factors.

The meteorological factor in raw material extraction is determined entirely by seasonal fluctuations - outdoor work. During drying and especially during burning of articles in ring kilns, air temperatures reach 30 and 50 ° C, combined with intensive infrared radiation (measured 5000 $kJ.m^{-2}.h^{-1}$) when loading or unloading bricks. **Powder** is released at high concentrations in the extraction and preparation of the raw materials - measured from tens to hundreds of $mg.m^{-3}$. At lower concentrations is the dust when removing the articles from the furnaces (2-25 $mg.m^{-3}$). The free crystalline silica is between 1 and 20%. The dust particles have a high dispersion, which determines their high respirability. **Chemical compounds** (CO, SO_2, CO_2) are released into the air in the dryer and mainly in furnaces as a result of incomplete combustion of fuels. Highest noise levels (over 100 dB/A) as well as **general vibrations** are generated when the raw material preparation equipment (crushers, ball mills) is used. The noise of the means

of transport is of higher sound pressure in the low and medium range. The general vibrations here are of considerable amplitude (thrusts).

Work process. The group of technological workers is formed by the following professions: metering, milling, molding, cutting, furnace attendant and loading workers in furnaces (filling, extraction). The working position of all is standing for 75-95% of the working time. The work is of average physical load, except for manual (non-mechanized) loading of dryers and furnaces of unfinished bricks and removal of dried / baked products. The total load transferred for one shift reaches 10 t. For most professions, work has a free rhythm. For service belt presses, conveyor lines the rhythm is imposed. For all professional groups the work is related to uniform operations and expressed monotony.

Illnesses of workers. At the highest incidence in the structure of LS are the diseases of the ITD and PNS (mainly of the upper limbs). They are associated with both the overload of the skeletal-muscular system and the significant physical load as well as the unfavorable cooling or overheating microclimate. Acute and chronic diseases of the respiratory organs, production and domestic traumatism are also increased. Occupational diseases are characterized by the pneumoconiosis "kaolinose" (silicatosis). With a higher content of free crystalline SiO_2 (in the sand fractions of the clay) develops mixed silicosis (not pure silicosis). There is an increased risk of occupational intoxication with carbon monoxide in the furnace service.

Prophylactic measures include: using technology with the plastic method instead of dry and semi-dry; complex mechanization of the entire technological cycle; use of up-to-date equipment - tunnel dryers and furnaces, electric furnaces instead of ringing; covering (encapsulation) of crushers, conveyor belts; humidification of input powders (especially sand) and wet cleaning of production premises; good thermal insulation on the surfaces of the driers and furnaces, shielding, air showering; working and rest regime in the conditions of overheating; appropriate work clothes and PPE; under conditions of overheating adequate drinking regime and protective professional nutrition; preliminary and periodic medical examinations.

Production of concrete. Depending on the type of binder used, the concrete is: cement (most common), silicate (based on lime), gypsum, polymer concrete (based on synthetic resins - polyester, epoxy, carbamide etc.). By using different types of cement and special chemical additives, concrete with different properties can be obtained. Plasticizers, accelerators, retarders, air blowers, foam- and gasifiers, sealants, antifreeze, etc. are widely used. In the specialized plants the production of concrete is fully mechanized and partially automated. The technological process takes place in the following stages: 1) storage of the raw materials in a warehouse for preliminary preparation of inert materials (sieving, flushing); 2) transport of raw materials in a concrete workshop (in silos) and from there to the dispensers; 3) dimensioning of materials of automatic or semi-automatic scales (dispensers); 4) loading - direct feeding of the measured materials from the dispensers into concrete mixers and mixing them with water; 5) dosing, feeding and stirring the additives in the concrete mixture; 6)delivery of ready concrete by pumps in concrete trucks or special metal buckets.

Working conditions in concrete production are characterized mainly by high dusting, cooling or overheating dynamic weather factors and intense noise. **Dust** is the leading harm in the working environment. In the warehouse it is mainly released during unloading and preparation of building materials. The highest concentrations of unloaded cement from the wagons/cement plants, although in most cases pneumatic transport is used. Concentrations reach several hundred $mg.m^{-3}$. Lower quantities of dust are released when transporting inert materials - when pre-wetted to 10-20 $mg.m^{-3}$. When transporting dry materials with open belts, dust concentrations are very high (between 300 and 400 $mg.m^{-3}$). The powder has a very high dispersion (70-98% of the particles have a size up to 5 μm).

Noise - when moving conveyor belts - 75-86 dB/A; when working on pumps, serving pneumotransport and dosing devices - 90-110 dB/A; when paddle mixers and concrete mixers - 89-105 dB/A. Generated noise is constant, over-rated (over 85 dB/A), mostly low and medium frequency. When working on concrete mixers, **general vibrations** transmitted on the floor, exceeding the norms of 6-9 times, are generated. **Weather conditions** are especially unfavorable for workers in a warehouse - outdoor work. Meteo- conditions in silos, dosing devices and concrete mixers are similar, as air is free-flowing through the production area. Most often, the microclimate is characterized as being cooling and dynamic.

Work process. From the technology staff, the main profession is the operator - on "dosing devices", "concrete mixers". The work of the operators in the majority of working hours is a low physical load, whereas in the case of breakdowns, with manual work, there is a medium to heavy physical load (in the case of manual - non-industrial preparation and transport of concrete, labour is heavy and very heavy physical load). For the most part, the operator's work is associated with nervous sensory and nerve-mental

intension due to increased requirements for the attention activity for permanent control of panels and timely reactions. The operative position of the operator is mostly sitting, but also standing and forced depending on the manual operations performed. The job does not require high skill, accuracy, speed and processing of a large amount of information. In the warehouse, the main profession is a craneist. The working posture is constantly sitting. Workers are exposed to adverse weather conditions, noise, general and local vibrations (from the control levers). When working with dry aggregates and a poorly sealed or enclosed cabin, dust exposure is also possible. The work is with a low physical load, but with nervous-sensory, nervous-mental and nervous emotional intension due to continuous work at height and in tightly closed space. Requires high attention, good visual-motor coordination, accuracy and speed.

Illnesses of workers. Workers have experienced the following occupational diseases: occupational chronic bronchitis, bronchial asthma, allergic rhinosinusitis, cement pneumoconiosis ("cementosis"), skeletal and muscular system overload, peripheral nervous system, occupational deafness, dermatoses The structure of non-professional diseases high frequency are acute respiratory diseases, lumbosarral radiculitis, domestic and industrial traumatism.

Prophylactic measures. They concern mainly dust, unfavorable weather conditions and noise-vibration impact. Dust protection measures include: wetting inert materials prior to transportation; encapsulation of conveyor belts; good sealing and hermetically of equipment, incl. soft connections to and from dispensers; local exhaust ventilation device for dispensers, concrete mixers; good isolation of the cabins of the control panels; use of PPE - anti-dust masks and half masks when leaving the cabins; provide adequate work clothing and PPE for eye protection as well as bathing conditions after work. In order to avoid the adverse impact of the meteorological factor, it is recommended: to provide warm premises and hot drinks for the employees in the warehouse; local heating of the crane and control panel cabins; appropriate warm work clothes; when working at high temperatures, providing ventilation in the crane cabins as well as drinking (mineral) water. To avoid the impact of general vibrations, it is advisable to: foundation the concrete mixers properly; providing crannets with shock-absorbing seats and a comfortable semi-soft air-tight back; providing PPE for hearing protection - antiphon, earbuds. In order to avoid heavy physical load, it is recommended to maximally mechanize the technological process.

Also, health control according to the Ordinance on preliminary and periodic medical examinations.

Manufacture of reinforced concrete products. Ferro-concrete elements, panels (eg, facade, floor, bulk, for building), as well as beams, columns, pipes, etc. are made of reinforced concrete in specialized factories. Prepared elements are mounted directly on the construction. The process involves: 1) preparing a concrete mixture; 2) preparation of reinforcing nets, metal profiles and structures and installation of the fittings; 3) forming of the products; 4) heat-moisture treatment of molds; 5) release of finished articles from shapes and finishing operations; 6) quality control and expedition.

Raw material for **reinforcing meshs** are rolled concrete iron, which straightens, sizes, cuts, bends profiles. The cut iron is welded to special molds by spot or arc welding. Depending on the degree of mechanization and automation of production, we use: automatic metal cutting machines, automatic and semi-automatic bending machines, spot welding machines with hand-held electrode, automatic multi-spot welding machines, electrowelding, automatic cutting machine and armature shears. **The forming process** is carried out in metal moulds of different sizes. It consists of the following operations: cleansing of concrete remnants (hand-blasting, levers, air blowing); daubing moulds with antiadhesive (formwork) emulsions (hand brushes or spraying); closure of moulds; placing the reinforcing net in them and filling the concrete mixture; sealing the concrete in moulds by vibrating means (hand vibrators, vibrotables, vibroplatforms) and non-vibratory methods (compression, centrifugation, vacuum compaction). **Heat-moisture treatment** (for accelerated concrete hardening and rapid release of the molds by: steaming (steam 70-85 ° C, 8-12 hours in the chambers, steam tubing with steam feed under the mold casing), in hot water pools autoclave treatment, electro heating; **Releasing** of hardened reinforced concrete products from crane, telfers, straightening on stands and **finishing hand operations** (cleaning, tapping, loosening of connecting fitting, filling of slots and joints with cement mortars, puttying, etc.) Quality control and storage.

The most important factors in the working environment in the production of reinforced concrete products are noise, vibrations and unfavorable microclimate. Which is important in some stages is the separation of dust (cement, silicate, metal-iron oxide) and chemical compounds (naphthenic aerosols and vapors, alkaline aerosols - sodium and potassium-bases, welding aerosols and gases). High **noise** levels are generated by: construction of the fitting (cutting - 94 dB/A, single-spot welding - 91 dB/A, multispot welding on welding stand - 90-92 dB/A; forming (compressed air cassette cleaning - 88 dB/A,

concrete sealing on vibro platforms - 115 dB/A, on vibro tables up to 126 dB/A, manual vibrators - 67-95 dB/A; of cranes - 82-85 dB/A. The noise is variable, with sharply fluctuations in the sound pressure, including impulse noise. Its frequency characteristic is predominantly low and medium frequency, with vibro tables - it also includes high frequencies. Vibrating means used for sealing concrete, are the main intensive source of **general and local vibrations.** Of vibroplatforms and especially vibro-tables, there are several times exceeding the norms in medium and high frequency range (16-63 Hz). Hand vibrators, depending on the model, generate: the "internal" (so called "shaped") - medium frequency vibrations (80 Hz) , "surfaced" - high - frequency local vibrations The exposure of workers to noise and vibrations when operating vibro tables and vibrometers is about 26-30% of the working time and 48-54% when working with handheld vibrators. Another source of general and local vibrations are the cranes - vibrations on the floor, seat and controls. At all stages of the process the **microclimate** is unfavorable. When making the fittings, the microclimate is most often dynamic and cooling. In the molding of the reinforced concrete products, regardless of the method of the heat treatment used, the relative humidity of the air is increased to 90-95%, and during the warmer year period the air temperature is up to 28-34 ° C. The greatest is the humidity and the heat intake when the reinforced concrete elements are removed from the molds, in the steam chambers. The production of **dust** is divided into 3 technological stages - production of fittings, molding and finishing operations. A metallic powder (disintegration aerosol) is released during the production of fittings in the unwinding, straightening and mechanical cleaning of the ferrous iron rust - iron oxide. Much of the particulate matter has significant dimensions (over 10 μm). The condensing aerosol (smoke) in the electro-welding process (is also used in the preparation of the fittings) - metallic aerosols of iron, manganese, cadmium and zinc oxides, chromium, nickel and fluorides, finely dispersed (less than 1 μm). At forming, dust is released in a "mold cleaning" operation at the highest concentrations exceeding the MAC. The dust of the composition is mineral, mainly cement. Contains also silicates and free crystalline SiO_2. The dust is rough or finely dispersed depending on the cleaning method. Similar is dusting in finishing works - knocking, rough grinding. In case of patting, gypsum powder is also released.

Harmful **chemicals** are released during the production of reinforcing nets and constructions - in the case of arc welding and spot welding. In workload of welded mills, CO (20-35 $mg.m^{-3}$) and nitrogen oxides (1.5-5 $mg.m^{-3}$) are determined at higher concentrations. In molding, chemical substances are released during the treatment of heated forms with antiadhesive emulsions, especially at aerosol application (injection forming) - petroleum aerosols, vapours of kerosene (300-450 $mg.m^{-3}$) and alkaline aerosols (sodium or potassium base) - 1.5-2.5 $mg.m^{-3}$. When used with additives in concrete - synthetic resins, vapors are released from monomers, plasticizers and hardeners, but usually in low concentrations below the MAC.

Work process. The main professions in the industry are steel fitters, arc welders, crane operators, formworkers-concretors, steamers, patters. The physical workload for these professions is average, with the exception of crane operators, where it is mild. The working posture is imperative, often forced, for the crane operators - who are always seated. The occupational activity of all professions includes many manual operations repeated over and over for the shift. The rhythm of labour is free, does not require great speed, accuracy, high qualification. Occupational risk exists from industrial traumas, incl. steam burning. Nervous-sensory and nerve-mental intension is only present in automatic lines operators in modern productions and crane operators.

Illnesses of workers. In the structure of the general morbidity of workers, with very high frequency and loss of work are acute respiratory diseases, diseases of the LS, PNS and traumatism. Occupational morbidity is mainly caused by injuries caused by the effects of noise and vibrations. In formworkers - concretors, the high-frequency professional deafness is registered. For vibratory tables workers, professional deafness is observed in the first 2-3 years of their work experience. Vibrational disease is found primarily in hand-held vibrators. In crane operators, as a professional disease of general and local vibrations of higher frequency, vegetative polyneuropathy occurs. In formworkers - concretors and workers performing finishing work, incl. patters, professional dermatoses, cases with bronchial asthma, chronic bronchitis, cement pneumoconiosis, diseases of the LS and PNS, incl. vegetative polyneuropathy.

Prophylactic measures. Hygienic activities are focused on mechanization and automation of the technological process, protection against noise and vibrations, including technical means and PPE, isolation of vibro tables, prohibition of stepping on formwork and work in vibration; use of vibration-free concrete consolidation agents; sealing of heat treatment equipment; ensuring efficient ventilation of production premises; medical-prophylactic events - preliminary and periodical medical examinations; appropriate work and rest regime, etc.

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B. Stefanov

17.8 LABOUR HYGIENE WHEN WORKING WITH COMPUTER EQUIPMENT

The microcomputer is used by a variety of professional groups. As a fast and intelligent tool, it is an integral part of everyday life not only of professionals (programmers, computing center operators, etc.), but also of editors, journalists, accountants, bank and library staff, researchers, dispatchers, production processes, operators secretaries and many others.

Regardless of its main subject, a microcomputer worker performs work whose specificity is determined by the specifics of the task being performed, the organization of the workplace and the factors of the working environment. Of particular importance are the type of components of the computer configuration used.

Working environment conditions

Cathode ray tube displays emit electromagnetic waves with a wide spectrum of X-ray, ultraviolet, visible and infrared rays, and RF wave-fields (EMF). The majority of the latter (over 70%) have a frequency below 300 kHz.

In the kinescope the electrons accelerate with high voltage, which, depending on the model, is from 10 to 25 kV. This results in the appearance of mild **x-ray radiation**. Its intensity is too small and it almost completely holds off the screen glass.

The image on the video monitor screen is formed by an electron beam. Various magnetic and electrostatic systems serve to focus and divert it. The beam is controlled by 15 to 22 kHz current, resulting in **radio frequency electric and magnetic fields**. The latter also arise from the horizontal diversion system and its power generator.

The electromagnetic radiation from the displays is broadband - from 10 kHz to 26 GHz. At 10 cm from the screens, the intensity of EMF is within the hygienic norm (below 50 V/m), at 30 cm the radiation is below 6.5 V/m. However, maximum EMF values are measured at the back of the displays, which should be taken into account when disposing of them in a room.

There is evidence that the video monitor's kinescope also emits **ultraviolet rays**. Their intensity is less than 1 W/m^2, which is significantly below the norm (maximum permissible value up to 10 W/m^2).

At work, the microcomputer also emits **infrared radiation**. Its main source is the heating of the cathode cathode. However, the values of this factor are also too small - the maximum is less than 1 W/m^2 with a norm to 10 W/m^2.

In order to accelerate the flow of electrons in the video monitor's kinescope, an electrode with an extremely high potential is used. It becomes a source of **electrostatic field**. When the electron ray (beam) enters the luminescence layer on the screen, electrostatic charges are also created. Their presence can be felt even when you touch the screen. A source of static electricity is not only the display, but also the electrified synthetic materials that abound the modern working environment (carpets, plastic coatings, including synthetic fabrics clothes).

In a number of cases, the adverse effect of computerized workplaces is **noise**. A source of steady noise is the fan of the power supply unit and the processor. Along with this, noise also generates the working printer or plotter. Room noise also comes from outside sources. The measured noise values of the printers are within 40-51 dB/A. According to our hygiene regulations, the noise level for service of computer equipment should not exceed 55 dB/A.

Psychophysiological characteristics of labour

Work with the use of a microcomputer is not uniform. Depending on the task, it ranges from monotonous performers (input of large arrays of data) to intellectual creative work (programmers). In general, it is characterized by:

a) processing of significant volumes of information requiring intension and attention;

b) significant load on the psychosensory sphere, primarily the visual analyzer;

c) nervous emotional stress resulting from the responsibility of the work in proportion to the importance of the information being processed;

d) a high degree of monotony with frequent, accurate, uniform, short-term movements at high rates and repetitions (in some cases);

e) a fixed seating position with a load of mainly the small muscles of the upper limbs and a forced position of the head, neck and upper limbs.

The dialogue with the computer takes place through a series of cycles, each of which involves three main activities: 1) entering commands or data, 2) monitoring the screen, and 3) reacting messages.

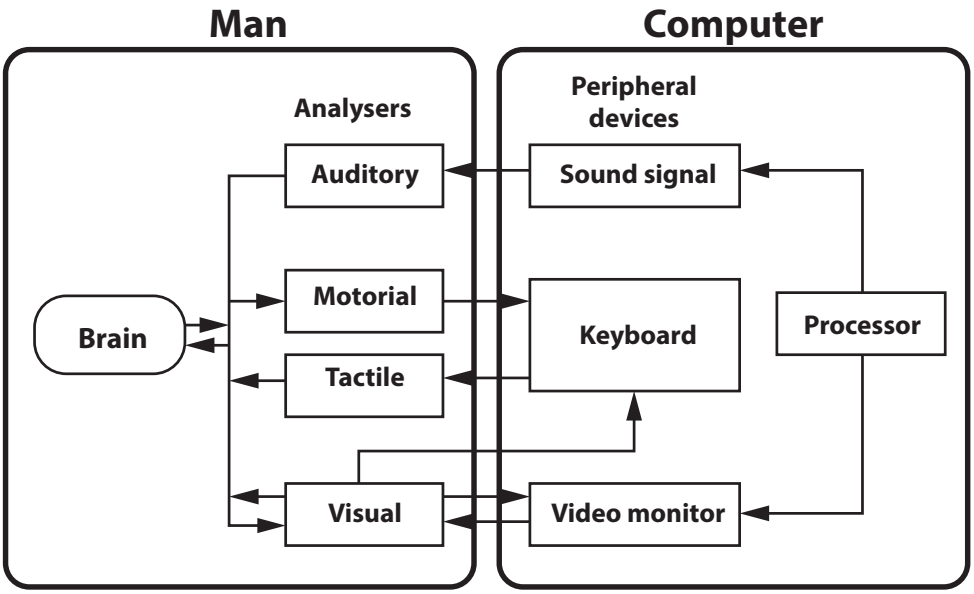

Fig. 1. Scheme of human-computer interaction

In a more developed form, the human-computer interaction is represented in the scheme of **Fig. 1.**

The specificity of the microcomputer is largely determined by the fact that there are consistently observed basic objects with different characteristics: documentation, keyboard and screen of the video monitor; in some cases even a printer, a scanner, and more.

Complaints with long-term computer work arise not only from screen observation but also from the awkward working posture. Working with a microcomputer is characterized by a fixed sitting position with the head, neck, and upper limb being forced. Prolonged work in such a posture is a prerequisite for limited motor activity (hypokinesia).

The working movements of the computer, respectively with keyboard and "mouse", are mostly of Class I and Class II - active fingers and hands. Most busy are so-called big toes - index finger and middle finger. Relatively rarely there are active movements of forearms and armpits (III and IV grade). Small muscle strength and a small amplitude of movement are required. When entering large data sets, the high speed of performing well-coordinated stereotypical movements becomes necessary.

Professional computer work is associated with significant neuro-psychical and intellectual effort due to the high signal density (large number of signals/hour) that requires attention and memory intension.

Sometimes it is necessary to work under the conditions of an acute time-pressure, which is a prerequisite, along with responsibility, for time-stress. It too often leads to more pronounced changes in the body, particularly in the central nervous system than in physical work.

Impact of display work on visual activity. Diseases.

One of the most characteristic effects on the body when working with video monitors is **visual fatigue**. It is similar to the one on long-term TV viewing.

The most common symptoms in display monitoring are: burning eyes, nausea, tearing, redness, blurring, smearing and merging of letters, feeling of pressure in eyeballs. Eyelid complaints are usually combined with headaches, most commonly in the head and forehead, to a lesser extent in the temporal area. Sometimes there is a feeling of nausea and dizziness.

Symptom of visual overload is also the decrease in the ability of the eyes to focus on the subject observed and to retain the fixation. Binocular vision deteriorates with the appearance of a double image (diplopia) and blurring of the image.

Along with subjective complaints, longer work with video display worsens practically all visual functions: significantly reduces accommodation, convergence, contrast and differentiation sensitivity, and the clear vision stability.

A connection between the appearance of permanent eye changes and display work is not found. Concerns about the occurrence of cataracts and myopia when working with a video monitor are not substantiated by sufficient facts.

In addition to general and visual fatigue, local dynamic and static fatigue also develops in the operator's body. The first is the work with the keyboard and the second because of the fixed working posture.

Incorrect organization of the work process and

the workplace is one of the prerequisites for the occurrence of possible **occupational diseases**. Prolonged static intension combined with a fixed posture and rapid, often repetitive wrists and fingers can cause damage to the type of tendovaginitis.

The need to carry out many frequent and rapid stereotypical movements, e.g. when entering large arrays of data, leads to dynamic intension on the fingers. As a consequence, coordination neuroses, neuralgia and polyneuropathy, microtraumopathy of tendons, muscles and joints may occur, which may subsequently cause chronic pain syndrome, that is increased in activity.

Conditions resulting from physical overloading of muscles and joints during prolonged repetitive movements are known under the name of Repetitive Strain Injury (RSI) or Carpal Tunnel Syndrome (CTS). They are characterized primarily by discomfort or pain in the muscles and/or joints. Local manifestations are pain, inflammation, edema, soreness, numbness, impaired coordination of movements. There is also fatigue, muscle weakness, back pain, neck and shoulders.

Long staying in a fixed working position with head and body inclination is associated with the static load on the muscles of the neck area, shoulder belt and waist area. In addition to a faster fatigue, the development of myofibrosis in these areas is also possible.

Prevention of work

Prevention includes various organizational, planning, technical, technological, and oth. events. The main ones are:

- professional selection;
- optimization of the environment parameters;
- proper workplace organization and work position;
- compliance with the ergonomic requirements for hardware and psychophysiological requirements for the software;
- optimizing the visual activity
- introducing appropriate work and rest regime.

The illumination of the workplace must be based on the nature of the work performed and the type of screen. It is recommended to reduce the ambient light when working with a darker screen. Under negative contrast (bright characters on a dark screen) it should be at least 300 lx, and with positive contrast - 500 lx.

The window should not fall into the field of view of the operator. In some cases it is necessary to place windows with vertical lamellae, and on the computer - a sunshade.

The luminaires should be located parallel to the windows and have a 30° protective angle. The light (natural or artificial) should always fall sideways, if possible to the left of the workplace.

Lighting should not dazzle the operator. Large reflective surfaces in the room should be kept to a minimum. Bright objects and surfaces are not smooth, but matte. The same applies to the surface of the display screen.

The ratio of screen **brightness**, keypad and work surface of the table significantly influences work efficiency. If it is irrational, visual fatigue occurs faster. The recommended proportion in our country is 10: 3: 1 or 5: 2: 1.

To limit the **noise** level, it is recommended that the printer have shutters and silencers. If there are several printers, they should be placed in an adjacent room if possible. This does not apply to low-noise (jet and laser) printers.

To reduce the **static electric field**, it is recommended to increase the relative humidity of the room, correct selection of floor coverings and clothing for workers with a minimum content of synthetic materials.

The work area for microcomputers typically includes several areas: keyboard and mouse, video monitor, input text or other information, scanner, printer, external modem, and more.

Areas that are more closely monitored (keyboard, screen, input text, etc.) should be approximately the same distance from the eyes. This greatly reduces the need for rapid accommodation adaptation and may generally reduce visual fatigue.

The observed object is best viewed when it is within the optimum **viewing angle**. In a horizontal plane, it is up to 40° left and right to the line of sight for volume images and up to 60° for flat. In a horizontal plane, this angle is: optimal - from 0° to the line of sight to the 30° downward, permissible - from 30° up to 40° downwards.

The height of the **work surface** (table, desk, panel) and seat height should provide an approximately horizontal position of the forearms. It is desirable that the height of the table can be adjusted from 680 to 760 mm. If it is unregulated, its recommended height is 720 mm **(Figure 2)**.

Work chairs must be adjustable. It is recommended that the height of the seat can be changed from 40 to 50 cm above the floor. The backrest should provide support for the spine in the area of the waist - resting in the area of the lumbar vertebrae around a palm above the seat. This is possible at a height of its median to the seat of 17 to 25 cm.

In some cases it is advisable to provide support for the legs. The most ergonomic dimensions for such a stand are: 45 cm wide, 35 cm deep, 0 to 15 cm high, 0 to 30° slope.

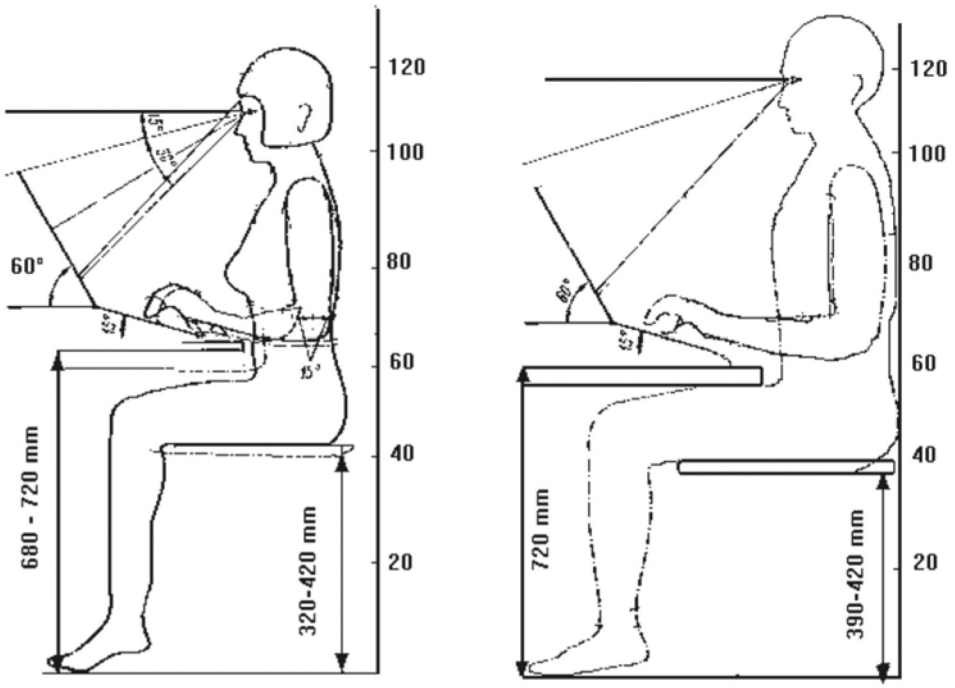

Fig. 2. Working furniture for use with a computer

When there are several computers in one room, it is of no doubt what their mutual placement will be. The ordering of workplaces one behind the other is not the most appropriate because maximum values of electromagnetic radiation (albeit substandard) are measured to the back of the displays. In order to reduce the potential occupational risk, it is advisable to work respectively computers are in line with one another or symmetrical about one axis **(Figure 3)**.

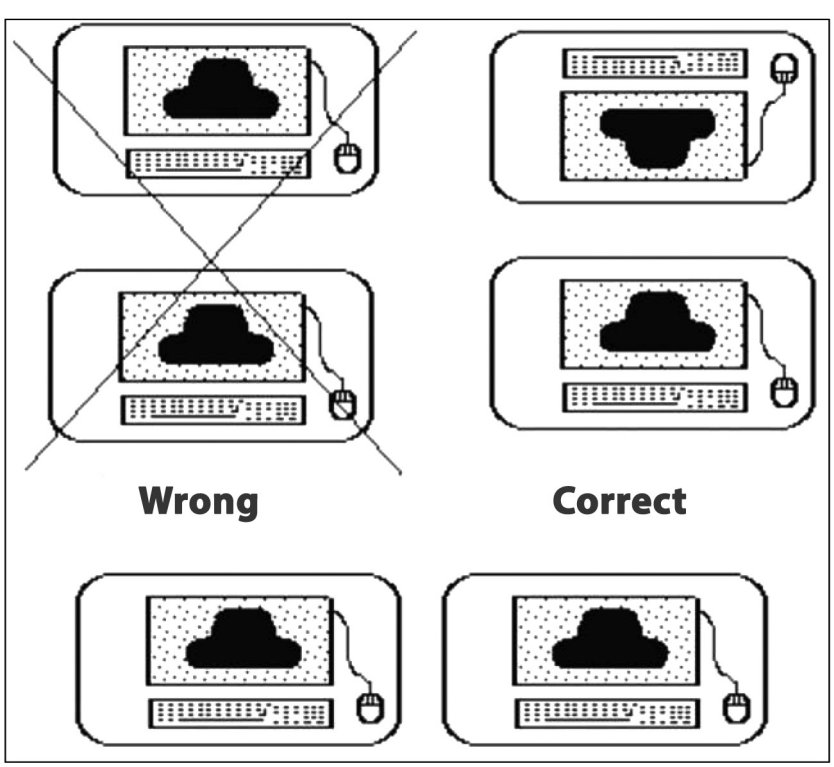

Fig. 3. Mutual deployment of multiple computers

A basic condition for avoiding disruption and damage to health is that all components of the computer configuration meet the ergonomic requirements.

For optimum visual performance, good **video quality** is required: with minimal flicker, higher resolution, higher separation rate, more raster lines and lower-level of radiation(Non Glare, Non Interlaced, Low Emission, Low Radiation).

Preferred is the **positive contrast** of the screen - dark characters on a light background, where glare and reflections are smaller than negative contrast, and the adaptation of the eyes to different brightness is easier.

An important indicator of display quality is the frequency of horizontal beam deflection. To achieve a higher resolution (e.g., 1025 x 768 dots), a non-interlaced, non-skipping lines and associated image flicker is required. This requires a frequency of about 50 Hz. If a higher resolution graphics adapter is used, ergonomic positions require a frequency greater than 75 Hz.

To customize the screen, it is desirable to have a "sponge" stand that allows the monitor to be adjusted horizontally and vertically. Optimal for vision is the front vertical (or sloped back to 15°) screen position.

To avoid the intension of the muscles of the eyes, neck and back, the horizontal line of sight should fall at the top of the screen. The recommended eye distance for different authors is 400 to 700 mm.

To prevent visual fatigue, it is necessary during the breaks to make a light massage of the eyeball with closed lids. This has a favorable effect on the blood supply to the eyes and the eye pressure. To reduce accommodation fatigue, it is desirable for the eye to move away for a short time to more distant objects - more than 2 m from the eyes.

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17.9 ELECTRICITY PRODUCTION

In the modern world, electric power production is in the base of economic life. In Bulgaria, this sector is mainly represented by the production of electricity in the thermal power plants (TPP), where about 2/3 of the electricity is produced and by the nuclear power plant (Nuclear Power Plant), which produces 1/3 of the electricity. A small part, about 10%, is produced in hydropower plants (HPP). Unconventional ways of production electricity, such as wind, solar radiation or biomass, are little included for our country, and in other countries are also at the experimental stage or with limited, local and partial power supply.

17.9.1. THERMAL POWER PLANT

The TPPs operate with liquid, solid fuel or gas. In our country the most common solid fuel is used, such as coal from the Marish coal-mining basin, Bobov dol or imported. The TPP consists of the following main sectors/facilities: 1. Coal/oil sector; 2. Boiler-turbine sector (power equipments).

1. Working conditions.

a). Factors of the work environment. Exposure to coal dust can lead to pneumoconiosis and chronic pulmonary bronchitis. In spent fuel (periodic cleaning of furnaces), due to the burning of carbon and organic materials, silica in the dust increases relatively.

Noise is another unfavorable factor in the working environment in the thermal power plant. Generally, it can be characterized as constant "white" noise with considerable intensity. At most work places in "Boiler" and "Turbine" sectors, the noise exceeds 85 dB/A. Abnormal noise levels are also found in a number of workplaces and in the transport and repair mechanics workshops.

A large percentage of workers work in an environment of unfavorable microclimate. Many of the workers in the process of coal feeding and slag purification work all year round in the open, in the conditions of overheating microclimate during the summer and mosaic or overcooling in winter. In the machine rooms of "Boiler" and "Turbine" and in the slag removal, the air temperature is up to and above 40 ° C during the warm seasons.

In the process of combustion of coal in the working environment, carbon monoxide, sulfur and nitrogen gases are released. Their quantity varies widely depending on the trouble free of the technological and purification facilities (feeders, electro filters, etc.) and can exceed the MACs several times. Workers from the "Chemical Cleaning" and "Reagent" departments are in contact with hydrogen chloride, sulfuric acid and hydrochloric acid, ammonia, benzene, hydrocarbons, etc., whose concentrations also, although rarely, can exceed the limit value. In the welding activities, the gas welders and other repair staff are also exposed to various metal aerosols, ultraviolet and infrared radiation.

Other widespread environment factors in power generation and power distribution are the electric and magnetic fields with industrial frequency (50-60 Hz). They are emitted in open distribution devices, from indoor distribution devices (IDD), and from high-voltage power transmission lines themselves to intermediate transformer stations or to consumers. Intensity of the electric field is directly related to the voltage (V), while the magnetic induction is higher at a higher current (A). The two electromagnetic field components (electric and magnetic) are rapidly diminishing by moving away from the power lines, while in the ODD and the IDD their intensities depend on the location of the equipment.

b). Factors of the labour process. The uninterrupted process inevitably necessitates a shift operating regime for all operational and part of the service staff. Working on two extended 12-hour shifts - day and night, the rotation takes 2 consecutive shifts and provides longer breaks and frequent breaks between shifts (24 or 36 hours). Such mode is preferred because it reduces transport time and the adverse effect of night work, which is only in two consecutive nights.

In most work places, labour can be characterized as labour with moderate physical workload, and that of the operating staff has marked nervous sensory and mental intensions.

2. Main professions.

For operational staff, the main professions are those of boiler operators, turbine operators, fuel transport belt operators and switchboard operators. Their workplaces are mainly in the control rooms but they also carry out checks on the control devices located in the machine rooms and on the course of the equipment. Their work is characterized as a

high-responsibility nervous-sensory. In normal operation, the information on the indicator devices is rarely changed. The rare occurrence of relevant signals leads to an information deficit that, along with the monotonic noise (around 65 dB / A) and reduced motor activity, creates a condition to develop unpleasant mental conditions such as monotony and annoyance, and to reduce the human body's vigilance and reliability in man-machine system. Operator fatigue occurs as a result of voluntary efforts to maintain activity when all other factors lead to suppression of activation processes.

A large group of professions (fitters, welders, electricians, craneists, etc.) can be joined as repair and maintenance personnel. This group has been exposed for a long time to the harmful factors of the work environment. Work is usually moderately in terms of dynamic physical loads, but it involves considerable static effort.

3. Health Effect.

Occupational diseases can occur as a result of dust exposure (chronic bronchitis, anthracosis, mixed type pneumoconiosis). High noise levels may be the cause of professional deafness development.

Overheating microclimate and shifting work favor the development of gastritis and stomach ulcer. The extra-aural impact of noise, shift work and a number of unfavorable work-related psychosocial factors increase the risk of cardiovascular disease and dyslipidemia.

Prophylactic events should be addressed to technical improvements (eg. reduction of noise by shielding, reduction of thermal radiation by shielding), and other medical-prophylactic measures. The latter first require regular prophylactic examinations aimed at uncovering yet unmanifest clinicaly professional pathology - early functional changes. Emergency workers need to be expelled in a time their unhealthy working conditions (eg. patients with ulcer disease at least during periods of exacerbation do not work at night shift). Another group of medically-prophylactic events is aimed at limiting the specific impact of adverse factors on the work environment, eg. a suitable nutrition and drink regime for those working in a overheating microclimate, the provision of antiphonics to workers exposed to abnormal noise levels, dust masks for workers exposed to dust.

17.9.2. HYDROELECTRIC POWER PLANTS

From the ecological and economic point of view, HPPs are the preferred source of electricity. They produce the cheapest electricity and do not pollute the environment. An adverse ecological effect may exist with respect to ecosystem damage when water resources are diverted to artificial reservoirs and canals.

In the HPP, unlike TPP, there are no such factors as dust and overheating microclimate. The main adverse factor of the working environment remains noise. In turbine halls, it exceeds the ALL-85 dB/A. In the control room the noise level is within the hygienic norm - 65 db/A. Secondly, electromagnetic fields, whose intensity depends on the location of the distributiv devices, transformers, etc.

In a hydropower plant where the turbine and command room are underground, the artificial light problems are not only in terms of their physical and ergonomic characteristics but most often with the absence of changing illumination as a psychological equivalent in the dark and the light. The latter is important for the activation processes of the operators. In the halls underground there may be an increase in the natural background radiation, the intensity of which varies depending on the geochemical composition of the rock/earth masses.

Major professions in HPP are those of turbine operators, maintenance and repair staff.

17.9.3. NUCLEAR POWER PLANTS

Electricity from the NPP is cheaper than the TPP at an average of 1.2 to 1.9 times (in Bulgaria it is cheaper 2.3 times). Logically, here are a number of pros and cons, such as: presence of local or imported raw materials; environmental pollution; removal of technological waste; operational safety; providing qualified personnel; sociopsychological feelings of the population and others.

Since their creation in the late 1950s, nuclear power plants have not undergone any significant change in terms of principle. Today, in the world, power plants are operated from the following main types of reactors: light water reactors with pressurized water; light water type "Boiler"; light-water reactors with graphite neutron retarders; reactors with heavy water under pressure; heavy-water gas-cooled reactors. Reactors from Kozloduy NPP are water-water reactors - WWR-440 model B 230 and WWR 1000 - model 320. They are light water with pressure water, working with slow (thermal) neutrons.

How does the reactor ("Boiler") work? In the NPP turbines (as well as in thermal power plants), the kinetic energy of high-pressure hot steam rotates the generator rotor, generating electrical energy. Working turbine steam pass into a condenser where it delivers its residual heat and liquefies (this also produces secondary steam for technological purposes). The liquefied steam (water) is pushed by pumps to the

steam generator, which evaporates as a result of its contact with pipes in which circulating heated water up to 320 ° (the so-called "primary circuit") - this is taken away by the nuclear reaction in the reactor Heat that flows through the walls of the pipes, in so-called "second circuuit". It is important for radioprotection that in both circuits there is independent water flow from each other.

The nuclear reactor is a thick-walled metal cylinder made of high-strength steel. In his bottom, in so-called "Scoop" locates: 1. cassettes containing several dozens of fuel elements (zirconium alloy tubes) with nuclear fuel (a mixture of ^{235}U and ^{233}U in an appropriate ratio). 2. control elements (rods similar to the fuel elements but made of boron steel) which are arranged in the form of a honeycomb; 3. neutron retardants - light hydrogen is used in the form of water. In WWR reactors, this water is used both as a cooler and as a heat carrier.

The Kozloduy NPP has 4 power units with the WWR-440 reactor type (closed in 2007) and two WWR-1000.

1. Working conditions.

a). Factors of the work environment. Most attention and control deserves the ionizing radiation factor. The operation of the Kozloduy NPP during the whole operation period has not shown an impact on the radiation parameters of the surrounding objects and the environment. Relatively higher doses of radioactive radiation are recorded there in non-servicing (with rarely human presence - only in repairs and exploitation abnormalities) premises, and in others they are far below the allowable. The annual dose limit (ADL) for irradiation of workers in the environment of ionizing radiation (including NPP personnel) is 50 mSv. According to the Kozloduy NPP dosimetric service data from 1986 until now the radiation above 50 mSv has received only 48 workers from a total of 2000 controlled, which dose is compensated for the next 5-10 years (according to radiation protection requirements). The average annual individual dose there for the entire lifetime is in the order of 9-10 mSv. The main, so-called "Dose expense" was realized during the repairs of the first four blocks (WWR-440).

Noise is a widespread unfavorable factor in the work environment. Generators of excessive noise levels (over 85 dB/A (turbines, pumps, compressors, etc.) In the power control rooms, the noise is below 65 dB/A.

The microclimate is overheating in the halls of the "Turbine" shop where during summer the air temperature reaches 36-38 ° C, but there workplaces are non-stationary.

Other factors are the electric and magnetic fields of the generated 50 Hz electric current.

b) Factors of the labour process. NPP is complicated, requiring modern management energy complex, which operates 24 hours a day.

The most responsible place in the plant is the operating staff who controls the operation of the reactors and the turbines, the engineers responsible for the entirety operation, the managers of the power units, the shifts, etc., Their work is classified as labour with very high neurosensory and neuro-emotional intension, and the operational working time is over 95%, which shows an extremely intense workload. Reactor parameters are also monitored, and some (which constantly monitored and are most important) being call actively on the control system displays. The work requires a very frequent audio connection (vertical and horizontal) with different groups and process managers. It imposes great demands on the memory, the processes of attention, thinking, requires the consideration of many facts. The speed of technological processes often requires responsible decisions to be made in a time deficiency. This, together with the extremely high responsibility, determines the high stressful character of the work.

Another large group of workers are those from the repair and maintenance staff. Unlike similar professions from other industry branches, there is required a lot of knowledge, precision and accuracy to perform the job tasks.

It works in 3 shifts of 8 hours, the total working hours balance for the month being calculated based on a 6-hour working day.

2. Health effect.

As a result of the radiation impact, stochastic and non-stochastic effects are possible. The stochastic effects are linearly dependent on the absorbed dose and are considered to be non-threshold - these are the damage to the somatic and genital calls genome. The dose/effect relationship is expressed in the statistical probability of increasing the prevalence of cancer in the population.

In the case of non-stochastic effects, injuries (diseases) are manifested when the dose is exceeded. Increased dose increases the severity of the disease. Non-stochastic effects are acute and chronic radiation illness, radiation skin burns, eye cataracts, embryonic damage.

High nervous-emotional intension, shift and night work increase the risk of cardiovascular disease, gastritis, stomach ulcer, and other psychosomatic diseases.

Personnel working in the strict regime zone (zone A) undergo special medical surveillance, periodic medical examinations are directed to the search for

early stochastic and non-stochastic effects. These persons are dispensary and the medical information is stored (and compared in subsequent inspections) up to 30 years after leaving the job. Persons over age of 18 are admitted to work; which have no haematic disease, organic and systemic diseases of the central and peripheral nervous system, precancerous diseases, chronic diseases of the gastrointestinal tract, diseases of the liver and kidneys and other diseases attacking the critical organs.

The dosimetric control of the personnel is carried out by a special service of the power plant. Zone A workers are subject to personal control by individual dosimeters for external exposure. Strict control of contamination of working surfaces, working clothes, skin, air, and transport equipment is carried out.

For workers in category B work, on controlling the dose rate of external radiation and the concentration of radionuclides in the air.

In order to maintain a high level of work capacity and security at work, effective professional selection of staff, optimal working and rest regime, providing conditions for high motivation for work and constant qualification are required.

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Iv. Paunov

17.10 TRANSPORT

Transport hygiene is a complex prophylactic medical discipline that combines the hygiene of rail, water, road and air transport, as well as the hygiene of labour in road construction.

In the structure of stationary transport sites (factories for new construction, repair plants, landfills, garages, hangars, etc.) are usually included the corresponding departments of the machine-building industry. The working conditions in them essentially are different from those in the machine-building plants.

The implementation of an uninterrupted 24-hour organization to ensure safe movement is carried out by several main categories of personnel: drivers who directly manage the means of transport (railway drivers, assistant railway drivers, captains, ship pilots, car/bus/truck drivers, aviators, etc.); personnel providing remote control or route for the movement of transport means (motion managers, air traffic controllers, railway-, airline-, naval dispatchers, radio operators, etc.); personnel servicing vehicles, passengers and freight during the movement (railway assistant drivers, conductors, chieftains, stewardeses, stewards, cabin attendant, etc.) and during their stay (control wagons, maneuvers, signalmans, flight mechanics, luggage and ticket cashiers, repairers of various modes of transport, etc.); staff who are currently supporting the means of transport (repair workers in rail, road and water transport); staff for electrification in railway transport, new construction of railway tracks, sea and river canals, roads, tunnels, loading and unloading workers, etc.

17.10.1. WATER TRANSPORT

Water transport by origin is the oldest mode of transport. The main professional groups in it are the crews of the ships. The latter, depending on their purpose, are: passenger - motor, underwater wings, etc.; cargo - bulk (ore, coal, grain, etc.), liquid or gaseous (tankers), container vessels, etc., and special vessels - ferries, thrusters, towboats, to maintain the cleanliness of port and marine areas (oil-collector for seaport harbors and for the open-seas, solid waste collectors, motor and non-power feed tankers, barragers, etc.), fishing and processing fish vessels (trawlers), construction and technical fleet's (CTF) vessels - supporting and cleaning the depth and bottom of the canals, new construction, extraction of inert materials, etc. - dredgers, suckers, floating cranes, etc.

The main professional groups are: Group I - for the ship-navigation and its direct service. It is subdivided into a navigator command (involved in ship navigation) - captains mates, captain's, pilots and helmsmans and deck command - radiotelegraphist, boatswain and sailors; Group II - service engine room, electrical equipment, navigation and refrigeration installations and other auxiliary mechanisms and aggregates (machine command) - chief engineer, electrician-, refrigerator- and repair engineers and technicians, machine boatswain, motorists, etc .; Group III - ship administration, household and other service staff (housekeeping command): providing the nutrition of the ship - host, chef, baker, confectioner, cameramen, loundresser; on passenger ships - hotel and restaurant managers, waiters, barmans buffet attachment, telephonist, librarian, doctor, dentist, nurse and others.

In the water transport, besides the floating staff, there are also coastal positions - coastal radio operators, dispatchers, light-housekeeper, ship repair workers, dockers, mechanics of the ports, etc.

Microclimate. The meteo factor of the ships is one of the most dynamic and varied in intensity of physical factor that constantly affects their crews. In different areas of sailing it is different and is determined by climate and weather.

The microclimate of open decks wholly depends on the macro climate, but they are not identical. Differences exist especially during warmer periods and intense solar radiation that heats ship's metal deck and superstructure. The latters give heat, whereby the air temperature on the deck rises by an average of 2 ° C. Thermal radiation also increases. In winter, conversely, cold metal structures create conditions for negative radiation balance.

Modern systems for heating, ventilation and air conditioning of ships throughout the year create acceptable levels of microclimate in the ship's operating and living spaces, of exceptionthe engine room, the kitchen unit, and some other rooms with heat sources where over-values are measured, reaching 40-42°C. The creation of central control panels (CCPs)

and the remote control of marine engines minimizes the exposure of sailors to similar temperatures - only for repair works in the engine-boiler section (EBS).

A characteristic feature of the microclimate in water transport is the ability of crews to change climate zones several times during one trip, which brings increased requirements for thermo-regulation of the body.

Noise. Noise in ships is supported by a large number of sources, but the main engines, auxiliary mechanisms (diesel generators, compressors, pumps, etc.) in the EBS, the screw propellers, the heating, ventilation and air conditioning systems, some household facilities, and in the specialized ships at the machinery and equipment specific to the ship. Noise in ships, apart from airborne, also spreads indirectly as a structural noise. Another feature is that it has effect not only during the shift, but also during leisure, i.e. around-the-clock, throughout the trip.

Maximum values are measured in EBS - 101-102 dB/A (in thrust, towboats and trawlers); in ships supporting seawater purity - 96-104 dB/A; in ferries and cargo ships - 107-108 dB/A; to 118-120 dB/A in underwater wingships.

In other work and living areas, the noise level depends on the type, and size of the ship, the location of the superstructure over the EBS, the availability of its own sources of noise, and so on. - 84-86 dB/A in the radio station cabins, 54-58 dB/A captain's bridge, 43-56 dB/A crew saloons, 34-52 dB/A sleeping rooms, and so on.

Infrasound. In underwater wing ships, the infrasound rate is on average 93 dB. Opening the side windows increases its level to 94-104 dB. In the engine compartments of this type of vessel, the infrasound is 86-96 dB (stopping ships) to 86-103 dB (during movement).

In the EBS on sea vessels, the infrasound intensity is from 64-88 dB (on the quay) to 82-114 dB (in motion). In the living premises of these ships, the infrasound level is 77-97 dB. When moving ships in a ruffle sea (Beaufort number 6), the level of infrasound increases sharply in all rooms and reaches 124 dB.

Vibration. Numerous external and internal sources generate vibrations of varying frequency and amplitude - low frequencies with a long period (swinging) from the impact of water masses, periodic - from screw propeller and basic - from the operation of engines and other aggregates of ships.

Measurement results show that for most ships the vibration velocity of general vibrations is over-norms, with a peak usually at 8.0, 16.0 and 31.5 Hz - 0.2 to 1.0 $m.s^{-1}.10^{-2}$.

Local vibrations, with over-norms values, are typical of ship repair using hand-held pneumatic and power tools - drills, electric flexes, riveting guns, and more.

In all ships, looking at their vibrational characteristics, it is not possible to ignore the swinging of the ships (vertically, laterally and longitudinally), resulting in irritation of the vestibular apparatus and the occurrence of marine sickness.

Lighting. The natural lighting in the ships is made of illuminators (phyllins), side windows and sky-lights.

In the EBS, CCPs, technical workshops and compartments, holds, etc., located under the waterline all the day, are artificial lighting, which is fire- and explosion safety.

Chemical Dangers. Originally they are external (the background concentrations of the harmful substances contained in the atmospheric air of the area of residence of the ship) and internal ones. Also important here are the limited spaces of the working and household spaces in the ships, allowing for a short time their saturation with relatively high concentrations of toxic agents.

Internal sources of chemical damages are:

1. From the energy sources and the lubricating oils (aromatic hydrocarbons, sulfur compounds, etc.) and their waste products (exhaust gases) - CO, CO_2, NO, SO_2, H_2S, soot, carcinogens, incl. 3-4 benzpyrene and the like. They are predominantly found in the EBS, but can not be ruled out in the adjacent rooms. In 20% of the EBS samples, average petrol vapor concentrations about 450.0 $mg.m^{-3}$, unsaturated hydrocarbons - 220.0 $mg.m^{-3}$, CO - 30.0 $mg.m^{-3}$ are measured. Average concentrations of petrol vapor 100-150 $mg.m^{-3}$, unsaturated hydrocarbons - 50-70 $mg.m^{-3}$ and CO - 7-12 $mg.m^{-3}$ are recorded in the adjacent premises.

2. Harmures emitted by different facilities, installations, utilities and people: polymeric materials (various hydrocarbons, CO, phenol, formaldehyde, ammonia, styrene, polymethyl methacrylate, chlorvinyl, etc.); paints, lacquers, enamels, etc., used in the construction, repair and maintenance of metal and wood surfaces; refrigeration installations during repairs or accidents (ammonia, chloromethyl, freons, etc.).

3. Hazards arising from the transported solid, liquid or gaseous cargo - e.g. during loading and unloading activities of tankers in pumping compartments, concentrations of hydrocarbons up to 3000 $mg.m^{-3}$ are recorded, and in enclosed spaces up to 200-300 $mg.m^{-3}$.

Dust. The crews of bulk carriers (coal, ore, grain, etc.) are exposed to dust during port loading operations and their deck commands additionally and in transit - in hand-cleansing the holds and the deck.

Maximum total powder concentrations reached 300.0 mg.m^{-3}. In the living quarters of these ships concentrations of 1.0 to 62.0 mg.m^{-3} are measured during loading operations. The powder is mostly finely dispersed (less than 5 μm) with a free crystalline silica content of up to 2%.

Sources of pollution can also be ship machines and equipment that emit powder dust - smoke, soot and others.

The painters (when removing the rust and the old paint) and the electric welders are exposed by the dust.

Electromagnetic radiation. Sources of non-ionizing radiation - HF, VHF and UHF in water transport are the means of radio station and radio navigation. One of the most powerful sources is ship radio stations.

For example, the intensity of the HF EMF on the electric component is measured in river vessels at the work of SV and AM transmitters with average power respectively: around the feeder tract of SV 50-2000 V.m^{-1}, at AM - 150-2000 V.m^{-1}; at the workplace of the radiist - 20-300 and 50-600 V.m^{-1}.

Ionizing radiation. The crews of nuclear-powered ships may be exposed to ionizing radiation.

Another X-ray radiation source can be used in X-ray equipment. Ship crews may also be exposed to ionizing radiation and to the transport of radioactive material.

Biological factor. Ship crews, when sailing to different continents, can become infected and carry malaria, cholera, yellow fever, etc., and AIDS. At the risk of contamination, crews of ships are also subjected to the transport of animals or of their products - skins, bones, wool, etc., as well as fish processing and fish meal production.

Nature of work. The work of seafarers differs significantly from that of other transport professions - work and rest are carried out in the same place, in an environment separated from the typical social and family contacts on land.

In sea captains, labour is typically mental, with considerable nervous sensory and psycho-emotional intensions with great moral and material responsibility and high qualification.

The work of the helmsman is executive, with a significant sensory load, a standing posture and no significant physical effort.

The mechanics' work is mental, requires a long focus of attention, nervous sensory and psycho-emotional intensions, but also a considerable and frequent physical effort.

In the case of engine mechanics, fireman, and machine boatswain, labour is primarily physical, with a significant sensory load, especially on the hearing analyst.

The work of sailors from the deck command is also mostly physical and often occurs in forced labour postsure.

Work and rest regime. Continuity of the transport process, incl. and in the water transport requires a change of mode of operation (including at night). The working regime in water transport are characterized by: often long working hours, intermittent night work and frequent transition from one shift to another. For example, in the navigational and machine commands of ships based on an 8-hour working day, the shift is divided into two parts (watches) - daytime and nighttime of 4 hours with an 8-hour break between them. The Captain, chief mechanic and the radiist are on an unnormalized working day. The remaining members of the crew are on an 8-hour regular shift. There are no regulated weekends.

Factors aggravating the sailor's work and rest regime are the length of the voyages reaching up to 6 months or more (in trawlers).

17.10.2. RAIL TRANSPORT

Rail transport appeared at the end of the 19th century and was the second type of public transport.

The main professional groups in it are: drivers - drivers and assistant drivers; staff providing remote control of trains - motion-heads and dispatchers (train, wagon, locomotive, energy dispatchers); staff handling the means of transport, passengers and freight while in motion - train master, conducers, stewards, etc.; staff servicing trains, passengers and freight at railway station - auditor wagons, descriptor wagons, shunters, signalman, ticket and baggage cashiers, etc.; staff organizing and controlling driver graphics and health - depotmasters and personal performing pre-railway medical examinations; personnel assuring the technical condition of the locomotives and wagons before leaving the depots; maintenance workers on the contact network and the railway; workers in electrical substations; telephone operators; teletypists; informants; workers in locomotive and wagon-equipment depots, in factories and others.

Microclimate. Many railway workers carry out their labour duties throughout the working shift in the openair-auditor wagons, wagon-descriptors, shunters, signalman, repair workers and new construction of the contact network and the railway track.

Workers in the emergency maintenance of the contact network, the railway track, the telephone connections, the facilities along the railway track

and the railway station, etc. also work outdoors, but only when needed.

Existing heating in locomotives, passenger and service wagons provides for acceptable values of microclimate parameters during the cold periods of the year. During the hot seasons of the year, however, due to lack of reliable thermal insulation and ventilation in the cabs of diesel locomotives, temperatures of up to 40 ° C are measured, and in the electric vehicles - 36-37 ° C. Similar values are also recorded in passenger wagons (in new locomotives and air-conditioned wagons the microclimate is optimal).

Workers at depots and railway repair workshops, especially those in the halls, during the cold periods of the year often perform their labour duties at temperatures below acceptable levels.

Noise. The noise in rail transport is generated by locomotive engines, specialized self-propelled machinery and equipment used in the construction, repair and maintenance of the track. A certain part in the noise characteristic of the rolling composition is so-called. "Traffic noise".

In the cabs of diesel locomotives noise levels in the range of 64 to 119 dB/A are recorded, 66-90 Db/A in shunting locomotives and 42-86 dB/A in electric vehicles. In coaches during a flat-plan motion, the noise level is 42 to 78 dB/A, and when driving in a tunnel the noise level reaches 92 dB/A. Specialized self-propelled machinery supporting the track also generates high noise levels: ballast sieving machines - 57-105 dB/A, transversing - 60-107 dB/A.

Infrasound. For diesel locomotives, the main source of infrasound at a stationary locomotive is its engines, which generate infrasound with an intensity of 79-102 dB. The opening of the side windows increases its level by 4-10 dB. When the electric vehicle is stopped, the level of infrasound is 47-64 dB.

The level of infrasound increases with increasing speed - when moving with 130 km.h and an open one side window is measured 126 dB.

Vibration. The locomotive drivers, the staff handling the trains while driving, and especially the drivers of the specialized machinery for the construction, repair and maintenance of the track during work are subjected to combined general and local vibrations.

The vibration speed of the general vibrations on the floor of the electric cabs is 2,64-5,13 $m.s^{-1}.10^{-2}$ (at 2.0 Hz) to 0.08-0.11 $m.s^{-1}.10^{-2}$ (at 63 Hz). For diesel locomotives due to their higher weight, these values are lower - from 2.41-3.36 $m.s^{-1}.10^{-2}$ to 0.140.18 $m.s^{-1}.10^{-2}$.

On the seat, the vibration velocity of the general vibrations is: for the electric locomotives of 3,09-4.21 $ms^{-1}.10^{-2}$ (at 2.0 Hz) to 0.07-0.11 $ms^{-1}.10^{-2}$ (at 63 Hz), and for the diesel locomotives of 2,39-3.96 $ms^{-1}.10^{-2}$ to 0.16-0.17 $ms^{-1}.10^{-2}$.

As the locomotive's speed increases, the mean values of both measured locations increase.

The vibration velocity of the electric locomotive controller's for local vibration at 60 $km.h^{-1}$ at 8 Hz is 1.50 $m.s^{-1}.10^{-2}$. With increasing frequency, the values decrease and at 1000 Hz reach to 0.009 $m.s^{-1}.10^{-2}$.

For diesel locomotives at the same speed, the values are 0.83 $m.s^{-1}.10^{-2}$ and 0.007 $m.s^{-1}.10^{-2}$ respectively.

Objective the vibration load of the train drivers in% to the admissible level of fatigue for 8-hour shift (with three-component accelerometer according to ISO 2631) shows that the drivers of the motorways exceeded the norm by 48%, those of the highway diesel locomotives by 6% and the drivers of the shunting locomotives load only 1/3 of the allowable rate.

The vibratory characteristic of the specialized machines used in the construction, repair and maintenance of the track is even more complicated and difficult to estimate. The monotonous and steady state of the engine, the chain, the sieve and the conveyor belt, and the greater the length of the ballast sieving machines, determine constant high values of the general vibration. In the case of transversing machines, the general vibrations have a cyclical character, corresponding to the duration of the tie tamping, and are defined vertically and transversally. Longitudinals are considerably weaker, but their characteristic is compounded by low-frequency jump with low frequency and significant amplitude when moving the machine onto the next sleeper. The maximum exceedance of the permissible limits (18 times) is at 31.5 Hz and the minimum (2.3 times) at a frequency of 2.0 Hz.

The general vibrations of the sieving and tie tamping machines considerably exceed the permissible norms - 2-18 times, and the local ones are within the limits. However, when working with manual tie tamping machines, their values exceed the tolerances allowed several times.

Chemical dangers. By replacing steam locomotives with diesel, and subsequently with electric, chemical characteristics in locomotive booths abruptly improved (especially with the replacement of mercury rectifiers of electric locomotives with silicon).

At present, the main chemical hazards in railway mobile composition are registered with diesel locomotives and specialized machinery for the construction and repair of the track. They are separated from their engines.

In the cabs of diesel locomotives, petrol concentrations up to 600.0 mg.m^{-3}, benzene - 0.17-1.50 mg.m^{-3}, toluene - 0.14-1.72 mg.m^{-3}, xylene to 0.108 mg.m^{-3}, CO to 30.0 mg.m^{-3}, SO_2 to 2.0 mg.m $^{-3}$, NO_2, 15-4.20 mg.m^{-3}, CO_2 - 1930-6300 mg.m^{-3} and oily aerosols - up to 11.0 mg.m^{-3}.

High concentrations of ammonia or freons from refrigeration installations are determined in refrigerated trains - 76% of the measurements are above MAC 1 to 25 times.

Dust. In the railway transport of dust impact are exposed the loading and unloading workers, the workers in construction, repair and maintenance of the railway track and the tunnels, the workers in the repair of wagons transporting powdered loads - cement, flour and others. The dust is of a different nature, most commonly mixed - silica with metallic aerosols, iron oxide with lead minium, cereal powder and the like. Its concentration depends on the season and the humidity of the air. For example, a total dust concentration of 10.0 to 1250 mg.m^{-3} was measured during cement car repair. Over 80% is the fine fraction with content a free SiO_2 of 0.8-1.8% and fixed - 21-22%.

Electromagnetic radiation. Railway-specific sources are traction substations that generate EMF at work places up to 5.0 kV.m^{-1}.

Stationary dispatchers radio station screate EMF up to 10.0 V m^{-1} (on working tables) and up to 135 V m^{-1} (for antenna cables).

In the electric locomotive booths of the 27.5 kV mains, EMP values from 0.20 to 0.32 kV.m^{-1} are measured.

Nature of work. The dispatcher's work, heads motion, drivers and more is shift, under control (scheduled), with extended working shifts. The work has neurosensoral and psycho-emotional tension. High qualification and responsibility is required. Higher requirements also exist for the hearing, and for the drivers - and to the vestibular apparatus.

In dispatchers and heads motion work is operative, managerial, processing various and significant information, quick switching of attention, making important decisions, often in time deficiency.

Along with the operator's work in railway transport, despite the mechanization and automation introduced, there are still heavy-duty jobs - loading and unloading workers, working on the manual maintenance and repair of the track and tunnels.

Work and rest regime. The duration of the work shift of locomotive and train staff is different and depends on the category and type of train, while starting and ending hours are at any time of the day. This makes harder to in working and build a dynamic stereotype.

The main mode of work and rest in the railway transport is the extended 12-hour shift - a day (7-19 h) with a subsequent 24-hour rest, and a night (19-7 h), followed by 48 hours of rest. That's work head motion, dispatchers, depot masters, signalman, shunters, and others.

A specific working regime is performed by those working in the repair and maintenance of the railway tracks and tunnels. Here it works in a limited time, the so-called "Technological windows" when the movement of trains in the repair area is stopped. Their duration is usually 4-5 hours.

17.10.3. METRO

This transport is characterized by its higher traffic safety, greater passenger capacity and a higher operating average speed compared to other modes of public transport.

The main professional groups are similar to those in rail transport, but there are some peculiarities regarding working conditions.

The fact that a large number of workers perform their labouor duties in underground premises requires their equipment with powerful and effective ventilation not only in the working premises but also at the platforms of the metro stations and metro train wagons. Another key point is the realization of artificial lighting, which provides maximum comfort for work and travel.

The microclimate is poorly influenced by the macro climate and usually in the work rooms, and metro trains, is acceptable. low subterranean temperatures (below 10 ° C) and high air currents (7-8 to 10 m.s^{-1}) are measured in the cold periods of the platforms of the metro stations and in tunnels.

The driver's cab ***noise*** is 84 dB/A; the wagons are 88-94 dB/A; and 73 dB/A (upon arrival of the metro) and 86 dB/A (on departure) on the platform. The movement of the metro train raises the background noise in all working areas in the metro stations, and the continuous movement in the tunnel generates high levels of noise in the passenger carriages. This has high demands on their sound insulation.

The general ***vibrations*** in the metro train are low-frequency with a peak at 2.0 Hz - 1.7 m.s^{-1} .10^{-2} and sharply decrease with increasing frequency. The rating for integral vibration velocity of the local vibrations of the metro trains control levers is 0.4 m.s^{-1} .10^{-2}.

Toxic substances (CO_2, CO, NO_2, hydrocarbons, etc.) and ***dust*** can pollute airspace in metro stations and wagons through passenger flow, ventilation, after night tunnel cleaning, metro trains, etc. The electric brakes emit a dust with a concentration of

no more than 2.0 mg.m^{-3}, and the mechanical ones up to 70.0 mg.m^{-3}. The electro-thrust also produces insignificant amounts of ozone, which quickly passes into oxygen.

Biological factor. In the air in the working environment in the metro stations and wagons can be isolated conditionally pathogenic and pathogenic microorganisms, helminth eggs, etc., and the crowding of many people in one place creates prerequisites for rapid and easy spreading of air droplets and other infections.

The nature of the work of the professional groups serving the metro is broadly similar to those of rail transport, with an aggravating factor - work in the case of artificial lighting. Apart from the latter, the working conditions of drivers are less favorable than those of locomotive drivers due to increased accelerations and very frequent starts and stops, while the full shift-motion in tunnels in the absence of a panoramic view results in a stroboscopic effect on train drivers in nearness to the walls of the tunnel. All this determines the work of the metro train drivers as more strenuous, which to a certain extent is compensated by their different ***working regimen*** and intervals of rest - eg. now in Sofia with a 12-hour shift, the net driving time is 7-8 hours, and every 40 minutes the drivers rest in the metro stations for 20 minutes.

17.10.4. ROAD TRANSPORT

Road transport began its development in the early twentieth century. With the improvement of internal combustion engines (gasoline and diesel) and the ability to make door to door deliveries, it quickly became the most used transport.

The main professional groups in the road transport are: the drivers, which are divided into truck drivers - light, medium, heavy, international, working in open mines and quarries, etc., drivers of passenger cars - light (up to 8 people) and buses - urban, interurban; personnel organizing and controlling the movement of the vehicles - dispatchers; personnel conducting pre-hospital medical examinations for drivers; personnel servicing the passengers during the movement - conductors, stewards, etc.; staff servicing passengers and freight terminals - ticket and baggage cashiers; personnel ensuring the technical condition of the vehicles before leaving the garages; workers on current maintenance and repair of cars in garages; construction and maintenance roads staff.

Microclimate. Throughout the open-air work, staff are involved in the construction and maintenance of roads, extraction, processing and preparation of road building materials.

Existing heating in passenger and commercial vehicles ensures that the parameters of the microclimate are set during the cold periods of the year. Insufficient maintenance of heating systems and seals in old cars is the reason for the low values (up to 8-10°C) in driver's cabs and passenger lounges.

Problems arise during hot periods of the year when, due to insufficient thermal insulation, lack or insufficient mechanical ventilation in driver's cabs and passenger lobbies, temperatures in the range of 30-35°C are measured, and in heavy-duty vehicles, especially those working in quarries and open mines, above 35°C. Conditions are created for overheating, which is removed, to a certain extent, by opening the windows, with all the disadvantages of the emerging high air currents. In new cars with air conditioning, this problem has been eliminated.

The microclimate in the repair halls and auxiliary compartments during hot periods of the year is usually within the permissible limits - 20-25°C, relative humidity - 55-75% and air speed - 0.2-0.5 m.s^{-1}.

During the cold and transition periods in large halls and due to the lack of efficient heating in them, temperatures are considerably below the allowable.

Noise. The noise in road transport, which creates environmental problems, is mainly generated by motor vehicles, and in road construction and repair - from used machinery and equipment. Parts in vehicle noise are also characterized by "traffic noise", road condition, engine layout and technical condition, driver's cab and passenger compartment seaing, etc.

Noise levels in the range of 87 to 90 dB/A, 77-87 dB/A and 76-86 dB/A in heavy-, medium- and light-duty vehicles are recorded in vehicle's cabs. On buses, the noise level is 76 to 83 dB/A, in passenger cars - up to 78 dB/A and 77-86 dB/A in international transport heavy duty vehicles.

Different types of repair work generate noise levels of 77 to 99 dB/A, and 80-99 dB/A in the road construction (excavators, scrapers, bulldozers, etc.).

Infrasound. For the various trucks, at a speed of 60-70 km/h and closed windows, the driver's infrasound level has an average of 102 dB. Opening the side window increases its level to 109 dB.

In the driving cabs of passenger buses at the same speed and closed windows, the infrasound level is 98-112 dB (average 105 dB).

Vibration. Here, as in rail transport, drivers and staff handling buses while on the move are subjected to combined vertical, longitudinal and transverse general and local vibrations.

Measured values of floor and seat general vibrations are more favorable for buses compared to freight cars. This is explained by their structural fea-

tures (the trucks and dump trucks have a more rigid suspension) and the conditions of operation (type, relief and road condition). The unfavorable vibrational characteristic is largely corrected by the anticipated shock absorber systems of the driver and passenger seats.

Increasing the speed of movement increases the average vibration velocity of the general vibrations of both measured locations.

The objectivization of the all-shift vibrational load indicates (measured) that the drivers of trucks in extra-urban traffic exceed the norm more than 3 times, and in urban traffic - by 55%. Drivers of buses and international road transport exceed the norm about 2 times.

An integral vibration velocity assessment of the local on steering wheel vibrations shows values for different vehicles from 0.4-0.7 $ms^{-1}.10^{-2}$ (for lightweight) to 2.7-3.0-4.2 $ms^{-1}.10^{-2}$ (for heavy). When working on open mines and quarries these values were even higher - 4.5-5.5 $m.s^{-1}.10^{-2}$.

Chemical dangers. Road transport is one of the main pollutants of both the environment and its own work and living premises. The main source of toxic substances are the engines, their location, sealing, insulation and technical condition, used fuel-lubricants, the type of vehicle, the condition and the relief of the road, etc.

With modern cars, the possibilities of getting exhaust fumes in cabs and passenger lounges are minimized. The presence of chemical pollutants (CO_2, CO, hydrocarbons, SO_2, NO, etc.) is detected in non-sealed engines located in the cabin, and their concentrations are usually below the MAC. In case of aggravated engine operating conditions (climbing, forcing, driving on bad roads, open mines and quarries, etc.), concentrations up to 600-900 $mg.m^{-3}$ are measured.

CO_2 concentrations in the range of 2160-5900 $mg.m^{-3}$, CO-5,0-60,0 $mg.m^{-3}$ and sometimes traces of hydrocarbons are measured in the buses. The less favorable values are measured for buses whose engines are located in the cabs.

In trucks, particularly heavy duty, in mining and quarries, the hydrocarbon concentration in their cabins reaches 100-150 $mg.m^{-3}$, CO-15-60 $mg.m^{-3}$.

Dust. Pollution is not a characteristic indicator for driving, but with certain loads (bulk, earth), cabin dust concentrations of 2.0 to 330.0 $mg.m^{-3}$ are found. Maximum values are measured for dumpers working on mining and quarrying, when 70% of the samples exceed the MAC.

In road transport are exposed to dust handling workers, construction, repair and maintenance of roads and tunnels workers, repairers of dust-carrying vehicles - cement, flour, etc., open-pit and quarries workers, including and their drivers and others. The powder is extremely inorganic, most commonly mixed - silica with metallic aerosols, iron oxide with lead minium, cereal powder and others. Its concentration depends on the season, air humidity and condition of the road surface.

In the construction, repair and maintenance of roads the measured dust concentration is 3.12-234.84 $mg.m^{-3}$. The medium dispersion fraction was predominant, and the free SiO_2 content was up to 16% in 50% of the samples.

Biological factor. High microbial contamination can be recorded in the bus lobbies. Biological pollutants can also be registered for the transport of live animals or their products - fur, wool, etc., as well as in the working environment of conductors, cashiers, stewards and others.

Nature of work. The work of the drivers is nervous-sensory with great psycho-emotional intension. They make important decisions, most often in deficit of time, accept and process a large amount of information. The visual, auditory and vestibular analyzers are very load.

Drivers of urban buses (with an straight engagement of 82.0% on average) are the most burdened, followed by those traveling in interurban conditions (72.0%). For truck drivers, the hardest and most intension is labour is at opening of mines.

Main disadvantages in the work of service workers are the high percentage of forced labour posts, considerable static loading and physical effort in the manual carrying of heavy details.

Work and rest regime. The duration of the shift is different and depends on the type of vehicle (freight or passenger), driving conditions (urban, interurban, international, mixed).

Drivers' work and rest regime is norational with mounthly time accounting, extended working shifts, often with unregulated breaks and a significant percentage of night work.

International drivers must comply with the ILO Convention No. 153 of 1979, which regulates their working and rest regime and limits the driving time to 9 hours daily (48 hours weekly) and after 4 hours compulsory rest, and at least 10 hours rest in 24 hours.

Service workers usually work on a regular 8-hour shift, in some services there are first and second day shifts, and night-shifts in those serving public transport.

17.10.5. AIR TRANSPORT

Air transport is the newest mode of transport.

The main professional groups in Civil Aviation (CA) are: flight and passengers servicing personnel - commander, pilot, radio operator, navigator, boardman and stewardess; personnel providing airplane remote controls - flight and air traffic controllers; grounding staff - start-ups (starter, airmechanic, aircraft technicians, drivers, fuel lubricant workers) - serve landing and take-off aircraft and in the regulation - aircraft mechanics and technicians performing current and major repairs of the aircraft in the repair depots; staff servicing passengers and cargoes at airports - ticket and baggage cashiers, informants, etc.; medical staff performing pre-flight medical examinations, etc.

In agricultural aviation (AA), the main occupational groups are: on the worksite - pilots, aeromechanics and unskilled workers, loading the aircraft with pesticides or fertilizers; in the repair facilities - aircraft technicians and mechanics.

Microclimate. At open-air through all shift worked, workers on start at the CA and on AA loading depots, without pilots.

The air conditioning systems and the good airtightness of CA airplanes all year round create optimal microclimate values and barometric pressure up to 6300 m high. At higher flight altitudes (12,000 m) the atmospheric pressure in the airplanes is 0,57 atm, which is equal to the atmospheric pressure at a ground point at an altitude of 2300 m. An unfavorable moment is the dry air in the airplanes -humidity around 25%.

The microclimate in the CA and the AA hangars and the auxiliary compartments to them during the hot seasons of the year is usually within the permissible norms. During cold and transitory periods, temperatures are to a large degree below the allowable temperature.

In non-sealed pilot cockpits of the AA, the microclimate is determined by the macroclimate.

A characteristic feature of the microclimate in air transport is the ability of flight crews to change the climate zones for a short time (2-3 hours), which creates conditions for over-intension of the body's thermoregulatory systems.

Noise. Air traffic noise is a leading unfavorable factor affecting not only air transport workers but also the environment.

In the cockpits and passenger saloons of the turbo-propeller aircrafts the noise level ranges from 81 to 93 dB/A, in the reactive -67-87 dB/A, 87-103 dB/A in the helicopters, and 97-104 dB/A in AA aircrafts. At airplane startup, the noise levels are divided into 3 zones: near to aircraft -112-122 dB/A, a background of the runway - 81-101 dB/A, and in the enclosed work premises - 68-88 dB/A.

Infrasound. For turbo-jet airplanes, the overall level of infrasound does not differ from acoustic noise and ranges from 83 dB to 91 dB.

In jet planes, the acoustic noise is below the propeller at basically preserved infrasound intensity of 68-100 dB.

For helicopters - a total infrasound level of up to 114 dB, and in the cockpits of AAs - up to 106 dB.

Vibration. In air transport, general vibration is not as important a factor as in other modes of transport. Flight staffs are subjected to high frequency general vibrations (from aircraft engines) in combination with aperiodic, low-frequency thrusts (airplane movement and turbulence in the atmosphere). Maximum values are recorded for taxiing on the runway at take-off and landing. The less-favored position is the AA and helicopter pilots.

Local vibrations, with overnorms values, are typical of aircraft repairs using hand-held pneumatic guns, drills, rifles, grinders, and others.

Chemical Dangers. They have a leading role in the AA, and at certain times during the repair and star-tup.

The average CO and CO_2 concentrations of 25.0 $mg.m^{-3}$ and 2600 $mg.m^{-3}$ respectively are measured in the cockpits of cargo aircraft. In passenger planes these values are higher - CO - 75.0 $mg.m^{-3}$ and CO_2 - 3400 $mg.m^{-3}$.

On start-up, toxic substances are separated from the aircraft engines and the facilities used for their maintenance. Excessive hydrocarbons of gasoline and petrol engines - 129.0 $mg.m^{-3}$ average and of kerosene - average 83.0 $mg.m^{-3}$ are registered.

The hydrocarbons content of the aeromechanical working environment has an average concentration of 150.0 $mg.m^{-3}$.

At the start-up, the CO concentration is from 20.0 to 420.0 $mg.m^{-3}$, nitrogen oxides - 4.0-11.0 $mg.m^{-3}$ and ozone and photooxidizers - 0.2-1.3 $mg.m^{-3}$. Oily aerosols are in concentrations of 2.50 to 44.0 $mg.m^{-3}$.

In the various repair operations, benzene concentrations (average 31.0 $mg.m^{-3}$), toluene (13.0 $mg.m^{-3}$), xylene (9.0 $mg.m^{-3}$), acetone (118.0 $mg.m^{-3}$), phenol (9.00 $mg.m^{-3}$), and other.

The AAs have a less favorable sanitary and chemical characteristics compared to those of CA as determined by the additional toxic substances - PhOC, nitrogen fertilizers, etc. When repairing the AAs, PhOC over-norms concentrations were also measured, although aircraft were washed before repairs.

Electromagnetic radiation. A significant number of powerful sources are found in air transport, both in aircraft (radar and radionavigation equipment), as well as on ground-radars, radiostations, etc., and in their repair in specialized laboratories. For example,

up to the antenna of the portable radio stations is measured the VHF EMF with an intensity of about 25 V m^{-1}, under the forepart of the aircraft at 2 m from the radiating antenna - 25-40 V.m^{-1}.

Ionizing radiation. In some airplanes and helicopters there are radioactive anti-ice indicator devices in which there are Sr^{90} and J^{90}. They emit β and γ rays at the exposure dose rate of 800 mR.h^{-1} at a distance of 1 cm from the device, at 10 cm-150 mR.h^{-1} and at 1 m-0.3 mR.h^{-1}.

In some devices, especially in older aircraft, radioactive luminous dyes on scales containing Ra^{226} were used. They emit gamma rays with an exposure dose of 2-17 mR.h^{-1}.

A problem for the crews of the airplanes is also the cosmic radiation. (FCA) has estimated that for 960 flight hours (annual norme for flight hours) the crews receive a radiation dose of 0.24 to 8.9 mSv, which is below the recommended limit by the International Commission on Radiation Protection (ICRP) of 20 mSv per year.

Nature of work. The specificity of flight crew work is determined by the nervous and psycho-emotional intensions of simultaneous observation of many time-varying parameters, acceptance and analysis of a large amount of visual and auditory information, decision making, often in a complex environment and a deficit of time, great personal responsibility and high risk.

Work of flight controler and dispatcher's operations are extremely complex and diverse. There are over 25, time-varying, objects with high concentration and switching over attention, stress situations, responsibility, time deficiency, heavy loading hearing and visual analyzers, imposed tempo, requiring urgent and precision.

The work of technical staff from start and repair is categorized as low to moderate physical, moderately intense.

The work of AA pilots and mechanics, working on airplaces is additionally aggravated by adverse factors such as: open air, unevenness, nonrhythmicaly, frequent take-offs and landings, short flight times, active flying at low altitudes, and at the same time operate with spraying or fertilizing devices - worse working conditions combined with excessive concentrations of PhOC and others.

Work and rest regime. Flight activity over its duration is governed by a daily, monthly and annual rate.

A shift work with extended 48-hour rest periods applies to the flight manager.

For air mechanics and air technicians operating at the start, two 12-hour shifts are - daily (7 to 19 hours), followed by 24-hour rest, and night (19-7 hours), followed by 48 hours of rest.

At the AA airplaces there are aggravated working regimes with labour irregularities, extended and fragmented working days - from 5 to 9 hours and from 16 to 19.30 hours with insufficient intershifts rests. The number of working days is directly related to the flight hours, which are normalized depending on the kind and type of the aircraft, the number of pilots in them, the type of aerochemical work, etc.

MORBIDITY AND TRAUMATIZATION

The total morbidity (the totality of the registered and the newly discovered cases) is the highest in rail transport, followed by road, water and air. The highest indexes are those who are over 50 years of age. Prevalence of respiratory diseases (chronic bronchitis, ACUAP, etc.), blood circulation (arterial hypertension), digestive system (ulcers, gastritis, etc.), nervous system and sensory organs, musculoskeletal system (intervertebral disc disease), that can be identified as problematic for transport workers.

Similar is the morbidity of temporary incapacity, where the most common are ACUAP (about 1/5 of all illnesses), followed by household accidents (10-11%), osteochondrosis and hypertonic disease, etc.

Half of the registered cases of occupational diseases are in the railway transport. Followed by those in the automobile (36%), the air (7%) and the water (6%). The most frequent are neurites of the auditory nerve (28%), followed by vibrational disease (21%), vegeto-vascular syndrome (12%), vegetative polyneuropathy of the upper extremities (9%), myotendinoses and myotendinitis (7%).

Labour accidents are 2.2-3.0% of all cases of incapacity with temporary disability. The most serious and most frequent are the labour traumatism in water transport, followed by the road, railway industry and transport constructions, and the lightest in air transport.

HYGIENE MEASURES

Hygienic measures applied in other sectors to unfavorable working conditions can also be applied in the transport system. Transport-specific preventive measures can be summarized as:

1. Development of specific hygiene standards related to the occurrence of fatigue in personnel (especially for workers in operation and related with traffic safety) and providing comfortable conditions for travelers.

2. Models and systems for optimizing work and rest modes.

3. Effective medical and professional selection and expertise of the labour ability and suitability of the various occupational groups of transport workers.

4. Organization of systems for pre-road and periodic examinations, ensuring early detection of deviations in the functional state of the body related to

traffic safety and occupational diseases.

5. Providing reliable bioprophylaxis for transport workers through rational food-drinking regime, hardening of the organism, etc.

6. Reduction of hypodynamics in the main transport professions - drivers, dispatchers, etc., by creating appropriate recreational complexes.

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17.11 CHEMICAL-PHARMACEUTICAL PRODUCTION. PHARMACIES

Chemical and pharmaceutical production is part of the chemical industry and in many of the developed countries in the world is one of the most important and promising industries. It covers a large number of state and private structures (companies and healthcare establishments) that carry out research and applied activities for setting up, pre-clinical and clinical tests, production and sale of pharmaceuticals for the needs of human and veterinary medicine.

In Bulgaria there is a legal framework and an established legal order for legalizing the production, import, export, use and trade of pharmaceuticals, incl. the protection of copyright in the patenting of new medicines and trademarks, the reproduction of medicinal products and the right of company secret in production.

The overall activity for providing the population with the necessary medicines is managed by the Pharmacy Council at the Ministry of Health.

The production and sale of pharmaceuticals are organized in compliance with the requirements of the existing normative acts, which regulate:

- hygiene protection zones (HPZ).

They are: in the manufacture of synthetic medicaments, antibiotics and preparations for agriculture - 1000 m; production of glycerin, organopreparations and phytochemicals - 100 m; production of pharmaceutical potassium salts, tabletting and arrangment of finished dosage medical forms; distillation of ethereal oil products, cosmetic creams, shampoos and toothpastes in enclosed installations - 50 m;

- an environmental impact assessment, which is an overriding step in the construction of new production capacities;

- methods for collecting, transporting, storing and disposing of hazardous waste, to which part of the solid waste of pharmaceutical production belongs;

- the ambient air quality requirements with which the exhaust air purification devices must comply in production;

- the order and the way: - to control the quality of the working environment; - for work with video display; - the periodicity of carrying out a risk assessment in actual production conditions, which is a mandatory part of the overall assessment at the workplace; - the conditions and requirements and the development and introduction of physiological regimes for work and rest during work, taking into account the existence of work operations with significant physical effort (mainly loading and unloading of raw materials and finished goods);

- protection of workers against the risks associated with exposure to noise;

- control of compliance with the permissible concentrations of harmful substances in the air of the working environment;

- occupational safety rules in the chemical-pharmaceutical, microbiological and perfumery-cosmetic industries;

- the licensing rules for manufacturers and wholesalers of medicines;

- the documentation requirements for the registration of medicinal products;

- the structure, procedures and organization of pharmacies' operations and the nomenclature of medicinal products;

- water quality requirements for drinking and household purposes (since water is a raw material for the production of liquid pharmaceutical forms and its properties, especially in the manufacture of ampoules and eye drops, very stringent requirements are being put in place).

Compliance with the Company's secret law does not limit the control over the quality of working conditions and mainly of the air in the working environment provided by the Health and Safety at Work Act. These two aspects are directly related to the pharmacological and toxicological characteristics of the manufactured medicinal products. Pharmacological characteristics depend on their effectiveness as a prophylactic or therapeutic agent. The toxicological characteristics depend on the undesirable or side effects (organ toxicity) that the drugs may have. On the other hand, the presence in the working environment of the active ingredients of the drugs as well as of other chemicals in the form of raw materials, intermediate, final or waste products (chemical factor), combined with the accompanying production physical factors, forms specific working conditions. Working conditions have a definite, significant impact on both the quality of production and the health of workers.

The assessment of the compliance of the working conditions with the requirements of the Labour Code and the national standards and the risk for the health of the workers is part of the documentation for certification under ISO 9001 for quality of production. It is the duty of the employer to ensure healthy and safe working conditions.

17.11.1. PRODUCTION BUILDINGS

According to the requirements of the Health and Safety at Work Act and Ordinance No 7 on Minimum Requirements for Health and Safety at Work Workplaces and the Use of Work Equipment, **the sites for the construction of enterprises**, buildings, shall be determined according to the activity to be carried out in them, ensuring the norms and the requirements for protection of workers and the population. The above-mentioned normative documents concern precisely these two aspects of health prevention. When choosing a site must be the minimum protection zones to the sites, taking into account the direction of prevailing winds, the peculiarities of the terrain and climate. **Minimum protection zones** for objects in the pharmaceutical industry range from 1000 m to 50 m.

Buildings in which workplaces are located must have design and operational safety as intended.

The free areas of the plants are landscaped with appropriate grass, tree and shrub vegetation. Protected areas are not allowed to build industrial and residential buildings. The commissioning of enterprises, sites and work equipment shall be carried out with permission for use of the building, issued by the competent authority in accordance with the Territorial and Urban Planning Act. The owner or the user of the enterprise, the site or the working equipment compiles a file containing documents on the design of health and safety at work and the documentation (protocols and acts) proving the conformity of the completed construction with the requirements of the Health and Safety Work Conditions and all documents reflecting periodic and in-service checks.

The area of the enterprise (site) is enclosed and measures are taken to limit the access of outsiders. With compositional decisions within the enterprise ensure that all requirements related to safety and health, zoning, marking hazardous areas, servicing roads and railways etc. The roads in the territory are constructed and maintained with durable pavement and are marked with the necessary markings, road signs and signaling.

Work premises and workplaces must ensure safe working conditions and health protection for workers. It is not allowed to exceed the established standards for production microclimate, noise, vibrations, dust, toxic substances, lighting and non-ionizing radiation in work premises and workplaces. Technological processes and activities with dust extraction, toxic and harmful substances, noise and vibrations above established norms, with overheating microclimate, wet processes, etc. are organized in separate buildings or premises. Work rooms and work equipment, incl. ventilation systems are cleaned regularly.

The workplace is shaped according to the ergonomic requirements and the anthropometric characteristics of the workers. Sufficient area, volume and free unoccupied work area are provided in the workplace so as to ensure sufficient freedom of movement for workers to perform the work and fire safety for the activity concerned. The organization of work, dimensions and layout of the workplace take into account the physiological and ergonomic requirements for ensuring the normal passing of the work process.

The floors of work rooms must be rigid and stable, ensure that workers are isolated from health hazards, safe gradients, obstructions and openings. The floors and walls of work rooms are made of materials that do not emit, leak and carry noxious emissions and comply with fire safety requirements. They are made of materials and in a way that does not make it difficult for them to be cleaned regularly. Due to the specifics of chemical-pharmaceutical production and the risk of accidents (gassing, spilling of aggressive liquids, self-ignition or self-blasting), special requirements are made for the type, number, dimensions, places and materials from which the doors and gates are made.

The mechanized doors must have an easily visible and accessible opening mechanism. Doors on failure routes must be marked with the appropriate characters, be opened from the inside out without any obstacles at any time and without a key. In case of an emergency in the power system, the mechanized doors must be automatically opened and remain open. Doors and gates designated for passing only vehicles shall be marked with signs prohibiting pedestrian traffic.

Windows and skylights should provide enough natural light, to be cleaned regularly.

Electrical equioment and installations do not create conditions for an increased risk of electrotraumas. Road routs and hazardous areas are marked accordingly. The places for loading and unloading of raw materials and finished products are marked and equipped with the necessary mechanization to reduce physical effort at work. Approved and marked emergency routes and exits free to move workers at any time.

For workers in the open, measures are to be taken to guard against falling objects, noise, gases, vapors, dust, non-ionizing radiation, falling and slipping, and to prevent the effects of adverse weather conditions.

17.11.2. WORKING ENVIRONMENT AND PRODUCTION PROCESS

Hygiene requirements to the work environment include measures to provide the necessary workplace **lighting** and an optimal **manufacturing microclimate**. It is necessary to provide for preventive measures to prevent the harmful effects on health both in low-temperature and overheating rooms; in the presence of a surface temperature of the process equipment above 55°C; in manual handling with control devices above 45°C. For activities hazardous to overheating workers, conditions should be provided for rational cooling and a drinking regime to compensate for losses from liquids and mineral salts.

No **concentrations of harmful substances** in the air of the working environment are allowed above the specified hygiene standards. The MACs and the Tentative Safety Influence Levels (TSILs) of the most commonly used organic solvents, other chemicals, medicinal plants, certain antibiotics and finished pharmaceuticals are presented in **Tables 1-4**.

The production processes in chemical-pharmaceutical production must be maximized mechanized and automated. Since the handling of hazardous chemicals for high toxicity and pharmacological activity, the equipment must be maximally sealed. When handling powdery materials and materials, apply dust collection measures. Powder materials are transferred indoors. In sifting for separation, thick casing with the possibility of connection to a aspiration device is mounted on screens. To reduce secondary dust pollution use vacuum cleaners for workplace and work equipment, wet floor and surface cleaning. In case of short-term contact with harmful substances, which can not be prevented, when handling powdered substances (weighing, grinding, dry mixing, boiled layer drying, etc.) in the cleaning and repair of workplaces and equipment, which is often requires in the manufacture of medicines, that personal protective equipment (PPE) be used.

In dusty, toxic and other harmful substances, forced **ventilation** is provided. The local aspiration occurs at the source of the release of harmful substances. Where an emergency release of highly toxic substances or the creation of explosive and fire-hazardous concentrations in the work rooms or locations is possible, an automatically activated emergency ventilation system shall be provided. Local suction systems, serving high-toxic gas and dust extraction facilities and general-exchange ventilation installations in work rooms shall be installed in such a way that they are automatically switched on together with the technological equipment and block its inclusion in the case of a ventilation malfunction.

Quality control of the work environment is done by accredited control agencies. Periodicity for the control is determined by the specific conditions, of the Occupational Health Service's instructions, which assesses the health risk and organizes the preventive medical examinations. Ordinance No. 5 (1999) provides except for an initial risk assessment and another, to take place in the event of changes in technology, replacement of equipment or the introduction of new production. The hazardous substances used, incl. with carcinogenic potential, are necessarily filed. The chart contains data about their characteristics, described in the so-called "Material Safety Data Sheet", with the content of which is known to people in contact with chemicals.

If there are excessive **noise levels in the workplaces** depending on the technological possibilities, technical solutions of its limitation are applied by means of screens, casings, covers, fences, sound absorbing treatment of walls and ceilings, isolation, remote control of machines and equipments, soundproofed booths for staff and more. When operating under noise conditions above established norms and when all the noise reduction requirements are applied, workers use antiphons (external or internal -ear muffs).

Machinery and equipment - sources of **vibration**, are installed on projects that guarantee the mechanical reliability of the building structure of the buildings and the facilities and their individual elements. To reduce the vibration level, machines and equipment are mounted on separate foundations and/or technical solutions are used to reduce the vibration level.

The list of approved medicines for production and use in Bulgaria (presented in groups in Appendix 1) includes a wide range of preparations for the prevention and treatment of a large number of diseases and health injuries. This list contains the pharmacological groups which must be included in the nomenclature of medicinal products of pharmacies, according to Annex 1 to Art. 4 of Ordinance No. 8 on the structure, order and organization of medicinal products. The technologies for their production are varied and characterized by a number of specific features. Bulgarian pharmaceutical companies produce some of the famous medicines.

Annex 1 (Ordinance No 8/2000)
List of drugs in pharmacological groups

1. Drugs affecting the nervous system
1.1. Anesthetics
1.2. Analgesics and Non-Steroidal Anti-Inflammatory Drugs
1.3. Classical sedatives
1.4. Somnific preparations
1.5. Antiepileptic preparations
1.6. Antiparkinsonian preparations
1.7. Psychotropic means
1.8. Drugs used in alcoholism and tabacism
1.9. Vegatotropic agents
1.10. Miorelaxants
1.11. Revulsant and local anti-inflammatory drugs
1.12. Antiepileptics
2. Autocoids
2.1. H_1 - blockers
2.2. Serotoninergic receptor agonists and antagonists, effective in migraine
3. Drugs affecting the cardiovascular system
3.1. Anti-anginal (anti-stenocardie) agents
3.2. Medicines to treat heart failure
3.3. Antiarrhythmic agents
3.4. Antihypertensive agents
3.5. Anti-hypotensive agents
3.6. Anti-atheromatous (hypolipidemic) agents
3.7. Peripheral vasodilators
3.8. Venotonic and capillary-tonic (vasculoprotective) agents
4. Respiratory tract medication
4.1. Anti-asthmatic agents
4.2. Anti-cache
4.3. Expectorants and mucolytics
4.4. Pulmonary surfactants
5. Drugs affecting the digestive system
5.1. Antiulcer agents
5.2. Appetite affectants
5.3. Antiemetics
5.4. Means that affect the tone and motor of the gastrointestinal tract
5.5. Antiflatulente agents
5.6. Laxatives
5.7. Antidiararic agents
5.8. Intestinal anti-inflammatory drugs
5.9. Means that affect the liver and bile secretion
5.10. Pancreatic enzyme preparations
6. Medicinal products affecting the excretory system
6.1. Diuretics
6.2. Means affecting bladder dysfunction
6.3. Amino acid preparations for oral use in chronic renal failure
6.4. Uricolytic agents
6.5. Dialysis solutions
7. Drugs affecting the reproductive system
7.1. Gonadioactive agents
7.2. Ureteral agents
7.3. Aphrodisiacs and anti-aphrodisiacs
8. Haematological medicinal products
8.1. Antianaemic agents
8.2. Anticoagulants
8.3. Thrombolytics
8.4. Platelet anti- aggregates
8.5. Hemostatics
9. Infusion and dialysis solutions
10. Endocrine and metabolomic drugs
10.1. Antidiabetic agents
10.2. Anti-hypoglycaemic agents
10.3. Hypothalamic and pituitary hormones
10.4. Thioreactive agents
10.5. Antiparathyreoid agents
10.6. Corticosteroids
10.7. Anabolic steroids
10.8. Anti-gouty agents
10.9. Means affecting mineral homeostasis in bone tissue
11. Vitamins and trace elements
12. Antimicrobial drugs
12.1. Antibiotics
12.2. Anti-tuberculosis agents
12.3. Sulfonamides
12.4. Quinolones
12.5. Antifurans
12.6. Oxyquinolines
12.7. Anti-lues agents
12.8. Antiviral agents for systemic use
12.9. Antimycotics for systemic use
13. Anti-parasitic drugs
13.1. Antiprotozoal agents
13.2. Anti-helminthic agents
14. Antiseptics and disinfectants
15. Immunoglobulins, serums, vaccines and fortifiers
16. Biological response modifiers
16.1. Immunosuppressants
16.2. Immunostimulants
16.3. Immunoglobulins
17. Antitumour drugs
18. Dermatological drugs mainly for topical use
19. Ophthalmic drugs mainly for topical use
20. Otorhinolaryngologic drugs mainly for topical use
21. Dental medicaments for topical use
22. Biobandages, biomaterials for transplant, tissue adhesives
23. Diagnostic agents
24. Antidotes
25. Homeopathic medicines.

Synthetic drugs production include a large number of chemicals, which are raw materials or end products, and many other processes in the chemical industry. Chemical agents in pharmaceutical manufacture can be divided into 2 large groups: industrial reagents and drug-related substances. Consequently, a number of chemicals - industrial poisons - are found in the air in the working environment in the form of vapors, aerosols and dust. This is most often used as raw materials: reagents, initiators, catalysts, fillers and organic solvents. In addition, the same chemicals are also found in laboratories that control production quality and technological control. Workers are in contact with chemicals and when they are by-or waste products and are subject to removal and disposal.

Tab. 1. Organic solvents used in the manufacture of medicaments

Resolvent	Participation	Physical state	MSMAC[1] in mg/m^3	STMMAC[2] in mg/m^3	Specific effects
Amyl acetate	OS;* F**; E***	vapor	270.0	540.0	-
Amyl alcohol	OS; F; E	vapor	100.0	–	-
Ammonia (liquid /	OS; F; E	gas	14.0	36.0	-
Aniline	OS	vapor	2.0	–	Allergen. Skin-resorptive effect.
Acetone	OS; F; E	vapor	600.0	1400.0	-
Benzene	OS	vapor	3.25	–	Carcinogenicity to humans (1 cat.). Skin-resorptive effect.
n-Butyl acetate	OS; F	vapor	710.0	950.0	Irritation
n- Butyl alcohol	OS; F; E	vapor	100.0	150.0	-
DEA	OS; E	vapor	30.0	75.0	Allergen. Irritation. Skin-resorptive effect.
Dimethylamine	OS	vapor	3.8	9.4	-
Dimethylaniline	OS	vapor	2.0	–	Allergen.
Dimethylformamide	OS; F; E	vapor	30.0	–	Toxic for reproduction (2 cat)
1,4-Dioxane	OS; E	vapor	50.0	90.0	Skin-resorptive effect
1,2-dichlorobenzyl	OS	vapor	120.0	300.0	Skin-resorptive effect
1,4-dichlorobenzyl	OS	vapor	122.0	306.0	Skin-resorptive effect
1,2-Dichloroethane	OS; E	vapor	4.0	8.0	Carcinogen (Cat 2)
Ethyl acetate	OS; F; E	vapor	800.0	–	-
Ethylene glycol	OS; E	aerosol	52.0	104.0	Skin-resorptive effect
Ethyl alcohol	OS; F; E	vapor	1000.0	–	-
Isopropyl alcohol	OS; F; E	vapor	980.0	1225.0	-
o; m - „; p-Xylene	OS	vapor	221.0	442.0	Skin-resorptive effect
Methylamine	OS	gas	12.0	–	Allergen. Irritation
Methylen chloride	OS; F; E	vapor	100.0	517.0	-
Methylisobutylketone	OS; F; E	vapor	50.0	200.0	-
Methyl alcohol	OS; F; E	vapor	50.0	–	Skin-resorptive effect. Irritation.
Pyridine	OS	vapor	15.0	–	Irritation
Propyl alcohol	OS	vapor	300.0	500.0	Skin-resorptive effect. Irritation.
Tetrahydrofuran	OS	vapor	150.0	300.0	Skin-resorptive effect
Toluene	OS; F; E	vapor	150.0	300.0	-
Triethylamine	OS; F	vapor	8.4	12.6	Skin-resorptive effect
Formaldehyde	OS; F; E	vapor	1.0	2.0	Allergen. Irritation.
Formamide	OS	vapor	15.0	30.0	Skin-resorptive effect. Toxic for reproduction.
Furfural	OS	vapor	10.0		Irritation. Skin-resorptive effect.
n-Hexane	OS; F; E	vapor	180.0	400.0	Skin-resorptive effect
n-Heptane	OS; F; E	vapor, aerosol	1600.0	–	-
Chlorobenzene	OS	vapor	47.0	94.0	-
cHloroForm	OS; F; E	vapor	10.0	–	Skin-resorptive effect.
Cyclohexane	OS	vapor	500.0	–	-

[1] - Mean-shift MAC-MSMAC (Limit for 8 hours)
[2] - Short time maximum MAC-STMMAC (Limit to 15 min.)
* OS - Organic Synthesis
** F - Fermentation (antibiotics production)
*** E - Extraction

Table 1 presents the MAC and specific characteristics of organic solvents and other substances used in the chemical-pharmaceutical industry.

Organic solvents used for **extraction** are practically recovered as they are involved in closed production cycles. Few of them fall into waste water production depending on their water solubility and the nature of the equipment. **Precipitation** is a method of separating medicinal products from the liquid medium. **Filtration and extraction** from the composition of solid carriers (residues) are also employed. For this purpose, copper or zinc is most commonly used. Ion exchange resins or absorbers (active coal) are also used to extract the active substances. At certain stages of the technology, residual organic solvents evaporate.

Regardless of the chemical processes and apparatus common to the chemical industry, the pharmaceutical industry with a unique production with characteristics. The high pharmacological activity and the specific application of the end products - the drugs - indicate that they even in small quantities are hazardous to the health of workers. This predetermines the leading hygiene requirement in the chemical-pharmaceutical industry: **maximum sealing of equipment and automation of production processes.** In modern high-tech and technically secured workplaces, the main profession in the manufacture of pharmaceuticals is an operator-devices in which the dynamic and static physical workload is minimized.

Along with organic solvents, as industrial poisons as an element of the chemical agent of the working environment, there are also the active substances - intermediates or end products in the manufacture of pharmaceuticals. Most often these are natural or synthetic substances. Natural products are of plant and animal origin. Here, first of all, antibiotics, steroid and peptide hormones, vitamins, enzymes, prostaglandins and pheromones have to be mentioned here. Synthetic drugs may be products of both organic synthesis and biotechnology. For some of them produced in Bulgaria, the MACs have been developed and approved **(Table 2)**.

The pharmaceutical industry uses biological agents (for example, bacteria and viruses) for use in the production of vaccines, antibiotics, blood products and biotechnology.

In many countries (also in our country) in the chemical-pharmaceutical industry is included the production of dressing materials, incl. gypsum dressings. As a rule, in these compartments (workshops), one of the leading factors in the working environment is dust. On **tabl. 3** presents the hygienic standards for dust found at these workplaces as well as for products involved in other phases of drug production. Phytochemistry and galenical production are the places where natural drugs are produced by

Tab. 2. MACs in the air of the working environment of pharmaceuticals and other chemicals involved in production

Drug formulation	Physical state	MSMAC in mg/m^3	STMMAC in mg/m^3	Specific effects
Amidofen	aerosol	0.5	–	Allergen
Ampicillin	aerosol	0.1	0.3	Allergen
Analgin	aerosol	0.5	1.0	Allergen
Acetyl salicylic acide (Acetizal)	aerosol	0.5	1.0	Irritation; allergen
Gentamicin	aerosol	0.1	0.6	Allergen
Diazepam	aerosol	0.1	0.5	-
Caffeine	aerosol	0.5	–	-
Nivalin	aerosol	0.05	–	-
Oleandomycin`	aerosol	0.4	0.8	Allergen
Penicillin	aerosol	0.1	0.3	Allergen
Resorcin	aerosol	45.0	–	-
Streptomycin	aerosol	0.1	–	Allergen
Sulfosalicylic acid	aerosol	3.0	–	Allergen
Theobromine	aerosol	1.0	–	-
Theophylline	aerosol	0.5	–	-
Turpentine	vapors	300.0	–	Irritation.
Testosterone	aerosol	0.005	–	-
Urotropin	aerosol	2.0	–	Allergen
Phenacetin	aerosol	0.5	–	-
Halothane	vapors	20.0	100.0	Skin resorptive effect Toxic for reproduction.
Chlornitromycin	aerosol	1.0	–	-
Camphor	aerosol	12.0	18.0	Irritation
Okisid zinc (as zinc)	vapor	5.0	10.0	-

extraction. The treatment of plants from which active substances are extracted, with actions related to dusting. For control of the working environment in phytochemical plants and some other departments of pharmaceutical production, guidance values proposed for provisional hygiene standards in 1992 can be used **(Table 4)**.

In the body of workers, chemical substances, incl. the active ingredients of the drugs, can penetrate through the lungs by the breathed air, when swallowed with contaminated food, beverages or smoking in the workplace and through the skin.

Many additives (binders, flavor enhancers, colorants, emulators, consistency and volume substances, preservatives and antioxidants) are used in the manufacture of medicaments which mix with the active substances, conferring the necessary physical properties and pharmaceutical characteristics in various types of dosage forms (tablets, dragees, capsules, solutions, suppositories, creams and ointments). Almost all additives do not have a therapeutic effect or are minimal and their use is not associated with a health risk to workers. This is the reason why some of them are not included in the lists of the health-damaging chemicals whose presence in the air of the working environment is limited by the Act for Health and Safety Work Conditions.

In the past, raw materials for medicines have been herbs, animal organs and tissues, minerals and salts of nature. At present, despite the exceptional variety of known pharmaceuticals, they are the product of **three technologies** based on: **organic synthesis, fermentation and extraction** from plant and animal sources.

17.11.3. FERMENTATION (PRODUCTION OF ANTIBIOTICS)

Fermentation is a biochemical process using selected microorganisms and microbiological technologies for the production of chemical products. The technology of production of microbiological synthesis is characterized by periodicity of the processes and consists of a number of successive steps:

- preparation of seed material and nutrient media;
- cultivation of producer-strains (fermentation);
- filtration and separation of culture solutions;
- drying;
- distributing and packaging of finished products.

The basic equipment consists of:

- heat-isolated fermenters that allow sterilization of vessels by pressured steam;

Tab. 3. MAC of non-toxic dusts in the air of the working environment

Type of dust	MSMAC (limits) mg/m^3	Specific effects
Gypsum containing less than 2% free crystalline silica in the respirable fraction. Inhalable fraction.	6.0	Irritation
Wood dust: Soft wood inhalable fraction. Solid wood and mixtures thereof with other wood species. Inhalable fraction.	5.0	Carcinogenic (Càò.1)
Dust of vegetable origin (sugar, starch, flour). Inhalable fraction.	10.0	Irritation
Dust from plant fibers (cotton, hemp, jute) and mixed with action synthetic fibers. Inhalable fraction.	2.0	Irritation Allergen
Talc (talkomagnezit, medical talc) containing less than 2% free crystalline silica in the respirable fraction. Fibrous particles (fine). Inhalable fraction. Respirable fraction Fiber-respirable fraction; number of fibers/cm^3	 6.0 3.0 1.0	Fibrosogenic effect

Tab. 4. Recommended values for some chemicals and natural products used in the pharmaceutical industry

Chemical, natural product	Indicative value mg/m^3	Physical state	Specific effects
Yarrow, chamomile, valerian, mead, hollyhock, raspberry	3.0	aerosol	-
Hawthorn, linden, devil's mouth, mint, comuniga, wild strawberry, marigold, wounds leaf, St. John's wort, red podabniche, patch, grass	5.0	aerosol	-
Dikain	0.3	aerosol	-
Iodoform	3.0	aerosol	-
Potassium bromate	0.1	aerosol	-
Novocaine	3.0	aerosol	-
Salicylic acid	1.0	aerosol	Mutagen, allergen

- separators, reactors for separation and purification of the preparation;
- filter-presses with periodic action;
- drum vacuum filters;
- dryers;
- packing machines.

Seed material from the microorganisms producing the respective antibiotic is prepared in sterile boxes in advance. The fermentation process begins with the cultivation of the seed material on a particular food (fermentation) medium in the preparatory fermentes. For crop and fermentation media, corn extract, peptone, glucose, soy flour, various mineral salts, glycerin, etc. are most commonly used. Further fermentation is carried out in working fermenters by continuous aeration of the culture fluid with sterile air.

Upon completion of fermentation, the culture fluid is released from micelles by means of filters. For this purpose, the native solution is processed into continuously operating rotary drum filters or filter presses. The extraction of the antibiotic from the native solution and its chemical purification is carried out in special installations by three basic methods: extraction with subsequent desorption (elution) using various synthetic ion-exchange resins and precipitation in the form of insoluble salts with subsequent crystallization. Some of the substances used in these processes are regenerated partially or completely and reused. They determine the spectrum of chemicals found in the air in the working environment and the exposure of workers.

After extraction from the chemical solution and purification, the antibiotic is dried in a drying apparatus or lyophilized. The dried (lyophilized) antibiotics are granulated and then the granular mixture is dispensed for tabletting or encapsulation with subsequent packaging. The distribution of the antibiotics in vials is carried out using different batching devices.

The risk of harming the health of workers in the production of antibiotics is created by: the moving parts of the machines; the presence of high pressure steam lines; contact with hot water and hot surfaces creating overheating microclimate at workplaces; the use of aggressive fluids with irritating and corrosive action on the skin and mucous membranes; the heavy physical dynamic loading during manual loading and loading of finished products; manual handling of monotonic elements in manual packaging; exposure to high-intensity noise.

Workers are exposed to increased concentrations of organic solvents in recovery and extraction operations. Exposure may be due to uncleaned filtration equipment or insufficiently sealed pumps, valves, or multiple interruptions during extraction and purification. Since isolation and growth of microorganisms are essential for fermentation, biosafety is ensured by the use of non-pathogenic strains, maintenance of equipment leakage and decontamination of the broths used prior to disposal.

Fermentation is safer than organic synthesis because fermentation is primarily based on "water" chemistry and technologically requires during the seed preparation and fermentation process limiting processes to the usual hazards of pharmaceutical production. The fire and explosion hazards increase during solvent extraction, although the flammability of the solvents is reduced by diluting with water on filtration and recovery. The danger of thermal burning arises due to the high volumes of steam under pressure and hot water used in fermentation production.

17.11.4. SYNTHESIS OF MEDICINES

In organic synthesis processes, organic and inorganic chemicals are used to produce drug substances. Typical of this is that series of chemical reactions take place in multi-purpose reactors and the products are separated by extraction, crystallization and filtration. The final products are dry, milled and mixed. In organic synthesis plants, basic and auxiliary equipment is similar to the light chemical industry.

Recently, the pharmaceutical industry has become a complex of multi-stage processes where single-phase products become the starting materials for the next phase until the final phase of obtaining the desired drug product is reached. The basic mass of chemicals that are intermediate or end products can be exchanged between organic synthesis workshops for a number of technical, financial and legal considerations. The majority of intermediate and final products are produced in series through reaction groups (production processes run for a limited period of time - before materials, basic and auxiliary equipment are changed to conduct new processes). Many organic synthesis plants in the pharmaceutical industry are equipped in a way that ensures maximum flexibility (mobility). This has been achieved through the construction of equipment and the introduction of process equipment, which are modified and supplemented for each new production.

Multi-purpose reactors are the first process equipment in organic synthesis. These are stainless steel vessels with pressure-reinforced walls coated with a glass or metal mixture. The nature of the chemical reactions and the physical properties of the materials (eg reactivity, corrosivity, flammabili-

ty) determine the performance and construction of the reactors. Multipurpose reactors have an outer shell and internal coils that are filled with cold water, steam or chemicals with special heat transfer properties. The reactor shell is heated or cooled, depending on the reaction conditions. Multipurpose reactors have agitators, barriers, and many internal and external openings connecting them to other vessels, equipment, and stocks of the main chemical mass. Temperature, pressure and humidity sensors are installed to measure and control reactor processes. Reactors can operate at high pressure and vacuum, depending on technology, performance, and requirements of chemical processes. The heat exchangers are connected to the reactors to heat or cool the reactions or condensed vapours of solvents when their temperature exceeds the boiling temperature, causing reflux or recovery of condensed vapours. Air pollution control devices (ie scrubbers and traps) may be connected to the outlet ports of the vessels to measure and capture the reducing gases, vapours and dust emissions. Volatile solvents and toxic substances may be discharged into workplaces or into the atmosphere, unless they are controlled during the reaction by heat exchangers or air control equipment. Some solvents (see **Table 1**) or reagents are not condensed, absorbed or trapped by air control equipment (for example: methylene chloride or chloroform) due to their chemical and physical properties.

The basic mass of chemical products are recovered or isolated by separation, purification or filtration operations. As a rule, these products are contained in the "uterine" solutions dissolved or suspended in a mixture of solvents. "Uterine" solutions can be transferred between reactors or equipment through temporary or permanent pipes or hoses, inert gas pumps, vacuum or gravity. Material transfer is most often determined by the reaction rate, critical temperatures or pressures produced in the reactor, equipment performance, and leakage potential and chemical pouring potential. Special precautions to reduce static electricity are required when flammable gases or liquids are used or generated in the production. Filling of flammable liquids through submerged water or ground pipes and maintaining an inert gas atmosphere inside the equipments reduces the risk of fire or explosion.

The workshops for organic synthesis of pharmaceuticals are characterized by a high degree of automation and mechanization, the main profession is an operator-device. The physiological characteristic of the operator-device profession is dominated by elements of nervous sensory, neuro-psychic and nervous emotional intension, due to the high requirements for concentration of attention, rapid processing of information in the control of technological processes and responsibility for the quality of production . The dynamic physical load is negligible, with continuous production cycles working on shifts, but the mode of operation is free. However, synthesis operations create many health hazards for workers. These include the dangers of moving parts of machines, equipment and pressure pipes, physical loads in the transport of materials or equipment, steam, hot liquids, surfaces and overheating microclimate at the workplace, limited space or hazardous energy source (electricity) and over norm noise.

Acute and chronic health risks may be due to exposure to chemicals during synthesis. In case of breakdown, the chemicals with acute action can damage the eyes and skin, are corrosive or irritating, cause sensitization or allergic reaction, are asphyxiants causing suffocation or oxygen deficiency. Chronic effects may cause cancer or damage to the liver, kidney, lung, or to affect nervous, endocrine, sexual systems and organs.

Occupational health and safety can be controlled by applying acceptable control measures (technological, technical, organizational, PPE). Reactions to organic synthesis can pose a serious risk of the presence of high toxical materials, fire, explosion, or uncontrolled chemical reactions that affect the environment and the population. This requires that safety measures be complex. They need to be directed in different directions: technological (preliminary tests of dynamics of chemical reactions, properties of high toxical materials); technical (setting up, operating and maintaining the equipment); organizational (training of the operational and engineering staff, preparation for dealing with disaster situations and responsibility of auxiliary means and local communications). The technical instructions shall provide an opportunity for analyzes of hazardous processes and activities to manage and reduce the risk of chemical synthesis operations.

17.11.5. BIOLOGICAL (NATURAL) EXTRACTION

Large amounts of natural materials such as plants and animal tissues are raw materials for the extraction of pharmacologically active substances. At each step of these processes, the volumes of substances are reduced by series or group of processes to the final product, and this sometimes lasts for several weeks until the desired amount of finished product

is achieved. Solvents are used to extract water-insoluble fats by means of the extraction of the final substance. The acidity (pH) of the extraction solution or waste water can be corrected by neutralization with acids or bases. Metal compounds often serve as precipitants, and phenolic compounds for disinfectants.

Health and Safety at Work. Some workers develop allergic reactions or irritation of the skin in contact with different plants. Animal products may contain pathogenic micro-organisms (if no precautions are taken). Workers are exposed to solvents and corrosive liquids in the process of biological and natural extraction. There is a risk of fire and explosion when storing, processing and recovering flammable liquids. The movement of mechanical parts, hot steam, water, heated surfaces and workplaces with overheating microclimate, high noise are risk factors.

Safety measures are determined by the magnitude and nature of the reduction in the large bulk of plant and animal materials and are related to the limitation of open solvent extraction activities. Fire and explosion, solvent exposures, irritants and corrosive liquids are detected during extraction and recovery depending on the specifics of the processes and the contamination of the equipment.

17.11.6. PRODUCTION OF READY-TO-USE PHARMACEUTICAL FORMS

Medicinal products are formulated as dosage forms prior to use. The active drug substances are mixed with the necessary pharmaceutical additives such as binders, fillers, flavors and added volume, preservatives and antioxidants. These ingredients may be dried, milled, blended, compressed or granulated to achieve the desired properties. Tablets and capsules are the most commonly available ready-to-use pharmaceutical formulations for oral use; other formulations are sterile injectable solutions and eye drops. Typical operations for the manufacture of finished dosage forms are: weighing of active substances and excipients; mixing; granulation; precompression; re-granulation; sieving; tabletting, which includes the manufacture of tablets, film-coated tablets or dragees and encapsulation.

The pharmaceutical compositions may be compressed by wet granulation or direct compression to obtain the desired physical properties prior to final shaping of the molds. In wet granulation, the active ingredients and additives are wetted with water or solvents to obtain granules of different sizes. These granules are dry, mixed with lubricants (e.g., magnesium stearate), anti-adheres or binders and then compressed as tablets. During direct compression, a metal matrix maintains the measured amount of drug mixture while compressing the tablet. Drugs that are not stable enough to wet granulation or can not be compressed are injected. Injection, or dry granulation, mixes or presses relatively large tablets which are coated and sized to the desired size and then re-extruded to final tablets. Mixed and granular materials may be encapsulated. The hard gelatin capsules are dried, stacked, filled, and joined to encapsulation machines.

Liquid forms can be produced as sterile injectable solutions or eye drops; liquids, suspensions and syrups for oral ingestion; tinctures for dermal application. Conditions of production, equipment and raw materials are subject to strict control in the manufacture of sterile solutions to prevent dust and microbial contamination. All used equipment and work surfaces are cleaned to minimize contamination. High pressure and temperature water is used to purify from bacteria and other contaminants of sterile water for injection solutions. Parenteral solutions prepared with fully demineralized and no-pyrogenic water are sterilized by dry or moist heat under high pressure with bacterial filters. Solutions for oral or topical use do not require sterility, but eye solutions should be sterilized. Solutions for oral administration are prepared by mixing the active drug substance with a solution or a preservative against molds and microbes. Liquid suspensions and emulsions are produced by colloidal mills and homogenizers. Creams and onyments are prepared by attaching the active ingredients with vaseline, fats or softeners before placing them in metal or plastic tubes.

Major manufacturing operations

The weighing and distribution of solid and liquid substances is a common operation in the pharmaceutical industry. Generally, workers distribute and weigh the substances with precision electronic scales, manually, using blades for solids and pouring or pumping for liquid ingredients. The air in the working environment is contaminated with dust, vapors or aerosols. Local exhaust ventilation is required. Work surfaces must be smooth to prevent dust and dampness and allow thorough cleaning.

The loading and unloading of solutions and solids from containers or equipment is often a recurring operation in the pharmaceutical industry. It is usually done manually and this is associated with significant physical load, but other methods are also implemented: gravity, mechanical or pneumatic transport system. Special equipment, system for transfer and control at loading and unloading, protect workers from exposure to highly toxic chemicals. Loading by gravity from closed containers, vacuum

transfer, pressure or pumping reduces emissions of volatile substances. When transferring solutions and solids through open systems, conditions are created to contaminate the working environment with dust (aerosols) and vapors, which requires control of their contents for concentrations above the MAC.

Liquid mixtures are separated by methods based on their physico-chemical properties: relative weight, solubility and viscosity. This is also a common and often performed operation in the manufacture of medicaments. Toxic mixtures should be transferred and separated into closed containers connected to pipelines to reduce worker exposure. Eyewash showers and baths in case of toxic and corrosive fluids on the skin, mucous membranes and eyes should be located in close proximity to work places. It is necessary to provide for measures against spillage, fire and explosion when easily combustible mixtures are separated.

Very often, in the pharmaceutical industry, it is necessary to **transport solutions** from storage vessels, containers or reactors. The best option is that equipment for this purpose minimizes the risk of spilling and exposure of workers. Solutions are transferred through a system of pipelines that pass through pumping stations. These stations are the places where gassing and spillage are most often recorded. To reduce the risk, seals or seals of hoses and pipes are used. Closed-chamber drying or water purification systems are also required. Well-sealed, closed vessels, containers and pipelines are particularly needed when large volumes of solutions are transferred. Special precautions are taken when using inert gas under pressure to transfer solutions as the release of volatile organic substances or other toxic pollutants into the air is increased. Recirculation or condensation of waste gases and vapors reduces atmospheric air pollution.

The solids in the solution are separated by **filtration**. The filters used have different designs, different volume and possibilities for controlling liquids and vapors. When filtering toxic materials using open filters, workers are exposed to liquids, wetting agents, aerosols and vapors during loading and unloading of the filters. Closed equipment should be used in the filtration of extremely toxic and hazardous materials. Filtration should be carried out in places where spillage control and good ventilation are provided. The vapours of the liquid phase must be discharged through ventilation system in closed containers and controlling their discharge into the air, through emission capture devices (condensers, scrubbers or absorbers).

Solid and liquid substances are mixed (homogenized) in mixers (mixer granulators) by bonding operations to produce solutions, suspensions, syrups, flavors and pastes. It is recommended when mixing toxic materials using special equipment and transfer system. Buffering agents, detergents and antibacterial agents used to neutralize, clean and destroy biological agents can be dangerous for workers. Washing showers and eyewash cleaners reduce injuries if workers accidentally come into contact with corrosive or irritant substances. Due to wet surfaces, workers must be protected from electric shock and electric trauma. Combustion hazards create steam and hot water used to mix or clean the equipment. Occupational accidents due to burning or slipping and falling are prevented by isolating hot surfaces and maintaining the floors dry without spillage of liquids.

The dry and wet substances are **granulated** to change their physical properties. The granulators are of different construction and with devices for automated control of the mechanical danger and the air pollution with dust and aerosols. Closed granulators are ventilated by air control devices that reduce solvent vapours or dust emissions in the environment and the work places. Exposure of workers increases with manual manipulation with materials, requiring greater physical effort when loading and unloading. Mechanical equipment (lifting platforms, elevators and pallet jacks) is used to relieve physical stress. Eyewash showers and baths are needed in contact with solvents or irritating powders.

The water- and solvent softened solids are **dried** by a variety of operations. Fluid dryers are often used, but dryers may have different design and vapor control devices. Flammable solvent vapors and explosive hazardous powders can form a flammable and explosively hazardous environment. This attaches particular importance to ventilation to reduce the hazard. Dilution and maintenance of acceptable concentrations also reduce the risk of fire and explosion. It is necessary to control exposure to solvent vapours when wet briquettes are handling, and dust when dry products are discharged. When loading and unloading the troughs, the bins or containers of the driers, a significant dynamic physical load is observed. Mechanical equipment (drum-reel, elevator or work platform) reduces workload. In the close proximity of the workplace there should be eyewashes and showers in case of contact with solvent and powders.

The dry solids are **milled** to achieve a certain dispersion and free-flowing powders are obtained. The mills have different designs and devices for mechanical protection and control of air pollution. Hammer mills are often used, which are very noisy and heavily dust the working environment.

It is necessary to check in advance the physical properties and toxicity of the grinding materials. Explosion protection includes: the installation of dust-proof electrical equipment, earthing devices, electrostatic discharge; the installation of providing discharge valves in closed mills and the construction of anti-detonation panels in the walls. These measures are due to the explosiveness of certain drug substances and excipients and high concentrations of dust in the equipment.

The dry substances are mixed until a homogeneous mass is obtained. Mixers (homogenizers) have a different construction and sensors to control the risk of mechanical trauma and air pollution with dust. Workers are exposed to dust, excipients and mixtures when loading and unloading the mixers. During mixing by a flange with a return valve, the releasable dust is reduced. The loading and unloading of the mixers is associated with a dynamic physical load which is limited and reduced by the use of work platforms, winches and drums, pallet trucks, etc.

The dry substances are **compressed** or charged for compaction in order to change the state of dispersibility. Compression equipment is different in design and in various ways the risk of mechanical damage at work (mechanical trauma) and dust removal is controlled. These operations are accompanied by a high noise level. Encapsulation of the noise source, reducing equipment vibrations, changing workplaces and using headphones and earmuffs reduce the impact of noise.

Tablets and capsules are the most commonly produced oral dosage forms. Tablets are compressed or formulated using modern tablet machines that are equipped with computer control. The tablets contain mixtures of drug substances and excipients. They are "no backed tablets" and coated with water-soluble mixtures or solvents (film-tablets or dragees).

Pre-tablets are deodorized and fed to the dredger machines. Dyeing of tablets (dragees) is done in drums (dredgers), which are loaded with paint, hot distilled water, talc and titanium dioxide. Acetone, isopropanol, castor oil and glycerin are also used. Film coating (lacquering) is carried out with acetophthalate cellulose, ethanol, ethyl phthalate, ethyl acetate, sugar, gum arabic, gelatin and titanium dioxide, talc, demineralised water, coloring agent, polyethylene glycol, glycerin. The gloss solution contains polyethylene glycol, isopropanol, acetone and glycerin.

Capsules that are produced may be soft or hard gelatin shells. Tablet presses, encapsulating machines and coating drums are of different construction and equipment for controlling mechanical trauma and dust removal. Workers are also exposed to solvent aerosols when coating the tablets. Modern machines have a high capacity of activity and have control devices for excreting vapor and solvent aerosols. They are connected with ventilation to remove volatile organic substances. It is possible to re-use the recovered solvents. Modern tablet machines and capsule machines are also equipped with noise-reducing panels, dust extraction during operation. Wearing PPE also reduces the risk of encapsulation and tablet noise.

The production of sterile products in pharmaceutical practice requires modular equipment in clean workplaces and working surfaces, and high-efficiency dust extraction ventilation. The principles and practices for providing clean work places are similar to those in microelectronics. Workers wear protective clothing to prevent contamination of the products. Technologies for the production of sterile products include lyophilization, use of liquid or gaseous disinfectants, effective ventilation, isolation of modules by maintaining differential pressure, and loading and filling facilities.

Chemical danger creates the use of liquid disinfectants (formaldehyde and glutaraldehyde) and gases for sterilization (ethylene oxide). Where possible, it is advisable to choose less toxic substances such as alcohols, ammonium compounds. Sterilization of raw materials and equipment can also be carried out under steam pressure. Sterilization of vessels should take place at a specific place (room) with remote controls and a toxic substance control system, without air recirculation (toxic emissions to be removed). Workers should be instructed on the rules of safe work and personal responsibility in an accident. Gas sterilization rooms must be provided with wacuum system and air must be cleaned to reduce emissions to workplaces. Emissions from sterilization rooms should be through a ventilation system that includes carbon absorption or catalytic conversion to reduce environmental pollution. Securing safety is also provided for contact with steam and hot water, moving parts of machinery when washing, filling and packaging the finished products, high noise and monotone manual operations.

Packaging in pharmaceutical manufacture is done by machines and manually. The finished ready dosage forms can be packaged in different types of containers (plastic or glass bottles, foil blisters, bags or sachets, tubes or sterile ampoules). Mechanical equipment filled, encapsulated, labels, put in boxes and packs the finished products into shipping containers. Employees serving packing machines must be protected from their moving parts. Machinery must have signal lamps that are under voltage

and emergency shutdown buttons. The closure and isolation of equipment reduces noise and vibration during packing. The use of PPE (headphones and earmuffs) reduces exposure to noise. The ergonomic design of the machines increases labour productivity, improves the comfort and safety of workers and reduces the risk of injuries from forced labour pose, physical loading and monotonous work.

Unusual conditions are created when **cleaning, repairing and maintaining** the equipment and work places. Sometimes this is often done due to the change of the manufactured product, increasing the risk of performing non-standard tasks. Workplaces and surfaces can be contaminated with toxic substances or drug substances, which requires them to be thoroughly cleaned. The cleaning is done by washing or wet wiping and drying or vacuuming the powders. Dry suction and blowing with compressed air is not recommended. When cleaning dangerous substances and high active pharmaceutical powders, vacuum cleaners with microporous filters are required. Explosion-proof equipment and resistant materials are required to vacuum explosive powders. Eyewashes, showers and PPE reduce the risk of damage by accidental contact with corrosive and irritant substances - detergents and cleaning solutions. In the case of repairs or maintenance of equipment, workers sometimes have to carry out tasks related to the risk of mechanical, electrical, pneumatic or thermal injuries. Careful monitoring and control of these tasks is important preventive measures. This prevents fire, explosion or other serious health risks.

Laboratory activities (operations) in the pharmaceutical industry are diverse and accompany the various drug production technologies. They generate a biological, chemical or physical risk depending on the specific agents, operations, furnishings and work practices. There is a difference in the activity of the laboratory staff performing research and development activities and those who assess the quality of production and control the technological discipline of production. Laboratory activities should be evaluated individually, although general requirements for good laboratory practice are applied. Clearly defined responsibilities, training and information, training to mastering safe working rules and control measures as well as emergency situations are effective means of managing safety, health and environmental protection.

For example, safety and reduction of health risks from flammable and toxic substances are achieved by reducing their stay in laboratories and by keeping them in separate rooms. Laboratory tests and operations related to the release of toxic air contamination should be carried out in laboratory fireplaces. For biological protection, shields (lids) are used to provide airflow into the laboratory stove down and inward, preventing contamination of the work rooms with microorganisms. Safety training and instruction, training on personal responsibility in case of fire and spillage are important preventive measures. Do not eat and drink at work places in the laboratories. Particularly dangerous operations are carried out under the supervision of observers. The requirements for good laboratory practice include separate collection, treatment and storage of hazardous waste.

Physical factors related to health risk such as ionizing radiation and EMF are certified and the apparatus generating these factors is operated according to the specific rules.

17.11.7. PHARMACIES

The hygienic requirements for the structure, order and organization of work in the pharmacies are in line with the Health and Safety at Work Act and are regulated by the current regulations and other by-laws of the Ministry of Health. All pharmacies have medicinal products in the pharmacological groups according to Annex 1 (medicines licensed with color prescriptions require a license from the MH) and are divided into 2 main types. The first type of pharmacies provide medicines for the citizens and the second one - for the medical institutions with a stationary.

Pharmacies that provide medicines **to citizens** are 4 types:

- pharmacies that release pharmaceuticals except for extemporaneous dosage forms (only work with ready-to-use formulations);
- that release pharmaceuticals pharmacies, including extemporaneous forms,non-ophthalmic formulations and solutions for parenteral administration (do not prepare sterile formulations);
- pharmacies that release medicinal products, including extemporaneous formulations and the preparation of eye formulations;
- pharmacies that release herbs and herbal mixtures (herbal pharmacies), including extemporaneous forms.

In all pharmacies that release medicines to citizens, the cosmetic and sanitary hygienic devices listed in Annex 2 of Ordinance 8 / 23.06.2000 can be sold.

The pharmacies that meet the needs **of health establishments** with stationary are two types. Depending on the activities performed, they supply:

- Medicinal products, including extemporaneous formulations without preparation of eye

formula and solutions for parenteral administration;
- Medicinal products including extemporaneous formulations and eye formulations and solutions for parenteral administration.

This approach to dividing pharmacies suppose a different technological process that determines **the set of premises** and specific hygiene requirements. Pharmacies for dispensing pharmaceuticals of citizens who only work with ready-made forms and have only one work place are located on a total area of at least 20 m^2. On this area there is a reception room and a storage room with a total area of not less than 15 m^2 and a sanitary unit with insulated entrance hall and sink. For each additional workplace, a further 5 m^2 is required.

Pharmacies in which ready-to-use and extemporaneous formulations are realease, with one work place, but do not prepare eye and parenteral formulations are disclosed on a total area of 30 m^2 consisting of a reception and storage area with a total area of at least 15 m^2, one-work place assistant room, distilled water box and wash basin for the dishes and the equipment for preparation of extemporal formulations with a total area of not less than 10 m^2, sanitary unit with insulated entrance hall and sink. For each additional workplace, a further 5 m^2 is required.

Pharmacies for ready-to-use and extemporaneous formulations with one workplace and eye formulations, require premises with a total area of 40 m^2 consisting of a reception and warehouse with a total area of at least 15 m^2, an assistant room with one working place, box for distilled water and a washbasin for washing dishes and apparatus for the preparation of extemporaneous dosage forms with a total area of not less than 10 m^2, a work area for the preparation of eye solutions of a total area of not less than 10 m^2, box for aseptic preparation of eye formulations and sanitary unit with an insulating entrance hall and a sink. For each additional workplace, a further 5 m^2 is required.

A herbal pharmacy with a workplace where herbal mixtures and teas are prepared and distributed herbs must have a minimum area of 50 m^2, which is distributed between a reception and a storage room with a total area of at least 30 m^2, an assistant room with one work place with an area of not less than 15 m^2 and a bathroom with a shower and an insulated entrance with a sink. If a herbal pharmacy is released and ready-made dosage forms have to be aditional, a separate work place with an area of at least 15 m^2 shall be established for the storage and sale of these drugs. Where an extemporaneous dosage form is prepared in the herbal pharmacy, an additional room of at least 15 m^2 in one work place, a box of distilled water, and a sink for washing the dishes and instruments for preparing extemporaneous dosage forms is required.

The pharmacy to meet the needs of a medical establishment, including extemporaneous forms without eye drops and solutions for parenteral administration, should have a total area of 0.50 m^2 per bed but not less than 50 m^2. This area is distributed between a reception room, an assistant room, a sanitary unit with an entrance hall and a sink according to the requirements of the current norms for the design of hospitals of general type.

In the hospital pharmacy for medicinal products, including extemporaneous formulations with the preparation of eye drops and solutions for parenteral administration, the premises, areas and conditions for this type of activity and pharmacy are brought in accordance with good practice in the manufacture of sterile dosage forms.

The premises of the pharmacies are completely isolated from premises for other activities. There must be **functional links** between them. The reception room should be connected to the assistant with a direct connection and convenient access to the storage room. The assistant room has a convenient connection to the dishwasher. It is allowed to use premises on other levels (basement or second level room), but their area is not included in the pharmacy area.

The following **building requirements** are applied to the pharmacy premises: the clear height of the premises must be not less than 2.50 m. The walls in the dishwasher, aseptic box, the apparatus room, the dispenser box, and the sanitary unit are covered with a waterproof coating, allowing water cleaning and distillers at a height of 2 m. Floor coverings at all pharmacy premises must be wateproof and easy to clean. In the sector where pharmacists work, floor coverings should be easy to clean and be warm.

A mandatory requirement is the presence of natural **lighting** in the reception and the assistant room. Combined lighting or artificial lighting only is allowed in the storage room, the aseptic box, the apparatus and the sanitary unit. The artificial lighting in the rooms shall be not less than 300 lx. Workplaces in the production premises (reception and assistant) are provided with local lighting. Aseptic room and entrance hall to it, have bactericidal lamps.

A mandatory requirement is the presence of **natural ventilation** in the pharmacy's premises. Forced (artificial) ventilation is required in all indoor spaces to provide a maximum allowable dusting norm of 2 mg/m^3.

Heating, ventilation and air conditioning systems must ensure healthy and safe working conditions

and ensure compliance with the regulatory requirements for a microclimate of the working environment.

The water supply and sewerage of pharmacies must comply with the relevant building codes. Pharmacies should be provided with a sufficient amount of hot and cold water that meets the drinking water regulations.

The pharmacies are furnished with **suitable working furniture**: racks, tops, working tables, chairs and height changing chairs, etc., which meet the ergonomic requirements and are made of materials that allow wet cleaning and disinfection. Workplaces in the assistant room where extemporaneous forms are being prepared must be equipped according to the minimum standard set out in Annex 5 to Art. 14 of Ordinance 8 / 23.06.2000.

Distillation of water for the preparation of extemporal forms is carried out in separate rooms. Where there is no separate room, the water receiver is placed in a tightly closed glass box. In the same room, it is forbidden to wash contaminated containers and store easily evaporable drugs.

Distilled water is stored in cool, clean and well-sealed containers, pre-washed with distilled water. The vessel is the date of receipt of the distilled water and it is stored for not more than 48 hours.

Medicinal products, except for extemporaneous dosage forms, **are stored** on shelves or in tightly closed cabinets. Medicinal products in transport packs and sanitary and hygienic materials are stored on wooden grills. Strongly smelling, disinfecting, deratizing and cosmetic products, sold in pharmacies, are stored in separate, well-locked cabinets. Pharmacies licensed to dispense neuroleptics and psychotropics, containing narcotics, are stored in a metal cabinet or safe box marked "Drugs containing narcotic substances" and a list of medicinal products with the maximum doses indicated. In the same cabinet, the utensils needed to work with these drugs alone, are kept. Medicines that have expired, with a primary or secondary packet disorder, are stored separately at a designated site (with a permanent indication that the medicines are blocked).

Various factors influence the properties of the formulations. Among them are the **light and the parameters of the microclimate**. Light has a pronounced photochemical effect due to the high energy ultraviolet spectrum. Under the influence of UV rays, some photosensitive components of the drugs are destroyed. Impaired storage temperature and / or high humidity in the premises can also affect the medication. Unfavorable microclimatic conditions are fatal when preserving bio-products (vaccines and sera). They are stored in refrigerated chambers or refrigerators at temperatures not higher than 8 ° C. All others may store "in the dark, in a dry and cool room" at temperatures of 8-20 ° C and a relative humidity of up to 60%, unless otherwise specified in the current Pharmacopoeia.

Herbs are kept separate from other drugs in tightly closed cabinets. High humidity is also an unfavorable factor in herbal pharmacies, because in combination with high temperature it creates a danger of the development of fungi and molds on medicinal plants.

Extemporaneous dosage forms have an adverse effect on the already mentioned **light, high temperature and high humidity**. The quality of the extemporaneous forms is influenced by the presence of gas and dust pollutants of air and microorganisms. **The gas pollutants** (vapors of ammonia, iodine, formalin, ether, chloroform in the air of the pharmacy, or penetrating from the outside of highly polluted air containing significant amounts of sulfur dioxide, nitrogen oxides, CO and dust) are adsorbed and enter chemical interactions with some ingredients of the formulations prepared by giving them unpleasant odor and deteriorating quality. Powder pollutants are also carriers of microorganisms. They fall into the air with coughing, sneezing and talking to the pharmacy's clients, along with the dust of their clothes and the outside air.

For drug protection, they need to be packed in hermetically sealed or tight-fitting containers with the appropriate properties required by the pharmacopoeia and stored under appropriate conditions. In the preparation of extemporal dosage forms it is necessary to respect the adequate technology and the rules of personal hygiene. When preparing sterile antibiotic formulations, all sterility requirements and good manufacturing practice rules are respected.

The sanitary and hygienic regime in the pharmacies provides daily and immediately, upon contamination, to perform wet cleaning and disinfection of the floor, the surfaces of the furniture, the equipment, etc. according to Methodological Guideline 0-20 for modern ways and means of disinfection in healthcare facilities.

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17.12 ARMED FORCES

The State supports the armed forces to ensure its territorial and political integrity. For the past 20 years, the main tasks of the Armed Forces have been added to operations other than war - humanitarian, peace-imposing, peace-keeping, anti-terrorist, and others. In carrying out tasks related to these operations, soldiers work on all types of terrain, in air and under water, in different seasons and in various, often extreme, climatic conditions, handling specific combat techniques against an armed enemy.

In order to effectively respond to crises and to reduce combat losses, soldiers must possess basic skills, such as the proficiency of various weapon systems, accurate shooting, high physical training and aerobic endurance, discipline, ability to make quick decisions. Their maintenance is achieved through constant intensive training, which leads to the accumulation of physical fatigue. Incorrect or excessive training may result in serious injury. Maintaining a high level of readiness leads to frequent violations of the work-rest regime and to limitations in social contacts. Battle duties associated with prolonged awaking, intense mental and sensory loading contribute to the accumulation of physical and mental fatigue. Numerous other factors (noise, electromagnetic fields, long-distance transport) and increased media attention lead to increased intension among military personnel and especially commanders. Detachment for a long time from family and relatives and the possibility of immediate opposing enemy's actions contribute to the increase in the effect of stress. The nature of the work of the militaries, and especially in the land forces, poses a high risk of injuries from drafts, shrugs, explosive injuries, and varying degrees of thermal injury due to enemy activity or impotent handling of combat equipment. In recent years, anti-infantries mines and improvised combat devices have been increasingly used, which are difficult to detect, have a considerable striking power, and are made from commercially available materials. There is a high risk of damage from weapons of mass destruction (WMD) - nuclear, radiological, chemical and biological.

Energy consumption: Intensive mental and physical efforts are associated with increased energy consumption by the body (4200-4600 kcal for field operations, 4200-6000 kcal for foot marches, 4200 kcal for driving armoured equipment, 4000 kcal for pilots) and for dehydration and loss of electrolytes. In order to correct the energy consumption, it is necessary to include a number of nutritional supplements. In the event of crisis operations, logistical provision of normal nutrition is difficult. This requires the maintenance and use of special individual food rations of different energy and low volume. The daily survival ration contains at least 150 grams of carbohydrates and is designed to survive in extreme emergency situations for up to six days. The daily emergency ration has an energy value of 1000 kcal and maintains the fitness for 24 hours. Individual fighting rations have an energy value of 3200 kcal with a balanced ratio of the main nutrients and are used for no more than 30 consecutive days until normal military nutrition is restored. Humanitarian daily rations contain no more than 2200 kcal, excluding meat if they are used in areas with an ambient temperature above 35 ° C but if the temperature is around or below 4 ° C their composition is mainly meat - chicken or veal.

Temperature factor - militants often perform operations in extreme climatic conditions. The temperature of the environment and the nature of the work can lead to heat or cold damages.

Thermal effect - the increased heat output associated with the significant physical loads in the fulfillment of the assigned tasks combined with the elevated temperature of the environment is a prerequisite for the development of thermal damage. The use of ballistic protection means (armor, helmets) and WMD protection means further exacerbates the release of heat from the organism and increases heat stress on the body. Means of protection from WMD create their own microclimate - the air enclosed between the body and the equipment is heated by body temperature and is saturated with moisture. In this way, the immediate surrounding military environment becomes extremely hot and saturated with moisture. For the overheating, the microclimate also helps in combat machines and ships, as the temperature in them often exceeds 15-20°C that of the environment.

Cold effect - pronounced cold stress reduces physical capacity, affects the moral qualities of the individual, and can lead to the development of cold

disabilities. It arises not only from extremely low temperatures, but also from the combined effects of moisture and low temperature and falling into cold water. Cold weather contributes to increased respiratory diseases and non-military injuries (frosts). On the other hand, the operational requirements for intense physical effort, waking and inadequate nutrition disrupt the body's effective physiological response to the cold.

High factor - is characteristic of military operations in mountainous areas. Fast altitude climb at altitudes above 1500 m reduces the physical and recognizable abilities of militaries. Reduced atmospheric pressure and reduced oxygen partial pressure increase susceptibility to mountain disease. Low temperatures predispose to generalized hypothermia or local frostbite and combined with alpine conditions are a prerequisite for more frequent traumas. The inability to provide adequate sanitary measures increases the risk of developing infectious diseases transmitted by water or food.

Infectious diseases - the nature of military service accommodation under field conditions limits development activities to deployment areas. There are difficulties in complying with the rules of individual and collective hygiene. The placement of a small area with a small amount of personal space for each military is a prerequisite for a wide spread of airborne infections. The difficulty of providing sufficient water for drinking and domestic purposes leads to the development of waterborne epidemics. Owing to contamination of food products, epidemic outbreaks of food toxico-infections and intestinal infections are no-rare. The nature of military service places them in direct contact with flying and crawling arthropods, carriers of transmissible infections and natural-focal diseases, as well as increases the likelihood of poisoning snakes, spiders, scorpions and other dangerous representatives of flora and fauna.

Different types of armed forces have specific conditions depending on the type and nature of the tasks they perform.

Land Forces - One of the most important features of land troops is the use of various fortification systems for temporary and lasting protection of living force.

The open structures - trenches, ditches - are largely protected against the impact of conventional weapons and weapons of mass destruction. However, the military is exposed to adverse factors such as extreme temperatures, humidity, snow and rain, wind, static intension, soil composition, groundwater, asenization. The negative impact of these factors is increased by extending their stay. There are diseases of the respiratory tract - rhinitis, tracheobronchitis; and so-called "trenches diseases" - rheumatism, angina. A particular danger is the so-called "trench foot" - localized frostbite of the lower limbs, which can lead to amputation of the affected limb in severe cases.

Closed facilities - army shelters, bunkers, command posts, artillery fire protection facilities - isolate environmental militaries and create their own microclimate. They allow staff to reside for longer, to rest, and not to use personal means of protection against WMD. The parameters of the microclimate in these facilities are generally outside the hygiene standards, due to the staff stay in them and the combat operations they perform. The temperature in them can rise up to 30-40 ° C and represent a prerequisite for heat shocks, while the reinforced concrete constructions, which often build their structure, determine the possibility of overheating during the summer and cooling in the winter. The breathing of even one military can lead to hygienic norms being exceeded with condensation. On the other hand, breathing leads to an increase in CO_2 concentration and a reduction in O_2 partial pressure. In addition, increased concentrations of gun powder and exhaust gases, vapours from heated oil, battery gases, and more are found in the facilities. The duration of residence of personnel in a medium saturated with gunpowder is determined by the CO content in 1 liter of air. At a CO concentration of 0.25 mg/l the stay should not be longer than 25 minutes, at 0.50 mg/l the stay should not be longer than 10 minutes and at 0.75 mg/l the stay should not be longer than 5 minutes. Accumulator gases and aerosols containing H, O, P1, H_2SO_3 and those of the oils irritate the eyes, nose, trachea and bronchi.

Organized movement - "Marsh" is aimed at reaching a certain area within a given period of time, while maintaining a fighting. It can be carried out on its own walk (classic foot march), on cars or other combat equipment, by rail, sea or air transport or by combined means. In the ***classical walking march*** there is a heavy physical work (more than 4000 kcal is spent for a 30 km transition), depending on the conditions - the march is summer, winter, mountain and night. It can also be normal (up to 30 km per day) and forced (over 30 km per day). The main risk for summer foot march is the occurrence of thermal injuries - sun or heat stroke; the winter march is mainly the development of cold traumas and common cold; in the mountain marshes problem is the increased energy consumption due to the nature of the terrain and the danger of the development of mountain sickness in areas with altitude above 1500 m. When using moto machinery - motomarch - on the staff influence the

constant static intension (deteriorates the work of the lungs and develops stagnation of blood in the lower limbs), swinging, vibration, noise (in cars 80-90 dB/A, and in armored equipment 100-120 dB/A), ambient temperature (in summer creating conditions for overheating, in winter colds), the speed of air, dust, exhaust gases from the machines contain CO, and in war machines at shooting increases and the concentration of gun-powder gases and conditions are created for intoxication, any combination of chemical, radiological and biological agents.

Military equipment - optical quantum generators (OQG) (laser rangefinders and systems for fire control) have characteristics of weapons of electronic warfare, but act within the legal geometric visibility, have a smaller range of action and are influenced by weather conditions. In current conditions, the use of the laser beam to target and target intelligent bombs and jets is increasing. Different application missiles (for airborne, anti-tank, etc.) are also laser-directed. The OQGs are used for a laser location, such as laser reconnaissence instalations, as well as for laser simulators, imitation laser systems, laser telecommunication systems, laser gyroscopes, radar location stations suppressing devices (RLS). OQGs are also used in military medicine, mainly for surgical purposes. As combat weapons, lasers have characteristics similar to those of electronic weapons (REW), nuclear, biological and chemical warfare. They mainly interfere with or disorganize the operation of receiving devices that operate in the light or within the vicinity of the electromagnetic spectrum. Combat laser weapons are primarily aimed at destroying observation systems (periscopes, telescopes, night vision devices, etc.) When a laser beam falls directly on the exposed parts of the body and the visible mucous membranes, local burns occur from the first to the fourth degree. In the eyes - to irreversible disturbances of the functions of the eye.

Air Force - Personnel are divided into two main groups - Flight and Ground. Pilots of **combat aircraft** are subjected to the combined effects of gravity and acceleration during flight as well as altitude hypoxia. Particularly high is the load on the pilots of supersonic aviation, which are subject to high, rapidly changing overloads. Very high linear, radial and angular accelerations (5-7 G with a duration of 10-40 seconds) act on their body. Mechanical (anti-wearing suit, positive pressure breathing), physiological (muscle contractions, closed epiglottis expiration) and exercise (centrifugal training) methods are used to increase tolerance to these effects. Adaptation to hypoxia occurs with periodic residence in barocameras. The risk of decompression in military aviation is much greater than the civilian, due to the extreme overloading of aircraft and the possibility of hostile action. An important problem is the accumulation of fatigue associated with long-term stay in forced pose, information overload and the need for quick decisions. As a result of the operation of the aircraft engines, vibrations occur which are more pronounced in taxiing, take-off and landing. Flight turbulence causes aperiodic low-frequency thrusts. The spatial disorientation that occurs in these cases is also observed in flights at extremely low altitudes. For propelled transport aircraft, the major problem is the intense engine noise. Exposure to cosmic radiation is a problem in high altitude flight.

Helicopter pilots operate with relatively unstable aircraft, which implies constant sensory, physical and mental intension. The flights take place near the immediate vicinity or overcome various natural or anthropogenic obstacles. Spatial orientation can be lost in the absence of landmarks (flight over water basins, snow-covered surfaces). The use of night vision devices limits peripheral vision and makes orientation difficult. At the same time, they are an easy target for attack by various weapon systems, including light arms. Problem of take-off and landing is the generation of swirling powder aerosols, which may contain biological agents. Cabin ventilation is provided by a contrary airflow in the absence of sealing - there is a risk of crew intoxication when the fuel and lubricants are incompletely combusted. This specific risk is the highest in hovering mode when the cab is in the exhaust gases area. The level of vibration and noise is much higher than that on airplanes, the noise often reaches 110-140 dB/A. The vibrations are low-frequency, directed in the axial direction of the helicopter and are most pronounced in landing and take-off. They contribute to decreased visual acuity, difficulty in speech communication, increased strain of paravertebral muscles with subsequent overwork. Chronic vibrational effects often result in the development of vibrational disease. A specific problem of chronic vibrational effects on the vertebral and intervertebral discs is the appearance of back pain, disc hernias, protrusions, and radiculoneuritis.

The engineering and technical staff is subject to the impact of on-board and aerodrome radars, radio, navigation and target systems, powerful EMF army generators, sources of ionizing radiation, adverse climatic conditions, intensive noise and vibrations, dust, chemicals (kerosene, products from the decomposition of mineral oils, CO, CO_2, nitrogen oxides, acrolein, hydrocarbons, aldehydes, ketones, hydrogen sulphide). There is a high nervous-emotional intension, frequent disturbances of the work-rest regime, monotony of work, limited contacts in the workplace.

Zenith-rocket troops and radio-equipment troops - the soldiers are exposed to electromagnetic radiation around the antennas of radio stations and radio transmitting centers, in the transmission rooms where are the generators, feeders, waveguides, antenna systems of radiotechnical devices, magnetrons, klystrons, waveguides, also from the parasitic rays in the cabins and rooms, and in emergency situations it is necessary to work in the field itself. Stationary personnel are exposed to mild X-rays, intense noise (over 95 dB/A), vibrations, CO, CO_2, ozone, nitrogen dioxide, SML contacts, insufficient brightness, screen brightness, overheating microclimate (to 45 ° C), forced posture, great nervous intension combined with high responsibility, frequent disturbances of the work-rest regime. In radiolocation stations in scanning and rotation mode, the antennas irradiate work areas and living areas in the grounds, with a large number of staff not directly involved in their work. Those working on the repairs and tuning of the radio waves generators are working with a removed shield in overladen measuring rooms. The ZRTs personal is in direct contact with rocket fuels (solid and liquid, one and two components) and oxidants (nitric acid, liquid oxygen, nitrogen oxides, hydrogen peroxide).

Naval Forces - Personnel is exposed to the combined impact of various factors of origin and nature. Confined space hinders staff movements and increases the incidence of injuries to the locomotor system. Deck and mast radar stations and radio stations affect all personnel. Meteorological conditions are dynamic and varied and mainly affect the deck command. Daytime heating and nighttime cooling on the ship's deck make it difficult to maintain the microclimate and load the body's thermoregulatory function. During long voyages, crews often change several climatic zones, which puts the body's thermoregulatory adaptation. The temperature in the engine compartments often exceeds 45-50 ° C. Noise is intense and constant and prevents sufficient rest. Swinging of ships irritates the vestibular apparatus and causes kinetosis. Onboard submarines the main problem is maintaining air cleanliness and preventing its contamination. Personnel engaged in diving activities are at risk of developing oxygen toxicity from inhaled air and decompression (caisson) disease.

Medical insurance - should focus on the prevention of health risks. When introducing new armaments and equipment, it is being studied to determine the possible health risks and how to control them. Personnel are subject to primary and prophylactic medical examinations. When deploying personnel in an unfamiliar area, they undergo a medical risk assessment to determine what medical problems to expect and what action to take - vaccines, chemoprophylaxis, personal and collective protective measures, health promotion measures. Medical professionals must be trained to identify early staff health threats.

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B. PROFESSIONAL DISEASES

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1 OCCUPATIONAL DISEASES. INTRODUCTION

1.1. OCCUPATIONAL DISEASES – BASIC CHARACTERISTICS

Occupational disease is a disease that has occurred exclusively or primarily under the influence of the harmful factors of the working environment or the work process on the organism and is included in the list of occupational diseases.

Occupational illness also involves its complication and its later consequences.

Professional pathology studies etiology, pathogenesis, clinical picture, treatment and disease prevention, and work capacity expertise.

Occupational diseases and poisons are divided into the following groups:

Specific occupational diseases. Diseases in this group have only the harmful factors of the working environment and the labour process (silicosis, vibration disease, production poisonings, etc.) for etiological factor.

Conditional occupational diseases. The professional nature of the disease is accepted under **certain conditions and requirements**. The incidence of these diseases is significantly higher in people with certain professions (professional infectious and parasitic diseases, professional myopia, diseases of the nervous and musculoskeletal system, allergies etc.). This category of diseases also includes those of a general nature.

Paraprofessional diseases. These are work-related illnesses in which production activities are only a triggering or additional moment favorable to the development of the disease, not a main cause. Their etiology is complex, also depends on other factors outside the working environment (arterial hypertension, ulcer disease, neurosis, etc.).

Basic criteria for assessing the occupational nature of the disease.

1. Etiological clarification of the disease including:

- **General and occupational history. Professional history** clarifies the type, amount of action of risk factors, the duration of the work and all the details related to the nature of the work undertaken implemented production processes by the person and those close to his workplace. In addition, employers provide a **production record** with information including the qualification, the work route and the working conditions of the person concerned **(Decree No 133 of the Council of Ministers of 17.07.2000, Art.15, para.1)**.

2. Clinical assessment including a systematic clinical trial using routine clinical methods and recognizing early symptoms of occupational impairment of different organs and systems.

3. Paraclinic studies.

3.1. Clinical laboratory tests

- Toxico-chemical studies. They carry out the determination of toxic substances and/or their metabolites in biological media. These are specific laboratory tests (biomarkers) that are used to estimate the so-called "internal exposure" to a toxic substance and have a certain value to build the diagnosis and monitoring and evaluation of the applied therapy.

- Hematological studies. In most cases, haematological parameters are not specific. Some of them contribute substantially to the diagnosis and to conduct adequate therapeutic management (methemoglobin, carboxyhemoglobin, basophil-stippling erythrocytes, reticulocytes, siderocytes, cells Heinz, etc.).

- Clinical-chemical studies. A wide range of clinical and chemical laboratory parameters are used. They are routinely used in practice and also have the most non-specific nature in terms of professional pathology.

3.2. Immuno-allergenic studies. Investigation of IgE, IgG, variants of immunosuppressive tests, etc. (skin-allergy tests including epicutaneous, dermal and intradermal tests, etc.).

3.3. Functional and instrumental tests

- Breathing system (air obstruction of the nasal cavities, the functions of the mucous membranes of the upper respiratory tract, an x-ray of the lungs - general x-raygraphy, fluorography, tomography, computed tomography, radiological and ultra-

sound, pathomorphological invasive examination, bronchoscopy combined with biopsy, mediastinoscopy and non-invasive examination of sputa, nasal secretion, etc., microbiological study, functional examination of external respiration - vital capacity, respiratory volume, inspiratory and expiratory reserve volume, residual volume, forced expiratory volume, etc.; blood gas and acid-base status, etc.).

- Cardiovascular studies (ECG, overburden test, etc.).

4. Assessment of length of service. When assessing the nature and extent of the disease, the length of the specialized work experience is essential. With increasing work experience, the possibility of developing chronic occupational diseases is increasing. This does not apply to acute poisoning, allergic diseases, etc.

5. Sex and age. Adult and young individuals are more likely to be affected by toxic factors. In women, due to their anatomophobiological features, injuries to toxic substances, over-exertion of the musculoskeletal system, etc. are more pronounced.

6. Epidemiological test. There is an increased incidence of disease of persons with a similar clinical picture, from the same workplace.

7. Elimination test. After termination of the contact with the professional harmful factors, the total or partial disruption of the occupational disabilities (mostly in allergies) is established.

8. Exposure test. Repeated contact with the harmful factors of the working environment is observed in the worsening of the disease.

9. Assessment of general diseases (pre-existing or concomitant diseases); **family heredidary burdened, individual peculiarities, harmful habits, etc.**

List of Occupational Diseases

A prerequisite for recognition of the occupational nature of the disease is to be present in the list of occupational diseases. With the promulgation of an Ordinance on the procedure for notification, registration, confirmation, appeal and reporting of occupational diseases (State Gazette No 33 of 04.04.2001), **an illness not included in the list of occupational diseases may also be recognized when It is established that it was caused mainly and directly by the usual work activity of the insured person and caused a permanent incapacity or death of the insured (Art. 2, Par. 2)**.

The list of occupational diseases (Decree 175 of the Council of Ministers/2008) is built on an etiological principle **(Table 1)**. It contains 5 groups of diseases divided into 20 subgroups.

Occupational disease expertise.

The expertise on working capacity aims at preserving the patient's working capacity and preventing him from disabling. It determines the state of professional working capacity by examining the basic functions of the individual's body necessary for the specific work. The tasks of the occupational disease expertise shall include:

- diagnosis of the disease and expert evaluation of the state of professional working capacity;
- identifying the reasons for the disability;
- giving concrete job recommendations;
- systematic monitoring and control of the state of the working capacity;
- determining the percentage of permanently reduced or lost working capacity in percentage relative to the healthy person;
- carrying out measures for the prevention and elimination of disability.

Incapacity for work is temporary and lasting. Each one of them has its clinico-physiological characteristics, relevant regulation with certain normative acts and social security.

Occupational disease is not always associated with impaired working capacity. The issue of working capacity is always decided on a case-by-case basis and depends on the degree of expressiveness, the nature and the course of the disability, as well as the contraindications for the exercise of the previous profession.

When an occupational disease is detected, the expert judgment may be:

- temporary release from work in the presence of acute or exacerbated chronic disease with outpatient or stationary treatment;
- temporary transfer of another job without contact with the harmful factors that caused the disease;
- providing the person with reduced working conditions for the same job;
- translation for longer term of working without contact with harmful factors (with a disability group at disqualification);
- full incapacity for a job (permanent retirement and disqualification group definition).

Prophylaxis of occupational diseases.

For the prevention of occupational diseases, it is essential to build a wide range of measures:

- technological, technical and sanitary-technical, architectural, urbanisation, conditions of life, etc.
- labour-hygienic and healing-prophylactic;
- labour legislation, which regulates special legislation on safety in harmful industries.

Occupational-hygienic and healing prophylactic measures. Occupational-hygienic measures include occupational safety and hygiene - advance

Tab. 1. List of Occupational Diseases (State Gazette, 66/2008 - extract)

1. Occupational diseases caused by chemical agents

1.1. Occupational diseases caused by inorganic chemical agents

Code	Occupational disease caused by:	Clinical manifestations (Diagnoses, syndromes)	MKB-10	Grace period	Conditions for recognition	Examples of typical risky activities
1	2	3	4	5	6	7
1101	Lead and its compounds	Toxic cerebrasthenia Sideroahrestic anemia Haemolytic anemia Abdominal colic Nephropathy (tubulopathy) Motor type polyneuropathy (mostly upper limbs) Toxic encephalopathy	F06.8 D64.2 D59.4 R10.0 N14.3 G62.2 G92	* 1 year 1 year 1 month 3 years 3 years 3 years	1. Proven impact - at least 1 month, peripheral polyneuropathy and encephalopathy - at least 1 year. 2. Paraclinical supported sindromokompleks (elevated values of Pb in biological fluids, abnormal porphyrin metabolism).	Production and processing of lead acid batteries, pipes, cables, balls, paint plastics, stabilizers, leaded glass, metallurgy upon receipt of lead and lead alloys, soldering, glazing and decorating ceramic products with lead compounds; tin coatings production and use of lead oxides, salts and others.
1102	Cadmium and its compounds	Hypo-atrophic nasopharyngitis anosmia Chronic obstructive pulmonary disease Diffuse pulmonary fibrosis Nephropathy (tubulopathy) Osteomalacia (spontaneous fractures) Lung cancer	J31.1 R43.0 J44.8 J84.1 N14.3 M83.8 C34.-	6 months 6 months 5 years 10 years 3 years 12 years 40 years	1. Proven impact - at least 1 year, neoplasms - a minimum of 5 years, pulmopathy - a minimum of 10 years, for bone lesions - at least 20 years. 2. Paraclinical supported sindromokompleks (elevated Cd in biological fluids).	All activities of extraction and processing of polymetallic ores containing cadmium, production and processing of cadmium-nickel accumulators and batteries, cadmium plating surface in electroplating, producing fast melt alloys production cadmium pigments for paints, lacquers, glass, plastics, paper and pyrotechnics, cadmium lamps, welding of cadmium-plated parts and others.
1103	Manganese and its compounds	Toxic cerebrasthenia Parkinson's syndrome Vegetosenzorial polyneuropathy Psychoses Pulmonary fibrosis	F06.8 G21.2 G90.8 F31.- J84.1	* 10 years 6 months 1 year 10 years	1. Proven impact - at least 18 months for pulmonary fibrosis - a minimum of 10 years. 2. Paraclinical supported complex syndrome (EMG - stretch reflex).	Extraction and processing of mangan containing ores , upon receipt of cast iron and stainless steel, in production and processing of manganese alloy (duralumin, manganese bronze, etc.), production of ferro-manganese, electric welding with high quality manganese electrodes and others.
1104	Mercury and its compounds	Gingivitis (edentulism) Stomatitis, mucosal ulcerations in the mouth Pharyngitis Cerebrastheniya Encephalopathy (mercury erethism), mercury intention tremor, psychoorganic syndrome) Sensorimotor type polyneuropathy Toxic nephropathy (nephrotic syndrome and glomerulonephritis) Allergic contact dermatitis	K05.1 K12.1 J31.2 F06.8 G92 G62.2 N03.- L23.0	1 month 1 month 1 month * 3 years 3 years 1 year 1 month	1. Proven impact - at least six months. 2. Paraclinical supported complex syndrome (elevated Hg in biological fluids, positive CT).	All activities of extraction, processing and use of mercury and its compounds and products containing it. Production, use and repair of thermometers, barometers, manometers, pneumatic pumps, mercury lamps and other mercury instruments. Leather processing, production and use of pigments and paints, extraction of mercury from industrial waste and others.
1.2.	Occupational diseases caused by organic chemical agents					
1225	Organic mercury compounds	Conjunctivitis Blepharitis Gingivitis Stomatitis Pharyngitis Anemia Toxic cerebrasthenia Toxic encephalopathy (psycho-organic syndrome, psychosis) Allergic contact dermatitis	H10.4 H01.0 K05.1 K12.1 J31.2 D64.2 F06.8 G92 L23.-	* * * * * 1 year * 3 years 1 month	1. Proven impact - at least 1 year. 2. Paraclinical supported complex syndrome (mercury in biological fluids).	Production and use of mercury-organic compounds, such as fungicides and bactericidal agents for preserving plastics, textiles and wood and others.

* Do not allow discussion of professional character after termination harmful effects of professional factor.

2. Occupational diseases caused by physical factors

2.1. Occupational diseases caused by noise

2101	*Noise (constant , variable, hydrodynamic)*	Neuritis of both auditory nerve (hearing loss above 35 dB) Practical deafness (sensory neural hearing loss 65-90 db); Deafness (hearing loss above 91 dB)	H93.3 H83.3	1 year 1 year	1. Proven impact - at least 5 years, in the case of particularly intense noise (over 100dB/A) - at least six months. 2. Tone threshold audiometry in better hearing ear calculated at a frequency of 500, 1000, 2000 and 4000 Hz; the average loss is the mean, calculated from the loss observed at 500, 1000, 2000 and 4000 Hz, as factor of calculation are respectively 2, 4, 3 and 1.	All activities related to exposure to excessive sound loads, for example, work in workshops for boilers and cookers; hammering, bending and stamping metal; work with looms and shuttles; stamping on fabric; work with pneumatic hammer drill; in engine rooms of ships; with rotary machines in the graphics industry; assembly line filling (bottles, barrels, etc.) in the food industry; use or disposal of military munitions or explosives; construction work done with noisy equipment (bulldozers, excavators, shovel and etc.). tuning, testing and use of engines, jet propulsion and reactors (minimum exposure period - 30 days); discotheques and others.

2.2. Occupational diseases caused by vibration

2201	Local vibration *(vibration arm)*	Distal arterial and microcirculatory disturbances in the upper extremities	I79.8	1 year	1. Proven impact - at least five years, taking into account the existence of a synergistic factors. 2. Paraclinical supported complex syndrome (capillaroscopy, distal Doppler diagnostic, thermistor thermometers cold test, palesteziometry, EMG, X-ray, dynamometry, etc.).	Working with mechanical tools and machines generating vibrations (pneumatic hammers, saws, hammer-drills, grinders, grinding machines, metalworking and woodworking machinery, etc.).
		Raynaud's syndrome	I73.0	3 years		
		Vegetative disorders of the upper limbs	G90.8	1 year		
		Polyneuropathy of upper limbs	G62.8	1 year		
		Compression neuropathy m.medianus wrist (carpal tunnel syndrome)	G56.0	2 years		
		Compression neuropathy n.ulnaris the level of the elbow (cubital syndrome channel) or the level of the wrist (syndrome Guillon)	G56.2	2 years		
		Other compression neuropathy of the upper limbs	G56.8	2 years		
		cubital osteoarthritis	M19.2	3 years		
		Omartrozis	M19.2	3 years		
		Osteonecrosis of the os lunatum	M93.1	3 years		
		Osteonecrosis of the os scaphoideum	M87.8	1 year		
		Shoulder injury	M75.-	1 year		
		Medial epicondylitis	M77.0	1 year		
		Lateral epicondylitis	M77.1	1 year		
		Damage to the soft tissues of the upper limbs	M70.-	1 year		
2202	General vibration *(affecting the entire human body)*	Damage to the intervertebral disc (lumbar and lumbar-sacral segment) with radiculopathy	M51.1	1 year	1. Proven impact - at least six years, taking into account the existence of a synergistic factors. 2. Paraclinical supported complex syndrome (distal Doppler diagnostic, palesteziometry, EMG, EEG, audiometry, otoneurological research, image diagnostic, etc.).	Coal and ores mining, construction, transport, agriculture and others. (working with heavy machinery and vehicles - bulldozers, tractors, excavators, locomotives, harvesters, dump trucks, loaders and digging machines, etc.).
		Cubital arthrosis	M19.2	1 year		
		Humeroscapulary periarthritis	M75.0	1 year		
		Medial (ulnar) epicondylitis	M77.0	6 months		
		Lateral (radial) epicondylitis	M77.1	6 months		
		Stenotic tendinitis of the flexor of the fingers ("click finger")	M65.3	3 months		
		Stenotic tenosynovitis of the thumb (Disease de Quervain)	M65.4	3 months		
		Crackling chronic synovitis of hand and wrist (tendovaginitis)	M70.0	8 months		
		Other soft tissue injuries (myotendinosis the forearms)	M70.8	6 months		
		Chronic back pain	M54.8	1 year		
		Lumbar or lumbar-sacral radiculopathy nodiscogenic	M54.1	1 year		
		Disorders of the autonomic nervous system	G90.8	1 year		
		Polyneuropathy of the upper or lower limbs	G62.8	1 year		
		Auditory- vestibular disorders	H81.3 H81.4 H83.3	1 year		
		Hypothalamic syndrome	G90.8	1 year		

2.3. Occupational diseases caused by atmospheric pressure

2301	Increased atmospheric pressure	Myalgia Neuralgia Osteonecrosis (most often in the shoulder, hip or knee) Platispondiliya (bone infarcts of the lower thoracic vertebrae) Vestibulopathy Subacute or chronic otitis media to the point of irreversible hipacuzis Encephalomyeloaeropathy	M79.1 M79.2 M87.8 M48.5 H81.8 H65.4 G96.8	6 months 6 months 20 yeras 6 months 6 months 6 months 6 months	1. Proven impact - at least 1 year osteonecrosis - at least 3 years. 2. Paraclinical supported complex syndrome(radiographs, audiometry, otoneurological studies, EEG, etc.).	All activities carried out in an environment with increased atmospheric pressure (working with suits and underwater pneumatic chambers, work of divers, etc.).
2302	Reduced atmospheric pressure	Cerebroasthenic syndrome Cognitive impairment Vestibulopathy Tachypnea, dyspnea Tachycardy Cardiac arrhythmics Secondary polycythemia	F45.3 F06.7 H81.8 R06.0 R00.0 R00.8 D75.1	6 months 6 months 6 months 6 months 6 months 6 months 6 months	1. Proven impact - at least 1 year. 2. Paraclinical supported complex syndrome (complete blood count, ECG, EEG, psychological testing, otoneurological research etc.).	All activities carried out in an environment with reduced atmospheric pressure (aviopersonal, working in high sites - weather stations, cabins, rescue services, etc.).

2.6. Occupational diseases caused by ionizing radiation

2601	Ionizing radiation *(gamma rays, X rays, ionizing particles)*	Radiation cataract Anemia (hypo- or aplastic) leukopenia Thrombocytopenia Hemorrhagic diathesis Chronic radiation dermatitis Chronic mucous lesions Radiation bone necrosis Immunodeficiencies Skin cancer Osteosarcoma Lung cancer (inhalation) Malignant neoplasms of the target organ Leukosis	H26.8 D61.2 D70.- D69.5 D69.8 L58.1 L59.- M87.3 (D80-D89) C44.- (C40-C41) C34.- (C00-C97) (C91-C95)	5 years 3 years 1 years 1 years 1 years 10 years 5 years 10 years 2 years 20 years 20 years 20 years 20 years 10 years	1. Proven occupational exposure to manifestation depending on the magnitude of the effective dose to the target organ. 2. Paraclinical supported complex syndrome (blood count, image diagnostic, biopsy, etc.).	All activities related to sources of ionizing radiation, including radioactive substances of natural or synthetic origin (mining and processing of radioactive ores, manufacturing and handling of radioactive material (isotopes), production and application of radio pharmaceutical preparations, nuclear medicine, experimental nuclear reactors, maintenance and operation of equipment for radiotherapy and diagnostic gamma detection, nuclear energy, etc.).

3. Occupational diseases caused by biological agents

3.1. Occupational diseases caused by bacteria

3101	Mycobacterium tuberculosis and Mycobacterium bovis	Tuberculosis (respiratory organs, nervous system and other organs)	(A15-A19)	6 months	1. Proven professional contact. 2. Clinical examinations (Mantoux test), X-ray, sputum for BT).	Activities associated with contact with animals that carry the bacillus tuberculosis; manipulation of organic products and waste of animal origin (animal consulting rooms, slaughterhouses, processing plants and preserving of meat and meat products, incinerators, etc.). Activities related to the treatment and care of tuberculosis patients (bacteriological laboratories, hospitals, sanatoriums, etc.).
3102	Brucella	Brucellosis (Bang's disease)	A32.-	2 months (an acute form) 1 year (chronic form)	1. Proven professional contact. 2. Paraclinical surveys (KAP with brucellin, isolation of brucella from biological fluids).	Activities in slaughterhouses, processing plants and preserving of meat and meat products; dairies. Activities for raising goats, cattle, sheep and pigs and contact with their excrement and abortions products.
3103	Bacillus anthracis	Anthrax (cutaneous, pulmonary form, gastrointestinal form)	A22.-	1 month	1. Proven professional contact. 2. Clinical examinations (bacillus isolation from biological fluids).	All activities where there is contact with infected animals. Activities in tanning.
3104	Clostridium tetani	Tetanus	A35	1 month	Proven risk.	Activities in construction, agriculture, forestry and others.

3105	Salmonella	Salmonellosis	A02.-	1 month	1. Proven professional contact. 2. Clinical examinations (isolation of the causative organism from faeces).	Activities under cultivation and trade in livestock and poultry, production and marketing of feed protein flours. Activities related to the treatment and care of infected persons (bacteriological laboratories, hospitals, etc.). Repair and maintenance of sanitation and others.
3106	Pseudomonas mallei	Glanders	A24	1 month (an acute form) 1 year (chronic form)	1. Proven professional contact. 2. Clinical examinations (microbiology and serology).	Activities for animal production, veterinary practice, slaughterhouses and others.
3107	Francisella tularensis	Tularemia	A21.-	1 month	1. Proven professional contact. 2. Paraclinical tests (serological samples with tularin KAP).	Activities in livestock, hunting and forestry, tanning and more. Work in fumatoriums, pet shops and more.
3108	Rickettsia (Coxiella burnetti)	Q fever	A78	1 month (an acute form) 1 year (chronic form)	1. Proven professional contact. 2. Clinical examinations (serological).	Activities in agriculture, laboratories (vaccine production) veterinary practice and others.

3.2. Occupational diseases caused by viruses

3201	Hepatitis A virus	Acute viral hepatitis A	B15.-	2 months	1. Proven professional contact. 2. clinical examinations (ASAT, ALAT, GGT, AF, direct and indirect bilirubin, transaminase activity).	Repair and maintenance of sanitation. Work in large teams (barracks, schools, kindergartens) in the epidemiological situation. Jobs in health care and others.
3202	Hepatitis B virus	Acute viral hepatitis B Chronic viral hepatitis B Late effects of viral hepatitis B	B16.- B18.- B94.2	6 months 2 years 15 years	1. Proven professional contact. 2. clinical examinations ((ASAT, ALAT, GGT, AF, direct and indirect bilirubin, transaminase activity early marker - Anti-HBc-lgM).	All activities of collecting, handling and use of human blood and its derivatives. Work on supportive, washing and sterilization of materials and equipment that involve contact with the virus.
3203	Hepatitis C virus	Acute viral hepatitis C Chronic viral hepatitis C Late effects of viral hepatitis C (hepatocellular carcinoma)	B17.1 B18.2 B94.2	6 months 20 years 30 years	1. Proven professional contact. 2. Clinical examinations (anti HCV, antibody).	
3204	Rabies virus	Rabies	A82.-	6 years	1. Proven professional contact.	Activities in agriculture, hunting and forestry, veterinary practice. Work in laboratories, pet shops, zoos, vivariums and others.

and ongoing familiarization of workers with manufacturing processes and safety measures in their implementation; implementing effective control over workers qualification and protective production habits; organization of working and rest regime; ongoing control of concentrations of harmful substances in the air of the working environment; use of effective and functionally suitable personal protective equipment (suitably special work clothes, gloves, shoes, facial and respiratory protection); maintenance of the necessary personal hygiene and such in the working premises, etc.

Medical prophylaxis. Medical prophylaxis includes preliminary professional selection of admissions, periodic prophylactic medical examinations, dispensarisation and communication, registration and reporting of occupational diseases, preventive professional nutrition, physical prophylaxis, etc.

Prophylactic medical examinations (preliminary and periodic) are regulated by Ordinance № 3 of 28.01.1987, by the Ministry of Health and by Amendments and suppl. from 1991 and 1994.

The pre-selection is carried out on the basis of compulsory medical examinations on taking up em-

ployment. Do not work in contact with harmful factors of the working environment, persons with disabilities of different organs and systems regulated in Ordinance № 3. Special attention is paid to women (pregnant women, nursing mothers and in fertile age) as well as to the teenagers .

The rhythmic and proper conduct of periodic prophylactic medical examinations provides early diagnosis of the occupational disabilities of the body and helps to reveal the risk factors that contributed to the development of these disabilities. The time limits for conducting periodic prophylactic medical examinations are specified in Ordinance No. 3 and vary according to the type of professions and procedures. The quality of these reviews requires strict adherence to the Ordinance. The formation of the medical teams conducting the examinations should be done precisely with the participation of different specialists and the use of specialized paraclinical studies. This is a sure prerequisite for an ex act and early diagnosis of the injuries of those working in the harmful health conditions and resp. for their effective prophylaxis.

1.2. OCCUPATIONAL DISEASE AND OCCUPATIONAL ACCIDENTS. SOCIAL RISKS PROVIDED.

Occupational illness and occupational accidents are provided occupational risks, related to labour in an working relationship.

Legal regulation.

International regulation:

- Universal Declaration of Human Rights.
- Convention No. 12 on Compensation for Accident at Work - 1921 SG no. 44/1997
- Convention No. 17 on Compensation for Accident at Work - 1925 SG no. 253/1929
- Convention 18 on Occupational Diseases - 1925 SG no. 253/1929
- Convention No. 19 on Equal Treatment (Accidents at Work) - 1925 SG no. 253/1929
- Convention No 42 on Benefits for Occupational Diseases (revised) 1934
- Convention No 37 on Invalidity Insurance - 1933 SG no. 45/1997
- Regulation 1408/71 / EEC on the application of social security schemes to self-employed persons, independently and to members of their families, moving within the Community. European Union law on social protection. S. 1998
- Regulation No 574/72 / EEC laying down the procedure for implementing Regulation No 1408/71 / EEC. Col. Legal provisions of the European Union in the field of social protection. S. 1998.

Legislation in the country:

- Constitution of the Republic of Bulgaria. SG no. 51/1991
- Code of Compulsory Public Insurance (CCPI).
- Labour Code (LC)
- Ordinance on the Procedure for Reporting, Registration, Confirmation, Appealing and Reporting of Occupational Diseases. SG no. 33/2001, effective at 5.VII.2000
- Ordinance on the establishment, investigation, registration and reporting of accidents at work. SG no. 6/2000, amended and add No 61/2000

Insurance relationship.

The insurance relationship is governed by the legal relationship that arises in connection with the material insurance of the citizens in cases of incapacity for work and other reasons regulated by the law, which prevent them from carrying out labour activity, as a result of which they are deprived of income.

General Characteristics of the Insurance Relationship:

- Is property nature
- continuous legal relationship
- complex legal relationship.

The nature of the insurance relationship:

- Compulsory social security is public.
- Voluntary social security is private.

Types of insurance legal relationships according to the subject of insurance:

- In case of incapacity for general disease, occupational disease, labour accident, maternity, old age and death.
- In the case of health insurance
- Unemployment

Occupational disease is a disease that has occurred exclusively or mainly under the influence of the harmful factors of the working environment or the work process on the organism and is included in the List of Occupational Diseases issued by the Council of Ministers upon a proposal by the Minister of Healthcare.

In the legal definition, the legislator has included the two alternatives - exclusively or predominantly caused by the harmful factors of the working environment or the working process, guided by the **medical classification of occupational diseases (Table 1)**.

Similarly, European legislation in the CCPI is given a legal opportunity to be recognized as an occupational disease and illness not included in the List of Occupational Diseases when it is established that

it was caused primarily and directly by the insured person's usual work and that it has caused permanent disability or death of the insured.

Occupational illness also refers to its complications, both and its late consequences.

The practitioners and dentists in case of suspicion of occupational disease also send a notice to the territorial unit of the National Social Security Institute (NSSI), which carries out research, prepares documents and submits them to the medical expert bodies.

The Ordinance on the notification, registration, confirmation, appeal and reporting of occupational diseases (SG 33/2001) regulates the confirmation or rejection of the occupational nature of the disease by Territorial Expert Medical Committees (TEMC), determined by an Order of the Minister of Health and National Expert Medical Commitee (NEMC), including a specialist in occupational diseases and a specialist in occupational medicine. The meetings of the aforementioned committees are chaired by a specialist in occupational diseases or occupational medicine. The Commission shall also decide on the degree of reduced capacity in accordance with the order, principles and criteria regulated by the Ordinance on the Expertise on the Capacity for Work (SG No. 61/2000).

TEMC makes a decision to confirm the occupational disease based on the clinical examination of the person, the data from the medical record and the additional evidence collected within 40 days of receiving the medical file. An expert judgment on occupational disease is issued for a period of 3 years and a registration card for a recognized occupational disease is completed. The insured person, the insurer, the territorial unit of the National Social Security Institute before NEMC have a legal interest in appealing the expert decisions of TEMC within 14 days from the date of receiving the decision. Expert decisions may be appealed to the court under the procedure of the Administrative Proceedings Act.

An accident at work *is any sudden damage to health occurring during and in connection with or in occasion with the work performed, as well as any work done in the interest of the enterprise when it caused incapacity or death.*

An accident at work is also recognized as an accident occurring when traveling and returning from the workplace to the main place of residence or another additional residence with permanent character; a place where the insured person usually feeds on a business day; the place to receive remuneration.

There is no occupational accident when the injured has deliberately harmed his health.

In the investigation of the accident at work, the following are established:

- the reasons and the circumstances that led to the accident at work.
- type of disability
- other information qualifying the accident as labour

The insurer declares every accident at work to the Regional Office of the NSSI within 3 working days. In 6 days, the injured person himself can do so if the insurer does not declare the accident at work. The procedure for establishing, investigating, registering and reporting accidents at work is regulated in an Ordinance issued by the Council of Ministers.

In an accident investigation, the injured is entitled to attend or indicate to be present:

- an employee of the same profession;
- a family member, or an ascending or descending relative;
- a representative of a trade union in which he/she is a member;
- a representative of the employees of the Working Conditions Committee;
- the heirs of the deceased from the accident at work.

The statement of the investigation shall be valid until proved otherwise and shall be sent to the territorial unit of NSSI and shall be kept in the State Archives. For each occupational accident and occupational disease, the territorial unit of NSSI opens a file.

The CCPI regulates the *insurance funds*, which are divided into:

- "Pension"fund;
- "Accident at Work and Occupational Disease" fund;
- "General Sickness and Maternity" fund.

The specific feature of the Accident and Occupational Diseases Fund is that *employers pay contributions only to their employees* and their size is determined annually on the basis of the main activity of the enterprise, the frequency and severity of accidents at work and occupational diseases, the number and severity of violations in connection with the implementation of investment programs and the organization of activities to ensure healthy and safe working conditions, and depending on the degree of professional risk. The *other earnings* in the fund are raised from:

- State Budget Amounts;
- Revenue provided under other laws for occupational accidents and occupational diseases;
- Reimbursement of the insurance costs incurred and the cases provided for in the normative acts;
- Fines and penalty interest;

- Fees determined by tariffs of the Council of Ministers;
- Interest and dividends;
- Donations and wills;
- Other sources.

The means from this fund are spent for:

- Payment of cash benefits and pensions;
- Updating, indexing and offsetting of cash benefits, pensions and financial aids;
- Measures to prevent accidents at work and occupational diseases;
- Supporting and technical means related to disability;
- Other costs related to the insurance for accidents at work and occupational disease.

The above-mentioned fund does not provide for the treatment, re-training, re-qualification and compensation of the injured person for material and non-material damages, which is a substantial difference to the European legislation of the identical funds.

Legal consequences under the Labour Code.

For the workers injured by the occupational disease, the legislator has established guard and privileged regimes under the Labour Code with a view to their proper rehabilitation and the fullest possible use of their residual capacity. According to the provision of Art. 317 of the Labour Code, the employer is obliged to move the employee to an appropriate job, as prescribed by the health authorities, within 7 days of receipt. In case of non-fulfillment of the prescription of the health authorities by the employer, he shall pay to the employee a compensation in the amount of his gross remuneration from the day he receives the prescription for re-employment until it is fulfilled (Article 217 paragraph 1 of the Labour Code).

The fact that workplaces designated for employment by employed persons are exhausted is not a reason for not complying with the above provisions.

The employer may terminate the employment contract if the employee is unable to perform the assigned job due to sickness resulting in permanent disability or health contraindications, according to the expert opinion of TEMC. When the employer has another job appropriate to the health of the worker and he wishes to take it, the employment relationship is not interrupted. If the employer fails to comply with this statutory requirement (Article 325 p.9 of the Labour Code), his injunction to terminate the employment relationship will be unlawful and may be challenged in court within 6 months of the date of termination of the employment relationship. The Labour Code also provides for a special order for the dismissal of employees who are labour-readjusted or suffer from diseases referred to in Ordinance No. 5, in which list under item 5 an occupational disease is mentioned. For these persons, the employer is obliged to request the opinion of TEMC and the Labour Inspectorate before the dismissal.

Legal consequences of social security.

Qualification of the disease as a professional in due course gives rise to *the injured and social and legal consequences*:

- Right to compensation for temporary incapacity for work
- Right to invalidity pension due to an accident at work and occupational disease - Art. 24, point 1. CCPI
- Right to benefits - Art. 24, point 2 CCPI
- Updating, indexing and compensation of cash benefits, pensions and benefits - Art. 24, point 2. CCPI
- Assistance related to disability - Art. 24, point 4. CCPI

Civil law consequences. An occupationally qualified illness must be caused by guilty actions or omissions that have led to the harmful result - occupational disease. The legal basis for compensation for the person suffering from an occupational disease is settled in the institution at nopermitted damage (Article 45 et seq., from the LCL). Liability for payment of damages - property and non-material to the injured person - is the legal obligation not to harm others - "neminem lacdere". Everyone is obliged to repair the damage he has guiltily caused to someone else. Under the Obligations and Contracts Act, the prohibition is general and includes any other culpable harm to others.

Compensation for civil action by the employer.

The property damages in the case of a person suffering from an occupational disease is expressed as the difference between the actual remuneration he receives as a healthy worker and the later indemnity for temporary incapacity for work, labour readjustment or disability pension. The property damage suffered undergo on account of the disability, which temporarily or permanently deprived him of the income he had as healthy.

Apart from a reduction in wages, the worker injured by an occupational disease can also suffer material damage by terminating the receipt of certain *other permanent payments that he has had as health* - overtime labour, subsistence, civil contracts, etc.

In the case of claiming property damage, the injured person may seek, in addition to all the permanent claims, as well as the costs of the legal claim and the claim for compensation for costs incurred by him for medicines, for intensive food, special diet

and balneotherapy and any other costs are related to his/her health condition.

Judicial authorities are guided by the requirements of Art. 51 al. 1 of the LCL, i.e. the indemnity should correspond to the actual amount of the material damage and loss of benefits in respect of the occupational disease.

Persons having a legal interest in seeking property damage in the case of death from delict - occupational disease. These are *the relatives of the deceased* who have suffered material damages from his death - children, spouse, parent, brothers, sisters and other relatives. Those listed must have been affiliated with the deceased or be entitled to maintenance by the deceased. Determining the circle of eligible persons is done by means of two criteria:

1. Persons who are entitled to maintenance by the deceased by law.

2. Persons, who are incapacitated and have been supported by the deceased who, albeit by law, have no right to maintenance from the deceased. This includes the child bring up but still unadopted.

Non-property damage. Non-property damage is, by its very nature, a violation of the non-material rights of the person - political rights, life, freedom, honor, family rights, etc., resulting in pain, moral annoyances, suffering and anxiety for the injured. They are defined as damages that are disproportionate to money and therefore the benefit to them and the circle of persons entitled to them are determined by the principle of fairness under Art. 52 of the LCL. The determination of the amount is related to court judgment of objectively specific circumstances, such as manner of disability, additional complications, medical prognosis of the course of the disease, pains and sufferings, etc. In case of death from an occupational disease, the age of the deceased, his public status and other qualities are of importance.

Non-property damage can wanted all persons affected by the occupational disease and which have subject own non-property weefare-damaged person itself, but also the survivors of the deceased, may be sought for non-property damage. There is no text in the LCL that explicitly indicates which persons are entitled to seek non-property damages. The court determines them in each individual case, examining the presence of the components of the non-property damage. These facts are judged irrespective of kinship, as it sometimes refuses to award non-pecuniary damage to relatives who have been in a bad relationship with the deceased. The justice, which is stated in Art. 52 of the LCL as a measure for determining the non-prperty damage is not a source of right. It is only a measure to determine non-property damage.

When a person suffering from an occupational disease suffers a deterioration in his health, a consequence of the illness, the new complications or the late effects of the disease, he is entitled to bring a new claim for compensation. However, in order to accept that there is a deterioration in the state of health of the injured person, it is not sufficient merely to ascertain such manifestations. New complications should have led to a new percentage rate of disability as any condition necessary to establish new material and moral damages under Art. 51, para. 1 and 3 of the LCL.

The period in which the claims for delict are extinguished is 5 years in the sense of Art. 111, B. of the LCL. Where the claim is established by a court decision which has entered into force, the limitation period shall be 5 years, including the interest awarded - Art. 117, para. 2 of the LCL. The taking of delict is due from the day of the opening of the perpetrator, according to Art. 114, para. IV of the LCL.

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N. Tsacheva

2 EPIDEMIOLOGY OF OCCUPATIONAL DISEASES

The epidemiology of occupational diseases in Bulgaria studied the distribution, structure and dynamics at national and regional level and contains information about the number and frequency in different economic sectors and occupations. It provides an important basis for monitoring and prioritizing preventive action to improve the hygiene and safety of workers. The Epidemiology of occupational diseases determines the leading factors in the working environment and the labour process in the occupational health and occupational risk labour medical expertise.

In recent years, improvement of occupational health legislation in the country and its harmonization with European legislation is created regulations that allow for modern epidemiology of occupational diseases, evidence-based occupational medicine:

- Social Security Code - published in 1999
- Health and Safety at Work Act - since 2007
- Ordinance on Occupational Diseases - since 2008
- Statistical System "Occupational Diseases" - since 2004
- Ordinance on medical expertise of efficiency - from 2005

These documents regulate and ensure the investigation and registration of occupational diseases, shaping and storage of documentation and analysis of frequency, structure and dynamics of occupational diseases for certain periods.

In the Official Bulletin 10/2001 of the Ministry of Health by the President of NSI published registration forms necessary for the study of occupational diseases:

- **quickly notice** *report card MH №PI - 58/2001;*
- **production characteristic**, *report card MH No. P601 / 2001;*
- **protocol for the Study of Occupational Disease.** *Issue No-20 of the NSSI (Article 5 of the TFTP of the NFIPRI);*
- **expert solution** - *report card MH № 611a/2001;*
- **registration card,** *report card MH №304 / 2001*. It contains information on comprehensive surveys, recording and reporting the health status of persons of recognized occupational disease in Bulgaria and is a major document, which studies the epidemiology of occupational diseases.

Schematically, the process of epidemiology is shown in **Fig. 1** and includes:

I. Communicating occupational diseases

II. Study of occupational diseases

III. Confirmation and registration of occupational diseases

IV. Appealing occupational diseases

V. Reporting of occupational diseases

The Ordinance on occupational diseases of 2008 continues the maintenance of the established register of occupationally ill patients. In 2001, the MH, NCHMEN, NSI and NSSI established a EUROPEAN harmonized statistical system for occupational diseases. On this basis, the FIRST REGISTRY AND INFORMATION SYSTEM on occupational morbidity is located in the Center of Hygiene. The system provides comprehensive and summarized information about the patients and their illnesses of MH, MLSP and NSSI. Requests for summary information may also be provided to the sponsor, GPs and other interested institutions and organizations of employers and employees, subject to the confidentiality of personal data.

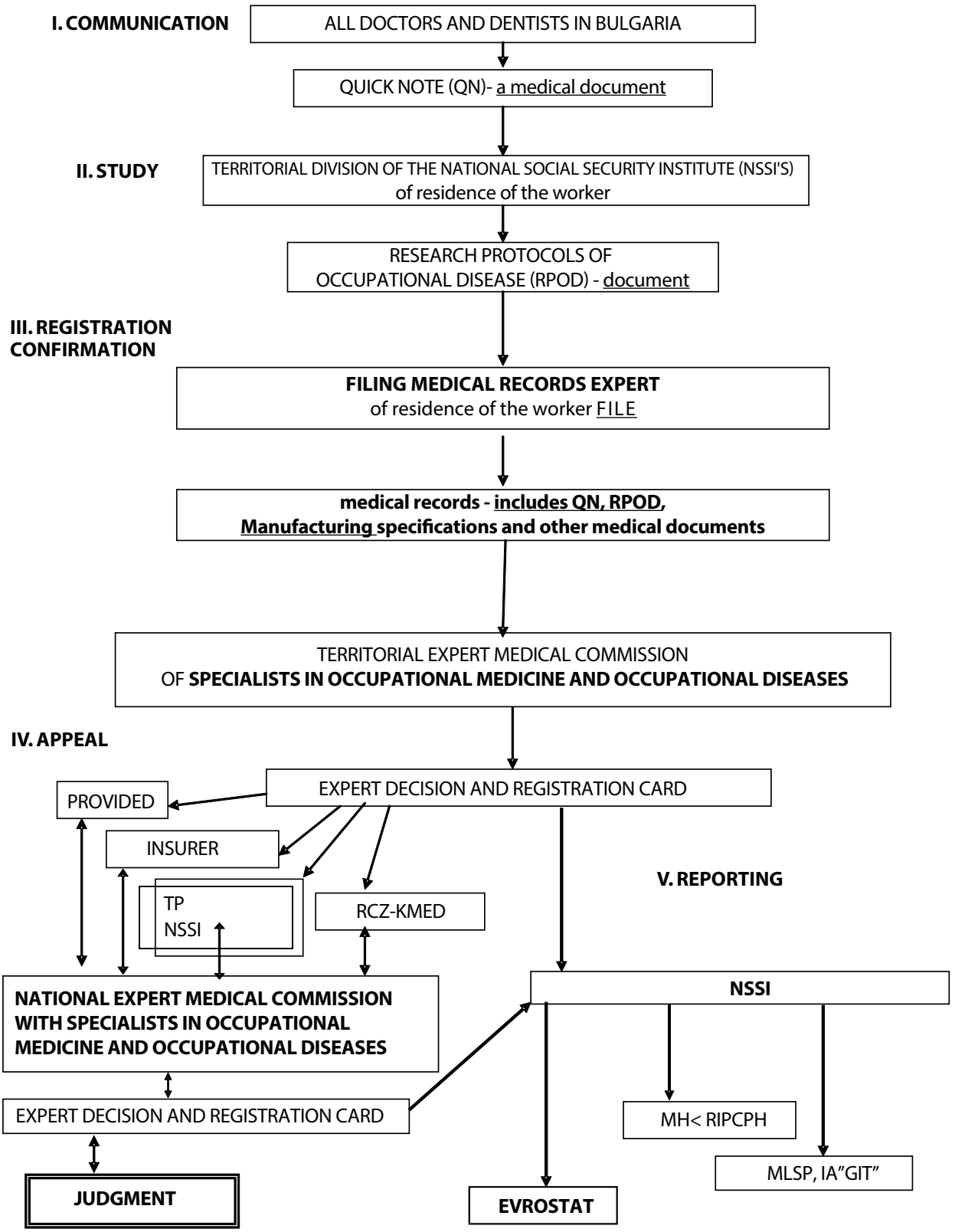
The dynamics of occupational diseases in Bulgaria in terms of the absolute number, the absolute growth and the rate of increase for the period 1975-2007 **(Table 1)** passes through several stages:

- 1975-1978 this stage is characterized by the reduction of occupational diseases, a one-year period of 2800 to 1800 cases.

- 1979-1990. In this greatest stage, occupational diseases are growing - over 3,000 cases per year, which is also the result of the increased revelation in the preventive examinations of the employees and by directed activities of the Clinics of Occupational Diseases in Sofia, Plovdiv, Varna, Pleven and Stara Zagora.

- 1991-1996. During this period the registration of the occupationally ill decreased due to various and complex reasons, with a growth rate of -2.2 to -39.1 (constant base 1975) and -5.0 to -3.5 on a chain basis.

Fig. 1. Stages of communication, investigation, confirmation, appeal and reporting of occupational diseases - institutions and health documentation



- 1997-2000. This stage is characterized by extremely low levels of occupational diseases - about 350 to 700 cases per year, as a result of a large share of "hidden" occupational morbidity, while the negative impact of different factors in the working environment and the labour process.

- 2000-2007. There is a positive trend towards an increase in the absolute number of occupational diseases and a chain growth rate of 35.6 in 2007. This result is also a consequence of the efforts made for better regulation in the field of occupational medical expertise .

Tab. 1. Dynamics of Occupational Diseases for1975 -2007

Year	Number of registered occupational diseases	Absolute growth		Rate of growth	
		Chain base	Permanent basis	Chain base	Permanent basis
1975	2791				
1976	2755	-36	-36	-1.3	-1.3
1977	2189	-566	-602	-20.5	-21.6
1978	1819	-370	-972	-16.9	-34.8
1979	3084	+1265	+293	+69.5	+10.5
1980	2859	-225	+68	-7.3	+2.4
1981	3150	+291	+359	+10.5	+12.9
1982	3789	-639	+998	+20.3	+35.8
1983	3705	-84	+914	-2.2	+32.7
1984	3542	-163	+751	-4.4	+26.9
1985	3402	-140	+610	-3.9	+21.9
1986	3556	+154	+765	+4.5	+27.4
1987	3381	-175	+590	-4.9	+21.1
1988	3304	-77	+513	-2.3	+18.4
1989	2945	-359	+154	-10.9	+5.5
1990	3157	+212	+366	+7.2	+13.1
1991	2729	-428	-62	-13.5	-2.2
1992	2382	-347	-409	-12.7	-14.6
1993	2091	-291	-700	-12.2	-25.1
1994	1806	-285	-985	-13.6	-35.3
1995	1790	-16	-1001	-0.9	-35.9
1996	1700	-90	-1091	-5.0	-39.1
1997	590	-1110	-2201	-65.3	-78.9
1998	700	+ 110	- 2091	+18,6	- 74,9
1999	357	- 343	-2917	-49.0	-87.2
2000	429	+72	-2363	-20.2	-84.6
2001	660	+231	-2131	+53.9	-76.4
2002	895	+235	-1896	+35.6	-67.9
2003	950	+ 55	-1841	+6.1	-66.0
2004	1168	+218	-1623	+22.9	-58.2
2005	1010	- 158	-1781	-13.5	-63.8
2006	805	-205	-1986	-20.3	-71.2
2007	1125	+320	-1666	39.8	-59.7

All registered cases of professional patients in Bulgaria as of 31.12.2007 are about 32100 **(Table 2)**.

The most common occupational illnesses have been shown, and have been reported as the most common occupational diseases that have not changed substantially as a nosological profile (structure) over the last 20 years.

The most widespread occupational risk factors for workers in recent years are:

- Manual labour, heavy physical labour, monotony, conveyor line work, and non-compliance with physiological working and rest periods during work, overtime, night work and more, related to the functioning and rational organization of work systems.
- Noise factor and vibration impact individually or with combined exposure. Most often they are accompanied by non-use of personal protective equipment, inefficient collective protective equipment, low health culture and health behaviour.
- Powder of different composition (crystalline silica, asbestos fibers, heavy metal aerosols, organic substances).
- Toxicochemical noxae as raw materials, intermediate and end products from the technological process, often combined with other occupational hazards - unfavorable microclimate, heavy physical labour, noise and vibrations.

In 2002, at control by labour inspection 27,081 enterprises with 1,091,920 employees, who did not meet the hygienic limit values were 20% (or 220,483 persons in very poor conditions). Their distribution by the individual factors is: in the case of an unfavorable microclimate - 41%; noise - 33%; physical

Tab. 2. Probable number of registered persons with cases of occupational diseases - total and groups of regions as of 31.12.2007

REGIONS (IN GROUPS)	TOTAL NUMBER OF OCCUPATIONAL DISEASES AND NOSOLOGICAL GROUPS *							TOTAL
	1	2	3	4	5	6	7	
Sofia, Pernik, Blagoevgrad, Kyustendil, Botevgrad	1589	1320	1069	761	511	309	120	5679
Plovdiv, Kardzhali, Smolyan, Madan	1638	1160	1193	1053	524	237	73	5878
Varna, Dobrich, Shumen, Targovishte	1131	1193	530	571	353	239	49	4066
Ruse, Silistra, Razgrad	1122	930	441	316	280	151	29	3269
Pleven, Lovech, Veliko Tarnovo	975	969	509	429	368	153	41	3444
Vratsa, Vidin, Montana	1170	1129	673	611	383	199	46	4211
St. Zagora, Burgas, Sliven, Yambol, Haskovo	1340	1099	1129	1191	512	210	72	5553
TOTAL	8965	7800	5544	4932	2931	1498	430	32100

* Nosological groups of occupational diseases:
1. of the Locomotor system (LS);
2. the Peripheral Nervous System (PNS);
3. Vibrational disease;
4. Neuritis of the auditory nerves;
5. Professional lung diseases;
6. Occupational poisoning.
7. Other

load - 32%; dust and toxic substances - 17%; lighting - 11%; vibrations - 6%; ionizing radiation - 1%. The highest number of occupational diseases registered in the Plovdiv region, incl. Kardzhali, Smolyan and Madan with developed mines from coal production and ore mining. The region of Stara Zagora follows with Burgas, Sliven, Yambol and Haskovo. Third is the region of Sofia, Pernik, Blagoevgrad, Kyustendil and Botevgrad.

Total morbidity with temporary disability to work (MTDW) is also related to occupational diseases. For individual economic activities and basic professions with high levels of occupational diseases, a high level of MTDW as well as for some groups of socially significant diseases is characteristic. In Sofia-city (the center for the most widespread economic activities, or the variety of professional injuries), make impression that the summary MTDW and the priority socially significant diseases, such as respiratory and cardiovascular diseases, have high and very high indicators **(Table 3)**. This applies both to health-insured working men and to a much higher degree for insured women. The frequency of cases, the frequency of the days and the average duration

Tab. 3. Morbidity with temporary disability to work of labour people in Sofia for 1996-2000 (total and by sex)

1. TOTAL MTDW

INDICATORS	FREQUENCY OF CASES			FREQUENCY OF DAYS			The average duration of 1 case		
WORKING	TOTAL	Male	Female	TOTAL	Male	Female	TOTAL	Male	Female
Sofia -city 1996	119,7	117,6	121,7	1320,9	1244,2	1388,6	11,0	10,0	11,4
1997	86,6	72,3	100,4	976,3	802,8	1142,4	11,3	11,1	11,4
1998	93,5	81,7	105,1	1200,1	1005,9	1390,2	12,8	12,3	13,2
1999	93,9	82,6	106,2	1210,8	1015,6	1388,2	12,9	12,3	13,1
2000	96,1	83,4	110,2	1210,8	1017,9	1401,2	12,6	12,2	12,7

2. MTDV RESPIRATORY SYSTEM

INDICATORS	FREQUENCY OF CASES			FREQUENCY OF DAYS			The average duration of 1 case		
WORKING	TOTAL	Male	Female	TOTAL	Male	Female	TOTAL	Male	Female
Sofia -city 1996	31,2	31,1	30,1	160,8	158,0	163,3	5,1	5,1	5,4
1997	23,9	19,5	28,1	119,1	95,6	141,6	5,0	4,9	5,0
1998	22,1	18,8	25,4	119,7	102,5	136,7	5,4	5,4	5,4
1999	22,8	18,9	25,9	119,9	102,9	136,1	5,2	5,4	5,2
2000	30,1	19,6	26,2	122,6	103,6	142,5	4,1	5,3	5,4

3. MTDV OF CARDIOVASCULAR SYSTEM INCLUDING HYPERTENSION

INDICATORS	FREQUENCY OF CASES			FREQUENCY OF DAYS			The average duration of 1 case		
WORKING	TOTAL	Male	Female	TOTAL	Male	Female	TOTAL	Male	Female
Sofia -city 1996	9,1	9,6	8,5	100,2	128,4	75,3	11,0	13,4	8,9
1997	5,9	5,8	5,9	71,8	85,6	58,6	12,2	14,8	9,9
1998	7,0	7,2	6,7	88,4	104,7	72,3	12,6	14,5	10,8
1999	7,1	7,3	6,7	85,4	104,9	73,2	12,0	14,4	10,9
2000	6,8	6,9	6,3	85,1	101,8	71,1	12,5	14,7	11,3

Tab. 4. Claims for recognition of occupational diseases of 100,000 insured persons

Country	1990	2000	Population thousands	Trend
Germany	192	211	80570	↑
Austria	151	103	7796	↓
Belgium	431	277	9979	↓
Denmark	549	545	5124	→
Finland	320	238	4998	↓
France	63	237	56634	↑
Ireland	4.4	7.5	3526	→
Italy	354	160	59104	↓
Luxembourg	113	82	365	↓
Sweden	1524	309	8587	↓
Switzerland	202	136	6876	↓

Tab. 5. New cases of recognized occupational diseases per 100 000 insured persons

	1990	2000	Population thousands	Trend
Germany	35	49	80570	↑
Austria	78	42	7796	↓
Belgium	186	112	9979	↓
Denmark	90	124	5124	↑
Spain	42	160	39434	↑
Finland	160	64	4998	↓
France	44	177	56634	↑
Ireland	2.3	3.3	3526	→
Italy	93	33	59104	↓
Luxembourg	8	14	365	→
Sweden	1242	138	8587	↓
Switzerland	162	109	6876	↓

of one case for the example five-year period, are increasing. The claims for recognition and recognized (registered) occupational diseases in the 1990s and 2000s in the 12 European countries are presented in **Tables 4 and 5**.

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3 OCCUPATIONAL POISONING *

3.1 METALS

3.1.1 ANTIMONY - J. HADJIEVA

Antimony is a silver-white metal insoluble in water. It is oxidized from concentrated nitric acid to antimony acid. Lighted in the air, antimony can burn to form antimony trioxide and antimony dioxide.

Usage. *Antimony and its compounds are widely used in metallurgy (such as antimony alloys), in the rubber, chemical, polygraphic and pharmaceutical industries, insect pests, rodents, etc.*

Biotransformations. *The antimony enters the body mainly via the respiratory tract and, more often than not, through the gastrointestinal tract. In the blood antimony (III) is bound to erythrocyte, and antimony (V) - predominantly in plasma. From the blood, the antimony quickly passes into tissues and spreads to the lungs, the liver, the kidneys, the skin and the bones. Metabolism is mediated by methylation processes. It is* ***eliminated*** *in urine - mainly antimony (V) and faeces - mainly antimony (III).*

Pathogenesis. *Antimony disrupts protein and carbohydrate metabolism. It binds to sulfhydryl groups and inhibits SH dependent enzymes from the oxidation of alpha-keto acids. Antimony is a hemolytic poison. It is believed to break the ionic composition of the cells, with a potassium deficiency observed. The antimony has a strong irritant effect on the skin and mucous membranes. Its carcinogenic effect is not excluded* ***(Table 1)****.*

Clinical picture.

Acute poisoning is characterized by marked irritant changes in the upper respiratory tract and eye lining. Inhalation of antimony aerosols may cause "metal fever", acute emphysema of the lungs and pulmonary edema. Gastrointestinal is characterized by nausea, vomiting, abdominal pain, diarrhea, dehydration and subsequent disturbance in the water-electrolyte balance. In severe cases of poisoning, phenomena of toxic hepatitis and toxic nephropathy, heart failure, collapse and death can be added.

Chronic antimony poisoning occurs with chronic irritative changes in the lining of the nose with subsequent necrosis and ulceration of the underlying tissue. Perforation of the cartilage of the nasal septum is observed. Ocular abnormalities, dry throat and chronic bronchitis have been reported. It is possible to develop toxic pneumoconiosis from antimony or mixed powders (silico-antimonyconiosis).

Cardiovascular disorders are most often characterized by conduction disorders, changes in repolarisation, myocardial diseases, and the like. Common skin lesions are dermatitis. Changes are placed on the limbs, thorax and genitals. Allergic manifestations are also possible - eczema.

Ovarian-menstrual disorders and spontaneous abortions are not uncommon. A carcinogenic effect of the antimony on the lungs and bladder has also been reported.

Clinical-laboratory tests. As an exposition test, the determination of antimony in urine (normal values of 0.2-1.3 μg/l) is used.

Hematological parameters - reduced number of erythrocytes and leukocytes, eosinophilia, haemolysis and hemoglobinuria.

Differential diagnosis. Antimony poisoning and its compounds should be differentiate from unprofessional diseases of the respiratory, digestive and cardiovascular systems, skin diseases, and mucous membranes, poisoning with arsenic, cadmium, chrome and manganese.

Treatment. In case of acute antimony poisoning, antidote therapy (dimercaprole or D - penicillamine) is administered. In hemolysis cases, transfusion is recommended. Treatment of chronic poisoning involves general and other symptomatic therapy (in

* Acute poisoning - co-authored by N. Mirchev

Tab. 1. Toxicokinetics and toxicodynamics of metals

Element	Roads penetration	Landfills	Pathogenesis	Elimination
Antimony Sb	respiratory oral	lungs liver, kidney skin bones	connect the SH groups of enzymes violates protein and carbohydrate metabolism highly irritative effect carcinogenic effect?	urine faeces
Arsenic As	oral respiratory dermal	liver, kidney muscles, bones hair, nails, skin	connect with SH gr of enzymes, breaking cell metabolism	urine
Beryllium Be	respiratory oral dermal	bones liver spleen	irritant and allergenic effect carcinogenic effect? berylliosis is a systemic disease - granulomas in various tissues and organs	urine – 80%
Vanadium Va	respiratory oral	bones parenchymal organs	impairs the metabolism of vitamins (vit C), biogenic amines, lipids and carbohydrates	urine - 60% faeces - 10% hair, nails
Mercury Hg	respiratory oral dermal	parenchymal organs muscles	potent inhibitor of enzymes having an affinity for free radicals and predominantly sulfhydryl; decreases cont. of cytochrome P-450 and OMF	urine faeces saliva, sweat, hair, nails
Cadmium Cd	respiratory oral	kidneys liver bones	violates the metabolism of Ca and P; reduces the activity of α- antitrypsin in lung mucosa, associated SH gr. of cellular enzymes and proteins, direct irritating effect on mucous membranes carcinogenic effect	Urine> 85% Bile and feces - 10%
Tin Sn	respiratory oral dermal	lungs, kidney, liver spleen, bone	general toxic effect carcinogenic effect	faeces urine
Cobalt Co	respiratory oral	liver lungs thyroid adrenal glands kidneys	essential trace elements, activating certain enzymes regulating internal respiration and hemapoesis. disturbs tissue respiration (inhibits cytochromoxidase etc.) allergenic effect	urine
Manganese Mn	respiratory oral	brain liver, kidney pancreas muscles	direct damage to extrapyramidal structures (n. caudatus, gl.palidus, corpus striatum) by enzymatic inhibition violates metabolism of dopamine	via bile and feces urine
Copper Cu	respiratory oral dermal	liver kidneys pancreas	essential trace element; plays an essential role in hematopoiesis, in the activity of several enzyme systems, in the functions of the endocrine glands. it has expressed irritative and hemolytic effect.	by bile urine sweat, hair, nails
Nickel Ni	respiratory oral	liver spleen kidneys	local irritation action suppresses the activity of glutationreductase stimulates the synthesis of cholesterol allergenic effect carcinogenic effect	faeces (80%) urine bile sweat, hair
Lead Pb	respiratory oral	bones liver kidneys	block. SH gr of enzyme porphyrin metabolism disorders direct damages hepatocytes	urine - 80% feces - 15% sweat, saliva, milk - 1-5%
Selenium Se	inhaled oral dermal	kidney, liver muscles brain	essential trace element; block SH gr. of enzymes	garlic breath! urine faeces hair, sweat
Thallium Tl	oral dermal respiratory	parenchymal tissues	binds SH gr. and block enzymatic systems, disturbing cellular respiration and protein sinthesis. Bonding with riboflavin explain it's neurotoxicity.	faeces urine
Tellurium Te	inhaled oral dermal	bones liver	inhibit cholesterol biosynthesis	urine bile and feces
Phosphorus P	oral respiratory dermal	liver kidneys bones	binds to proteins and upset the function of enzyme systems regulating the oxidation processes in the cells, impairs the metabolism of carbohydrates blocking phosphorylation	urine
Zinc z n	respiratory oral	parenchymal organs bones muscles brain	essential trace element; participates in many enzyme systems violates the electronic transport in the mitochondrial respiratory chain inhibits cytochromoxidase, violates the secretory function of the pancreas	bile and feces urine - 20% sweat, hair, nails

pulmonary edema - glucocorticoids, calcium preparations, diuretics, oxygen therapy) **(Tabl. 2)**

Examination of working capacity. Acute antimony poisoning is most often associated with temporary loss of working capacity. Chronic poisoning results in a temporary or permanent limitation of working capacity for specific working conditions.

Prevention. Sanitary-technical prophylaxis. Compliance with safety requirements (ensuring effective ventilation, mandatory use of individual protective equipment, current control of antimony concentrations in the air in the working environment, etc.).

Medical prophylaxis. Do not come into contact with antimony and its compounds, persons with respiratory, cardiovascular and digestive disorders, skin and mucosal diseases. Periodic medical examinations are performed once every 12 months with the attendance of a therapist and by indications-dermatologist.

3.1.2. ARSENIC - V. PETKOVA, S. PAVLOVA

Arsenic is a metalloid widespread in nature in the form of compounds with metals and sulfur. Of more toxicological importance are its compounds: (arsenic (III) oxide, arsenites and arsenates). Arsenic trioxide is a starting product for the synthesis of a number of arsenic compounds for the processing of leathers, semiconductors, plant protection products. It is used as an initiator in the polymerization, a reducing agent in the production of glass. Arsenic salts (arsenates and arsenites) are used as pesticides and antiseptics for preserving wood.

Routes of intake, biotransformation and distribution in the body. *Arsenic compounds enter the body through the airways, the skin and the gastrointestinal tract. Absorb in the upper respiratory tract, but also through the lungs. In the body oxidation of the trivalent arsenic is achieved in a five-valent and vice versa - reduction of the five-valent to trivalent, which is significantly more toxic. It accumulates in nails, hair and skin, which, according to some, is due to its chemical keratin association. It is excreted through the kidneys, with maximum urine output during the first 6 hours of exposure. The excretion of arsenic with faeces is insignificant and is of greater importance in its organic compounds.*

Toxic effect. *The arsenic compounds are toxic. The toxic effect depends on the valency of the arsenic and the solubility of the compound. Arsenic, as a classical thiol poison, binds to the sulfhydryl groups of vital enzymes, e.g. succinate dehydrogenase, glutathione and the like, disrupting cellular metabolism. The arsenic antagonism of selenium, iodine and phosphorus is also indicated as a possible mechanism of toxic action.*

Clinical picture.

Acute poisoning. Acute poisoning occurs more often when the arsenic enters the mouth. They occur with the clinical picture of a local and absorptive toxic effect - acute gastroenterocolitis with cholera similar as symptomatology, toxic cardiopathy, hepatorenal syndrome, disturbed consciousness, epileptiform convulsions, coma. Inhalation of higher concentrations of inorganic arsenic compounds leads to acute upper respiratory and lung manifestations - acute rhinitis, pharyngitis, laryngitis.

Poisoning with **arsine**. It develops after a latency period of 3 to 6 hours and occurs with a picture of haemolytic anemia, hepatorenal syndrome (acute renal failure).

Chronic poisoning. Under production conditions, arsenic affects skin, respiratory organs, liver, kidneys, haematopoietic organs and the nervous system in prolonged exposure to low concentrations. Initial changes in the upper respiratory tract - chronic rhinitis, pharyngitis, laryngitis, gastrointestinal tract - chronic gastritis and gastroduodenitis as an expression of the irritant action of arsenic. Characteristic of the initial stages of poisoning are neurosensory manifestations as well as abortive forms of toxic sensory polyneuritis - pain and decreased sensation in polyneuritic type, acroparesthesias - predominantly in the lower limbs. At this stage, the hemostimulating effect of arsenic is also noted - with polyglobulin and hemoglobin elevations.

In the next stages of chronic poisoning, typical poisoning syndromes - toxic polyneuropathy - ascending, vegeto-sensory with marked pain syndrome are being developed. Signs of toxic hepatitis and initial toxic nephropathy appear. The abnormal changes in the respiratory organs are greatly enhanced. Perforation of the nasal septum may occur.

Severe forms of chronic poisoning resemble acute poisoning - pronounced hepatorenal syndrome, sensory motor polyneuritis, myelopolyneuritis with characteristic severe ataxia, so-called arsenic "pseudotabes", toxic anemia, toxic arsenic cardiomyopathy with rhythmic and conductive disorders.

Skin changes are characteristic of arsenic exposure. Palmar and plantar hyperkeratoses, arsenic melanomas, contact dermatitis, ulcerations, trophic disorders, alopecia may accompany chronic poisoning and are a sure sign of arsenic exposure.

Carcinogenic risk. Arsenic and its compounds are part of a group 1 of carcinogens for humans in the IARC classification. The most common is lung and skin cancer.

Laboratory diagnostics. Urine excretion increased in excess of 1.47 μmol/l, which is considered

Tab. 2. Clinic, exposure tests and treatment of metals poisoning

	Acute poisoning	Chronic poisoning	Exposure tests	Treatment
Sb	irritative manifestations in the eye and UAP metal fever pulmonary edema gastrointestinal syndrome toxic hepatitis nephropathy	rhinitis, nasal septum perforation, impaired sense of smell chronic bronchitis toxic pneumosclerosis myocardiopathy dermatitis and eczema lung cancer and bladder	Sb_y> 1,3 µg/l reduction of Er and leuco, eosinophilia, hemolysis, hemoglobinuria	Dimercaprol (BAL) 5% 5 ml Symptomatic
As	gastroenteritis hepatorenal syndrome intravascular hemolysis cardiomyopathy, epileptiform convulsions, coma	rhinopharyngitis, perforation of the nasal septum, gastroduodenitis, distal sensory-motor neuropathy in the lower extremities + autonomous violations cardiomyopathy with rhythmic and conduction disorders anemia tox. hepatitis and nephropathy hyperkeratosis and hyperpigmentation lung and the skin cancer	As_{urine}> 1,47 µmol/l Ask_{hair}> 3-5 mg/g dry mass As_{nails}> 5-10 mg/g dry mass	Acute poisoning: Dimercaprol (BAL) 5% 5 ml i.m. -3 mg/kg body weight in 12 h-24 h Chronic poisoning: Dimercaprol (BAL) 5% 5 ml i.m or Unithiol -5% 5 ml of i.m.; course-5 days Succimer (Chemet) - capsules -0,350 mg - day dose 2,1 g - 5 days $CaNa_2$ EDTA 10% 10ml i.v. (if lacking of these antidotes)-1 g in 3 consecutive days.
Be	irritative manifestations in the eye and UAP beryllium fever bronhobronhiolitis toxic pneumonia	granulomatosis lung dermatitis - ulcerative and granulomatous	Be_y> 0,02 mg/l SH group-s. In serum; FID; skin allergic testing Rö graphy biopsy of lung biopsy of skin granulomas	alkaline inhalations, oxygen therapy, diuretics, antibiotics, broncholytics, corticosteroids $CaNa_2$ EDTA 10% 10ml i.v. (Twice daily 1 g in 3 consecutive days.)
Va	Irritation of the eyes and UAP	bronchitis, pneumonia bronchial asthma diffuse pneumosclerosis (> 10 yr.exp) dermatitis, eczema	Decreases in serum cholesterol Increase in V_{bl}	$CaNa_2$ EDTA 10% 10ml i.v. Vit.C
Hg	cough, shortness of breath, cyanosis, toxopulmonitis, pulmonary edema, gastrointestinal syndrome, proteinuria, oliguria, ARF	gingivitis, stomatitis, pharyngitis dental erosions tremor, erethism autonomic dysfunction (hypersalivation, sweating, tachycardia, changes in dermography, polyuria) distal sensory polyneuropathy in the lower limbs + autonomous violations psychiatric disorders anemia -proteinuriya, nephropathy, RF chronic dermatitis and eczema	Hg_{bl} > 0,02 mg/l Hg_u > 10 µg/l SH gr. in serum psychological tests tremorografia	<u>Acute poisoning:</u> Dimercaptol - amp. 100 mg/ml, Unithiol amp. 25 mg/ml 1 mg per 10 kg weight i.m. <u>Chronic poisoning:</u> Unithiol -5% 5 ml i.m. 1 ampoule per day; Course 5 - 10 days Succimer - capsules 0,350g- 2,1 g daily; course - 5-7 days Na thiosulfuricum 10-20% 10 ml (applied in the absence of the above antidotes), course five days vitaminoterapy - B_1,B_6,vit.C tranquilizers neurotonics, vasodilators
Cd	metal fever cathars of UAP dyspnea, pneumonia, pulmonary edema gastrointestinal disorders hepatorenal syndrome kidney failure	anosmia, rhinopharyngitis emphysema, bronchitis, pneumosclerosis anemia tubulopathy with proteinuria Fanconi syndrome osteomalacia, spontaneous fractures lung and prostate cancer	Cd_{bl} > 5 µg/l Cd_u > 2 µg/l $\beta_2$mikroglobulin > 34µg /mmol creatinine	$CaNa_2$ EDTA 10% 10 ml i.v.- for 3 or 6 days Cuprenil tablets 0,250 g - daily 2 g - course 5 or 10 days Vit. D forte- ampoule 600 000 E i.m. once a month calcium supplements
Sn	irritative symptoms of eyes and UAP metal fever p.o. - gastroenterocolitis, hepatitis	bronchitis, emphysema pneumoconiosis lung carcinoma	Sn_u > 0,11 µg/l	symptomatically

Co	irritative symptoms of eyes and UAP cough, shortness of breath nausea, vomiting convulsions	rhinopharyngitis bronchitis, pneumonia, all. alveolitis, asthma myocardiopathy gastritis, peptic ulcer disease hyperkeratosis, ulcers, allergic dermatitis	polycythemia-increasing HB, Er, Ret, Eo dyslipidemia $Co_{bl.}$> 0,5 µg/l	$CaNa_2$ EDTA 10% 10ml i.v. Dimercaprol (BAL) 5% 5 ml i.m symptomatically
Mn	metal fever CUAP pneumonia	cerebrasteniya Parkinson's syndrome Psychoorganic syndrome encephalopathy	psychological tests EMG $Mn_{bl.}$ $Mn_{u.}$ are raised, but did not correlate with toxicity $Mn_{bl.}$ > 3,4 µmol/l; $Mn_{u.}$ > 0,9 µmol/l	$CaNa_2$ EDTA 10% 10ml i.v.- 3 last days anti-Parkinsonian drugs vit. E - high doses vit. C and B com.
Cu	copper fever allergic dermatitis rhinopharyngitis, gastritis, copper colic acute hemolysis, hemoglo-binuria, jaundice, ARF	rhinopharyngitis, gastritis copper colic greenish-yellow or greenish-black discoloration of the skin locally-ulceration of the cornea of the eyes, skin, nasal septum	increased cont. of Hb, and ER Cu_{serum} > 24 µmol/l	symptomatic treatment
Ni	inh- CUAP metal fever p.o.- gastrointestinal syndrome sk-dermatitis, eczema	allergic dermatitis and eczema metal fever sinusitis, anosmia, perforated the nose. septum bronchial asthma cancer of the nose, larynx and lung	$Ni_{u.}$ > 2,5 µg/l	$CaNa_2$ EDTA 10% 10ml i.v Dimercaprol (BAL) амп 5% 5 ml i.m.
Pb	haemolytic anemia abdominal colic tox. hepatitis ARF encephalopathy	lead facies, gingival Pb strip abdominal colic sideroachreocythemia (anemia) toxic hepatitis uric acid nephropathy asthenia, arthralgia, myalgia cerebrastenia motor polyneuropathy (nn. radiales) psychoorganic syndrome, encephalopathy	Pb_k >2,88 µmol/l (60 µg/dl) Pb_y>0,53 µmol/l 5-DALA>10 000nmol/4 h. SE >15‰; RE>0,5‰; FPP>100 mg/dl	$CaNa_2$ EDTA 10% 10ml i.v.- 3 consecutive days, as necessary twice a repetition of the course at intervals of three days. CEMET capsules 0,350 g- 2,1 g daily -5 days Cuprenil tablets 0,250 g - 2 g daily - seven days
Se	inh - irritative events eyes and UAP; pneumonia, pulmonary edema skin burns	conjunctivitis, rhinopharyngitis, bronchitis reddish coloration of the skin gastrointestinal complaints neuropathies par. dermatitis, dispigmentation paronychia, alopecia	$Se_{bl.}$>0,4 mg/l $Se_{u.}$>0,1 mg/l	Respiratory resuscitation glucocorticoids, aqueous salt solutions When skin burns - unguents with a 10% solution of Natrium thyosulfuricum Vitaminotherapy
Tl	alopecia gastrointestinal disorders ascending paralysis, coma	alopecia fatigue, weakness distal sensory-motor polyneuropathy in the lower limbs, - axonal type sometimes lesion II, III and IV CN	hypokalaemia tubular proteinuria EMG $Tl_{u.}$ > 10 µg/l	Acute poisoning: Antidotum metallorum 100 ml in stomach tube, then rinse with water and abs., saline cathartic; drop infusion with an aqueous salt solution In chronic poisoning: Natrium thyosulfuricum-20% 10-50 ml CaNa2 EDTA 10% 10ml Dimercaprol (BAL) Amp 5% 5ml i.m. Vitaminoterapy -B, C
Te	irritative events in UAP gastrointestinal syndrome haematuria	metallic taste in mouth, nausea, vomiting	$Te_{u.}$ > 0,05 mg/l	$CaNa_2$ EDTA 10% 10ml i.v.
P	p.o. - Gastroenteritis, hepatorenal syndrome, hemorrhagic diathesis, jaundice, convulsions, delirium, coma. inh.-cough, dyspnea, pneumonia, pulmonary edema, cardiac arrhythmia. ataxia, paraesthesia, tremor, diplopia, convulsions, in contact - heavy burns	rinofaringolaryngitis, pneumonia chronic bronchitis, COPD chronic gastroenteritis anemia subikter toxic hepatitis toxic nephritis necrosis of the jaw spontaneous fractures of long bones	increase the activity of liver enzymes, hyperbilirubinemia leukopenia, hematuria, proteinuria	p.o.-gastric lavage with water and potassium permanganate; saline laxatives; 15 ml of a 1% solution of $CuSO_4$, intravenous i infusions, AB, hepatoprotective treatment 10% 10 ml of calcium gluconate; locally-3% solution of $CuSO_4$ - neutralize P on skin. vitaminoterapy

Cr	tracheobronchitis, pneumonia pulmonary edema gastroenteritis hepatorenal syndrome uraemia contact - allergic dermatitis, ulcers type "bird's eye"	conjunctivitis, keratitis atrophic rinofaringolaringiti Nasal septal perforation bronchitis, emphysema, bronchial asthma toxic pneumosclerosis Are bronchi (after 15-30g exp.) gastritis, ulcer perforation toxic hepatitis and nephritis (rare) anemia	reduced court. HM, ER, anisocytosis, poikilocytosis were, cells Heinz in Er.; leukocytosis with neutrophilia and eosinophilia methemoglobinemia> 2% Cr_{Er} >1 µg/l Cr_{y} >5 µg/l	symptomatic vitaminoterapiya - B_1; B_6; B_{12} in allergic dermatiti-corticosteroid ointments
zn	metal fever gastroenteritis uraemia Local $ZnCl_2$ - skin and eye burns	nasal sepum perforation bronchitis, pneumonia, pneumosclerosis gastroenteritis early atherosclerosis, arrhythmias hypochronic anemia neurasthenic syndrome allergic dermatitis	reducing HB, Er, leukocytosis hyperglycaemia glucosuria Zn_{serum} >24µmol/l	$CaNa_2$ EDTA 10% 10ml i.v. symptomatically

to be the most reliable indicator of occupational risk. The determination of arsenic in blood, urine, hair and nails is used as a biological marker for arsenic exposure. In the blood, in the initial stages - elevated Hb and erythrocytes, in more advanced cases of poisoning - normochromatic anemia, thrombocytopenia, granulocytopenia.

According to the latest data, arsenic disturbs heme synthesis by altering the activity of coproporphyrin oxidase, hemsyntetase and hemoxygenase. As a result, elevated levels of uroporphyrin, coproporphyrin and bilirubin in the urine were observed.

Differential diagnosis. In the differential diagnosis plan, alcoholic and diabetic coma, thallium intoxication, acrylamide polyneuropathy, polycythaemia vera, Addison's anemia, dimethylsulfate, antimony, chlorine, chromium, etc. intoxications should be considered.

Treatment. Treatment is etiopathogenetic. Chelation therapy with $Ca\ Na_2$ EDTA - 10% - 10 cc i.v. Healing course - 9 g in three three-day courses with a 3-day rest between them. Symptomatic treatment. Organoprotectors. Antioxidants - selenium as cofactor of glutathione peroxidase is thought to reduce arsenic toxicity.

Prognosis and labour-medical expertise. Permanently labour readjustment is required in residual manifestations following acute or chronic arsenic poisoning such as, for example, polyneuritis, perforation of the nasal septum, toxic hepatitis or precancer hyperkeratosis and arsenic melanosis.

Prevention. Preliminary medical examinations aiming at non-admission to work with arsenic compounds of persons with chronic skin diseases, often recurrent diseases of the respiratory organs, liver and bile ducts. Periodic medical examinations are performed once a year with the participation of internist, neurologist, ORL specialist and dermatologist.

Exposure control is performed by surveying the content of arsenic in urine, hair and nails.

Diet D from the preventive diet is recommended.

3.1.3. BERYLLIUM – T. KUNEVA

- tracheobronchitis, pneumonitis
- granulomatosis of the lung
- dermatitis (ulcerous and granulomatous)
- irritative manifestations in the eyes, nose, mouth

Beryllium in nature is not found in pure form but contains some minerals, of which the most common is beryl $Be_3A_{12}/SiO_3/_6$ and chrysoberyl $BeAl_2O_2$.

Pure beryllium is a silver-gray metal with a high melting temperature (1280 °). It does not interact with water, dissolves in dilute acids and bases. At high temperature forms compounds with oxygen, sulfur, nitrogen, halogens.

***Use** - due to its valuable properties - high strength, hardness, heat resistance and delayed oxidation, beryllium is widely used in industry: in the machinery and the building industry, in the production of corrosion resistant alloys, electronic equipment, X-ray tubes, radio fluorescent and neon lamps, ceramic paints.*

***Occupational exposure** - long-term exposure to dust and smoke exposed workers involved in the grinding and roasting of beryllium-containing ores and in the production of beryllium alloys. Significantly lower is the risk for persons in contact with beryllium compounds used in aircraft and electronics industry.*

***Absorption, metabolism, excretion.** Beryllium compounds have low absorption after inhalation, ingestion, or skin contact. Beryllium may last longer in the lungs or be deposited in the bones, liver and spleen. Renal excretion is weak and variable and confirms exposure because it is not detected in exposed individuals.*

***Pathogenesis.** Beryl and its compounds have local irritant, general toxic, allergenic and carcinogenic effects. Their toxicity is determined by their solubility and dispersion - soluble and more strongly dispersed are more toxic. Soluble compounds most often cause acute poisoning, and insoluble - chronic intoxications.*

In chronic intoxication no dose-effect dependence was observed.

Clinical picture. Berylliosis is a systemic disease, which is confirmed by the concurrent presence of granulomas in some tissues and organs - lungs, liver, skin, lymph nodes.

Acute intoxications may occur in the form of:

Cataraural inflammation of the mucous membranes of the UAP: rhinitis, pharyngitis, tracheitis. They run out relatively mildly and quickly - a few days or weeks.

Beryllium fever - a few hours after inhalation of a vapour occurs a prodromal period, manifested by general weakness, headache, nausea, vomiting, dry cough, shortness of breath. After 4-5 hours the real fever period begins - the temperature rises sharply to 39-40°C, after 5-8 hours it falls with abundant sweating. Blood is found in leukocytosis and rapid transient hyperglycemia.

Severe form of acute berylliosis - broncho-bronchiolitis and toxic pneumonia. Characteristic is the stormy start. It is possible to develop interstitial myocarditis, hepatitis, nephropathy. Radiographically, emphysematous changes and a number of small but intense homogeneous shadows are seen that are not clearly delineated and do not merge with each other. Bronchiolitis and pneumonia sometimes occur with high temperature and fever, recurrences are possible. Danger of acute pulmonary edema.

Chronic berylliosis occurs as a result of acute intoxication or as a primary chronic disease. Often it develops with minor acute exposure to beryllium or many years after termination of the contact. The latency period ranges from several weeks to 15 years. There have been cases of chronic berylliosis in people living near beryllium production. The onset of the disease may be gradual, unnoticed, with minor complaints or violent with pronounced clinical symptoms. For the fast-progressing form of chronic berylliosis, it is characterized by an acute onset of high temperature, fever, shortness of breath, paroxysmal cough, chest pain, abrupt weight loss (10-12 kg). Dyspnoea is a leading and most symptomatic symptom. Compensatory erythrocytosis (arterial hypoxemia), dysproteinemia, an increase in γ-globulins were observed in the blood.

Depending on the nature of radiological changes in the lungs, two forms differ: granulomatous and interstitial, and depending on their expressions - three stages.

The granulomatous form is heavier with the presence of focal shadows (granulomas), with the extension of the lung roots due to lymph node hyperplasia and early development of the pulmonary heart. Granulomatous berylliosis often involves hepato-renal and articular syndrome, bone damage with thick ribbed and long tubular bones periosteum and skin granulomas. The interstitial form of chronic berylliosis is clinically benign and is usually limited to stage I. Typical are "standstill radiiograms" with finely-reticulate design. Possible degenerative lesions of the liver and kidneys.

The most common skin lesions associated with beryllium are contact and allergic dermatitis, eczema, ulcers and granulomas. Typically, the exposed parts of the face, hands and neck are affected. The beryllium granulomas are slow running with fistulae, forming for months. Beryllium is often found in a biopsy.

Beryl and beryllium compounds are proven carcinogens for humans and it is necessary to avoid direct contact with them.

Laboratory diagnostics. In the case of acute pneumonitis, arterial hypoxemia is detected. Chronic berylliosis has hypergammaglobulinaemia, anemia, elevated liver enzyme activity, and hyperuricemia. Unlike sarcoidosis, angiotensin converting enzyme serum levels are normal.

The diagnosis of beryllium poisoning is based on proven contact, clinical syndromes, the presence of beryllium in urine over 0,02 mg/l, a positive skin test with its soluble salts. Skin tests prove sensitization to beryllium compounds. Lymphocyte blast transformation is an alternative to an effective individual assessment of reactivity to beryllium.

Differential diagnosis of acute berylliosis is made with colds, and chronic - with hemosiderosis, carcinomatosis, sarcoidosis, Hamman-Pich syndrome, miliarl tuberculosis. Diagnostic puncture biopsy of the lung is used in the most difficult diagnosis.

Treatment. In case of acute nasopharyngitis and tracheitis against the irritant phenomenon of UAP, alkaline inhalations are employed. A 2% solution of ephedrine is dripped into the nose. Anti-tussive and antihistamine agents are used.

In case of beryllium fever, intravenous administration of 40% glucose with ascorbic acid, intake of large quantities of sweet tea is recommended, as indicated - alkaline inhalations.

Acute bronchoconstriction and pneumonia require intensive and complex therapy for the elimination of acute respiratory failure and pulmonary edema - oxygen therapy, diuretics, antibiotics, broncholitics, corticosteroids. In case of skin and eye contact, thorough washing with water is required. In the presence of beryllium ulcers and granulomas, surgical wound processing and topical treatment with corticosteroid ointments is necessary.

The most commonly used antidote for acute poi-

soning is $CaNa_2$ EDTA twice a day for 1 gram in a 5% glucose solution of 250 cm^3, intravenously, three days in a row.

For the treatment of chronic berylliosis, antibiotics, oxygen therapy, corticosteroids are administered. In patients in stage III corticosteroid therapy is continuously administered with depot preparations, the dose depending on the clinical symptoms.

Prognosis. In mild acute poisoning - complete recovery with normalization of the X-ray picture; in severe forms of acute berylliosis (bronchiolitis and pneumonia) - development of interstitial pneumocosclerosis and chronic disease. At high concentrations of vapors and powder from beryllium compounds a lethal outcome is possible due to severe pulmonary edema.

Labour expertise. Temporary labour readjustmentwithout contact with beryllium is recommended for isolated ocular involvement (conjunctivitis, blepharitis), catarrh inflammation of the UAP, fever. Termination of contact is required after severe acute intoxication and in patients with chronic berylliosis. Manifest beryllium intoxication requires long-term treatment of patients who are at least with permanently labour readjustment.

Prevention. Prophylaxis of beryllium poisoning involves local and general ventilation, equipment sealing, remote control. It is obligatory for the workers to wash with a hot shower after work. Personal protective equipment is subject to daily cleaning.

Preliminary and periodic medical examinations are mandatory. Contraindications for getting into work with beryllium are chronic diseases of the respiratory and cardiovascular system, liver, gastrointestinal tract, organic CNS diseases, allergic, endocrine, blood and skin diseases. Periodic medical examinations are conducted once every 6 months with the participation of a therapist and an otorhinolaryngologist. Laboratory tests: blood count, FID, X-ray of the lungs.

3.1.4. KADMIUM - T. KUNEVA

Cadmium has similar physical and chemical properties as zinc and occurs naturally with it. In minerals and ores cadmium and zinc are usually in the ratio of 1:100 to 1:1000.

Cadmium gives high corrosion resistance to steel, iron and a number of other metals, which also determines its primary use for galvanic coatings of metal parts in the airplane-, ship-, machine-, and car engineering, electronics and electrical engineering. Today, however, only 8% of the processed cadmium is used for alloys and coatings. Cadmium-containing batteries are small, portable, easy-to-use and are used in mobile phones, resulting in a rapid increase in their use (55% of all cadmium used in the industrial countries in 1994 was for batteries).

Cadmium compounds (30% of the use in developed countries) are used as pigments and stabilizers in the manufacture of plastics as well as for alloys (3%).

Cadmium compounds are also used as paint pigments in the porcelain and glass industry (cadmium sulphide); as fungicides (cadmium chloride); semiconductor production (cadmium oxide).

***Occupational exposure** - cadmium vapors and dust are found in workers involved in the melting and refining of lead, copper and zinc ores. The following professions, classified by weight: casters at cadmium production, operators of electrolysis apparatus, press operators of cadmium-nickel accumulators, operators and fitters in maintenance, galvanic technicians, are also threatened.*

***Absorption, metabolism, excretion.** In occupational exposure, the main pathway to cadmium in the body is respiratory - by inhalation of cadmium dust or cadmium oxide vapors. Absorption in the lungs is between 20% and 30% (smokers up to 50%) and depends on the size and chemical composition of the particles. Gastrointestinal absorption of cadmium is between 2 and 6%. In people with low iron stores (low serum ferritin concentrations), higher cadmium absorption (over 20%) may occur. Gastrointestinal absorption also increases in calcium, zinc and protein-poor foods.*

Skin absorption is negligible.

*After absorption through the lungs or the gastrointestinal tract, the cadmium is transported to the liver, where it is linked to a low molecular protein rich in sulfhydryl groups - metallothionein (MT). About 80% - 90% of the total cadmium in the body is bound to metallothionein. Small amounts of metallothionein-bound cadmium constantly leave the liver and are transported to the kidneys. The kidneys and the liver are the **main cadmium depots** in the body (containing about 50% of the body load). Kidney concentrations in the renal cortex exceed about 15 times the liver concentrations prior to kidney damage.*

Elimination of cadmium is very slow - it accumulates in the body. The biological half-life of cadmium in humans varies from 7 to 30 years.

Clinical picture. Acute poisoning occurs relatively rarely. Two clinical forms are known: inhalation and digestive-resorption. The inhalation form is most often the result of inhalation of dust or vapours from cadmium oxide. Common symptoms occur after a latency period of several hours (1 to 8), most commonly flu-like and resembling metal fever - pain throat, nausea, metallic taste in the mouth, headache, myalgias. When the disease progresses, chemical pneumonia develops - coughing,

tightness in the chest, shortness of breath, fever. In the most severe cases, pulmonary edema and respiratory failure death occur. In severe forms of acute poisoning, pulmocardial (cardiovascular decompensation) and hepatic syndrome (toxic hepatitis) may develop. Acute exposure may be a cause of chronic pulmonary fibrosis.

In the conditions of **prolonged cadmium exposure**, the critical organ is the kidney and the most characteristic symptom is proteinuria. The nephrotoxic effect of cadmium is manifested by: tubular dysfunction - increased excretion of low molecular weight proteins (β_2-microglobulin, lysozyme, ribonuclease) and glomerular dysfunction - increased excretion of high protein molecules (albumin, transferrin, orosomucoid, immunoglobulin, urea, low creatinine clearance). Prolonged exposure is associated with Fanconi syndrome - aminoaciduria, glucosuria, hypercalciuria, phosphaturia. Renal impairment is irreversible and progresses after discontinuation of exposure. Renal tubular dysfunction may be a cause of nephrolithiasis and osteomalacia.

With prolonged contact with cadmium - over 20 years, bone is damaged. Pain in the bones does not have a strict localization, often occurs when moving or changing the position of the body. ("Itai-Itai disease", a severely painful type of osteomalacia with multiple fractures and renal dysfunction due to water and rice in environmentally contaminated cadmium areas, has been observed in Japan). Other clinical manifestations of chronic cadmium intoxication include anosmia, chronic rhinopharyngitis, pulmonary emphysema, pneumocosclerosis, mild hypochromic anemia, chronic gastritis, light yellow to golden brown dental pigmentation. Prolonged exposure (over 20 years) of cadmium in the air in the work environment certainly leads to the development of pulmonary emphysema. Cadmium is a carcinogen. In several large epidemiological studies, a dose-response relationship has been demonstrated between the growing lethality of lung cancer in cadmium-exposed workers. Interpretation is complicated by the competitive exposure of other metals that are known to be suspected carcinogens.

Laboratory researches. Blood cadmium - normally in non-professional contacts is less than 5 μg/l. Early toxic effects - at values above 10 μg/l; cadmium in urine - less than 2 μg/l. Renal impairment can be expected at concentrations above 0.01 mg/g creatinine. Trace excretion of β_2-microglobulin, lysozyme, albumin and total protein. β_2-microglobulin in urine should not exceed 34 μg/mmol creatinine.

Differential diagnosis - endemic nephropathy, other toxic nephropathies (beryllium, zinc, chromium, manganese, antimony).

Treatment. For acute poisoning, use of BAL (Dimercaprol) 100 mg every 6 hours intramuscularly or $CaNa_2EDTA$ of 0.5 g in 500 ml isotonic solution twice daily is recommended. With continuous control of renal function.

The inhaled form includes broncholytic and dehydration agents, intravenous glucose infusions with vitamin C, antibiotics against pneumonia.

The treatment of the digestive-resorption form is carried out by gastric lavage with sodium sulphate solution and medical charcoal, infusion resuscitation with water-salt and glucose solutions, hepatoprotective therapy with glucocorticoids, B and C vitamins, in severe forms - early dialysis.

In chronic poisoning: antidote therapy involves administration of $CaNa_2EDTA$ 10% 10 ml intravenous ampoules in 5% glucose or physiological saline solution. The antidote is given once a day for 3 or 6 days.

Symptomatic treatment - depending on the type and extent of organ damage.

Once a month, it is desirable to take vitamin D forte - ampoule - 600,000 ME intramuscularly. Prophylactically against bone damage is appropriate for calcium preparations.

Prognosis - favourable in timely diagnosis and discontinuation of exposure prior to glomerular dysfunction and bone fractures.

Expertise of working capacity - balneotherapy, vitaminotherapy (B, C, D) in high doses is recommended for cadmium carriers. Manifest chronic cadmium intoxication requires long-term treatment and long-term rehabilitation (% reduced ability to work without contact with toxic substances).

Prevention is aimed at lowering the level of contamination, using personal protective equipment and regular prophylactic examinations of the exposed ones.

Contraindications for work in contact with cadmium are atrophic rhinitis, chronic diseases of the lungs, kidneys, liver, organic neurological diseases.

Periodic medical examinations are carried out once a year, with the participation of a therapist and dentist, as evidenced by an otorhinolaryngologist. The early cadmium effect screening test is the determination of protein in the urine, along with the mandatory determination of cadmium in biological fluids. It is recommended that diet B be taken from protective professional nutrition.

3.1.5. CHROMIUM - J. HADJIEVA

The chromium is a hard metal with a grayish-white color. It dissolves easily in hydrochloric and sulfuric acid and is insoluble in water. Chromium exists in three valent forms (elemental chromium, trivalent chromi-

um (III) and hexavalent chromium (VI).

Trivalent chromium is an essential microelement, unlike hexavalent chromium, which is highly toxic.

Usage. *Chromium and its compounds are used in metallurgy and machine building, in the chemical, textile, ceramic, pharmaceutical and electronics industries*

Biotransformation. *Chromium and its compounds fall into the body via the respiratory tract, the skin and the gastrointestinal tract The chromium absorption depends on its valency and is its electrical charge. Hexavalent chromium (VI) is absorbed faster than trivalent chromium (III). Chromium accumulates primarily in the lungs, liver, spleen, pancreas, and bone marrow. A significant portion of the body's chromium (VI) is reduced to chromium (III).* ***Elimination*** *of chromium occurs mainly in the urine.*

Pathogenesis. *The pathogenetic mechanism of action of chromium is relatively well studied. It has a general toxic effect - on the liver, kidneys, hematopoiesis, cardiovascular system and others. The stimulating effect of chromium on cholesterol synthesis is known to cause the development of early atherosclerosis. Its role as a glucose tolerance factor is undeniable.*

Chromium has a locally irritating and sensitizing effect on the skin and mucous membranes. Its allergic effect on the skin is mainly due to the reduction of chromium (VI) to chromium (III) and the formation of protein complexes with antigenic properties provoking the appearance of antibodies to chromium.

His carcinogenic effect is well known.

Clinical picture. The severity of the clinical course of poisoning is mainly determined by the intensity of exposure and the manner in which the toxic substance is present in the body.

Acute inhalation poisoning involves symptoms of tracheobronchitis, bronchopneumoniae -pains behind the sternum, shortness of breath, cough, and in severe cases - development of pulmonary edema.

Acute oral poisoning - leading to symptoms of the gastrointestinal tract - persistent vomiting, severe colic pain, profuse diarrhea and subsequent dehydration, hypovolaemia and acute heart failure. Later symptoms of toxic nephropathy and acute renal failure were observed. It is also possible to diagnose heterologus rate of hepatorenal syndrome.

Chronic poisoning. Chronic chromium poisoning is characterized by irritative changes in the upper respiratory tract and the lung, atrophic rhinitis, pharyngitis, tracheitis, chronic bronchitis, pulmonary emphysema, bronchial asthma, pneumocosclerosis. The carcinogenic effect of chromium is predominantly directed towards the lungs, nasal cavity and sinuses. The shortest exposure to lung carcinoma is over 8 years and the incidence increases in 20 years. Proven carcinogenicity has zinc chromate, calcium and lead chromates and chrome semiproducts. Possible carcinogens are also the bichromates of lithium, potassium, rubidium and others.

Changes by other organs and systems: gastrointestinal tract - manifestations of dyskinesias, often exacerbating gastritis and ulcers with perceptions of perforation; liver and kidneys - slight functional impairment may develop to cirrhosis of the liver, nephrosonephritis; mucous membranes and skin - surface ulceration of the nose mucosa, perforation of the initial part of the nasal septum. Changes in the skin are manifested by hand sores localized between the fingers and nails, sometimes penetrating to the bone - type "bird eyes", allergic contact dermatitis and eczema.

Clinical-laboratory tests. Haematological syndrome-low hemoglobin, decreased red blood cell, anisocytosis, poikilocytosis, polychromasia; Heinz cells in erythrocytes, leukocytosis with neutrophilia and eosinophilia, increased methaemoglobin content in the blood, sometimes establishing chromosomal aberrations in leukocytes. Renal syndrome-proteinuria, haematuria, anuria and increased content of residual-nitrogen fractions in blood serum.

Determination of chromium in erythrocytes (standard values up to 1.0 μg/l) and urine (standard values up to 4-5 μg/l) is used as an exposure test.

Differential diagnosis. Acute poisoning with chromium is distinguished from other acute poisonings caused by mercury, cadmium, zinc, beryllium, manganese, acids, bases, nitrous gases and others. Chronic poisoning requires a differential diagnosis with unprofessional diseases of the respiratory and urinary systems, liver, skin and mucosal diseases.

Treatment. In acute poisoning, procedures are performed to stop poisoning in the body (skin and mucous membranes with aqueous sodium bicarbonate, gastric lavage with water and medicinal charcoal). Corticosteroids, dialysis procedures and the like are administered as indicated. A good effect is seen when using $CaNa_2EDTA$ and D-penicillamine.

The treatment of chronic poisoning includes mucosal protectors, vitamin therapy, general strengthening and other symptomatic remedies.

Labour expertise. Acute poisoning with chromium is most often associated with temporary loss of working capacity. In severe poisoning, the prognosis is a serious-possible lethal outcome. Chronic poisoning can cause temporary disability, and irreversible damage to the respiratory system, liver, kidneys - permanent impairment of working capacity.

Prevention. Sanitary and technical prophylaxis - sealing, effective ventilation (general and local - above metal and leather chrome plated baths), use

of individual protective equipment (special working clothes, gloves, etc.).

Medical prophylaxis - Preliminary medical examinations upon entry into employment. Do not work in contact with chrome, people with respiratory, digestive and urinary system diseases, with liver, skin and mucous membranes diseases. Mandatory periodic medical examinations are conducted once every 12 months with the participation of a therapist, an otorhinolaryngologist, and as evidenced-dermatologist, ophthalmologist and dentist.

3.1.6. COBALT - J. HADJIEVA

The cobalt is hard, hardly melting gray-white metal. Dissolves in dilute sulfuric and nitric acids.

Usage. *Cobalt and its compounds are widely used in the ceramic and glass industry. They are used as catalysts in some chemical processes (ammonia oxidation, nitric acid production, etc.), in the pharmaceutical industry and metallurgy in the production of special alloys. Cobalt alloys are used in the production of ferromagnets, in jet engines and others.*

Biotransformation. *Cobalt falls into the body by the respiratory and digestive tract. It comes in all soft tissues. Higher levels are found in the liver, lung, thyroid, adrenal glands and kidneys. Relatively lower is its content in skeletal muscles, heart, spleen, pancreas, brain. Inorganic cobalt is eliminated primarily (up to 80%) with the faeces unresorbed. Resorbed metal is excreted in the urine, less with bile, sweat and in hair.*

Pathogenesis. *As an essential oligoelement, cobalt activates a number of enzymes regulating tissue respiration and hematopoietic system. In elevated exposure, it exhibits toxic effects:*

- *disturbs tissue breathing (inhibits the activity of oxidative-reducing enzymes - cytochrome oxidase, succine dehydrogenase, catalase, etc.);*
- *allergies (allergic dermatitis, eczema, bronchial asthma, etc.);*
- *cardiotoxic, immune and embryotoxic effects.*

Clinical picture. Acute intoxications - occur very rarely. Cobalt's inhalation in the body causes a tingling cough, tightness and chest pain, shortness of breath, convulsions, hyperthermia, collapse. When the site of entry is the gastrointestinal tract, nausea, vomiting, epigastric pain, etc. are observed. In severe cases of poisoning, lethal outcome is not excluded.

Chronic poisoning. Under production conditions only chronic poisoning occurs. On the background of the general toxic effect of the cobalt, there are disorders of the respiratory system - development of chronic rhinitis, laryngitis, pharyngitis, chronic bronchitis, chronic interstitial pneumonia, allergic obstructive syndrome. Recently, cases similar to the clinical picture of exogenous allergic alveolitis have been described. The cardiotoxic effect of cobalt is well known - the so-called "cobalt myocardiopathy" with development of dystrophic (metabolic and ischemic) processes in myocardium and hypotension. Gastrointestinal syndrome is manifested by lack of appetite, development of chronic gastritis, ulcerative stomach disease, etc. Disorders of the endocrine system are most often thyroid-related. Its structure and function is impaired as a result of troubled oxidation processes and thyroxin synthesis. Skin manifestations include hyperkeratosis, skin ulceration mainly in the exposed parts of the body, allergic dermatitis and eczema. It is believed that the development of the so-called "cement eczema" is mainly due to the mutual action of cobalt and chromium. The carcinogenic effect of cobalt is debatable. His immune embryotoxicity was studied primarily under experimental conditions.

Clinical-laboratory tests. Hematological parameters - polycythemia (increased red blood cell, reticulocytes, eosinophilia, basophilia), increased hemoglobin and low serum iron, prothrombin time prolonged. Clinico-chemical indicators - total lipid, beta-lipoprotein, cholesterol and triglycerides as well as total protein and α_1 and α_2 - globulin fractions are increased. Toxic-chemical indicator - determination of cobalt in whole blood (standard values up to 0.5 μg/l).

Differential diagnosis. Chronic poisoning with cobalt imposes a differential diagnosis with unprofessional diseases of the respiratory, cardiovascular, digestive and endocrine systems, with skin diseases, etc.

Treatment. Acute poisoning that occurs as a result of entry through the gastrointestinal tract requires immediate washing of the stomach with water and medical charcoal, The shock condition requires the inclusion of antidote (dimercaprole, $CaNa_2EDTA$, unitiol), anti-collapses, etc. In case of chronic poisoning, general supportive symptomatic therapy is used.

Labour expertise. Acute intoxications are most often associated with temporary loss of ability to work. Chronic poisoning with marked symptoms by the respiratory and cardiovascular systems necessitates permanent rehabilitation without contact with toxic substances and, in some cases, identification of a disability group.

Prevention. Sanitary-technical prophylaxis - automation and sealing of production processes, use of personal protective equipment, personal hygiene, etc.

Medical prophylaxis-mandatory medical exami-

nation of incoming persons. Do not work in contact with cobalt and its compounds, persons with diseases of the respiratory, cardiovascular, digestive and endocrine systems and diseases of the skin. Periodic medical examinations are performed once every 12 months with the participation of a therapist and an indication of a dermatologist. Protective professional nutrition - diet "D" is recommended.

3.1.7. LEAD
D. CHARAKCHIEV, ST. ANDONOVA

Professional exposure to lead and its compounds poses a significant risk to the health of workers from non-ferrous metallurgy, production of tetraethyl-lead, industrial and artistic ceramics, battery production, the production of lead minium and lead glass, polygraphy, plastics production, etc. It is also one of the most important contemporary environmental pollutants.

Biotransformation. *Occupational exposure occurs mainly by the airway and less amount of digestive tract. Organic compounds of lead enter the skin. Reabsorbed lead is associated with hemoglobin and low molecular weight erythrocyte proteins, less with plasma albumin and gammaglobulin, or circulating as free ions. About 90% of lead accumulates in bones such as tribasic lead phosphate, and the remaining amounts are deposited in the liver, kidneys, brain structures, muscles, skin, hair, teeth, etc. Lead passes blood-brain and placental barriers as its concentration in the blood of the fetus is close to that of the mother. It is inactive in the bone depots, but in various conditions that alter the alkaline-acid balance such as infections, alcochol excesses, the reception of certain drugs (barbiturates, sulfonamides) and foods lead to its release and clinical manifestation of intoxication.*

Elimination of lead from the body to 80% occurs in the urine, up to 15% through bile and faeces, and the remaining amount with sweat, saliva, mammary glands, dropping corneous layers, hair, nails, etc.

The half-life of lead from the whole body at interruption of exposure is about 5 years, and bones and teeth more than 20 years. As safe levels of lead in blood, so-called plumbemia in professionaly exposed recommended to 400 μg/l, (for unexposed adults - about 150 μg/l and for children - around 100 μg/l, and for certain industries and environmentally polluted areas these levels are specifically assessed).

Pathogenesis. *Lead in its toxicological characteristics refers to the so-called thiolic poisons due to its ability to block sulfhydryl groups and other free radicals predominantly of enzyme structures, leading to disruption of metabolic processes and functions at different levels:*

- Porphyrin metabolism disorders - lead blocks normal biosynthesis of porphyrins by altering the bioactivity of enzymes, which carry out the synthesis of delta-aminolevulinic acid (DALA) from succinic CoA and glycine, and significantly more those participating in the synthesis of the porphobillinogen. This leads to increased accumulation in the blood and secretion of DALA, uroporphyrinogen III, coproporphyrin III and protoporphyrinogen IX. The other vulnerable level is disturbed heterologous synthesis of protoporphyrin IX and the blockade of the co-dehydrazes I and II involved in the inclusion of iron in the porphyrin ring and the formation of hemoglobin. As a result of these disorders, anemia with the development of compensatory reticulocytosis (lead found to have not only a blocking effect on the enzymes involved in porphyrin synthesis, but at some stages induce them - with less exposure). Lead has a direct toxic effect on red blood cells.

- Disturbance by nerve structures - an expression of direct damage to the vegetative and central brain sections, and the increased amount of porphyrins and their potential vasoconstriction. Surexcitation of the vegetative nerve structures determines lead colic, atonic manifestations of the digestive system, vasospasm of the vessels and elevation of blood pressure.

- Liver disorders - lead directly damages hepatocytes, resulting in changes in the detoxic, protein, enzyme, pigmentary and carbohydrate functions of the liver.

Diffuse degenerative changes are observed in nerve cells of the central brain structures, anterior horns of the spinal cord and peripheral nerve stems, hepatocytes, etc.

Clinical picture: Acute poisoning due to occupational exposure is rare. It can have a way of living character. It is characterized by the appearance of hypersalivation, epigastric pain, enterocolitis, or the development of typical "lead colic". Simultaneously with this symptom, anemia with hemolysis, signs of toxic hepatitis, nephropathy, neurological manifestations, polyneuritis is rapidly developing. Arterial pressure is increased. In the first hours there is anxiety, headaches that can pass into somnolentia and coma with severe cardiovascular failure. After acute acute intoxications with lead, residual albuminuria, astenovegetative events and encephalopathy may occur.

Chronic lead intoxication. The following syndromes are identified:

Lead facies, which is observed in heavy lead intoxications and characterized by a grayish-pale, earth-like colour of the face that disappears with improvement of the condition. Symptoms of professional plumbism in the *oral cavity* are lead spots or strips of grayish brown on the front gingiva rib, and a more often sweet-metal taste, anemia of the lips

and gingiva, violet tinge of cheeks and tongue, and slowly progressing dystrophic paradontopathy.

Neurological syndrome - asthenovegetative manifestations as an early sign of lead intoxication: vegetative polyneuropathy and encephalopolineurial syndrome as an expression of severe lead intoxication.

Gastrointestinal syndrome - with a number of disorders in the gastrointestinal tract, the most common is lead-colic - colic pain in the abdomen, around the navel of varying intensity and duration and often occurs with persistent constipation. It is accompanied by increased blood pressure and bradycardia (it is necessary to differentiate from "sharp surgical abdomen" here abdomen is soft).

Anemic syndrome - characterized by a decrease in hemoglobin and a reduction in erythrocyte counts, reticulocytosis and a sporadic increase in stipling erythrocytes in peripheral blood.

Hepatic syndrome - with laboratory abnormalities in liver function in mild intoxications (elevation of serum transaminases, LDH, decrease in ChEA), blockage of hepatic glycogen synthesis with increased glycogenokinase, disorders of bile pigment metabolism, damage to protein synthesis with manifestations of hypoproteinaemia, hyperglobulinemia and others. Subjective evidence of impaired appetite, weight in the right hypochondrium, constipation. In the more severe forms of intoxication, toxic hepatitis is observed. Subacute icterus or jaundice and significant abnormal liver function may occur.

Kidney damage is possible, but is less common. Changes are observed by the visual analyzer, the cardiovascular system, the endocrine organs.

Chronic lead poisoning has been observed: Lead poisoning to a slight degree with asthenous-vegetative manifestations, gastrointestinal with hypo- and anaciditis, hypotonia. Mild disturbances in liver detoxification. Clinically and laboratorically there is evidence of pronounced plumbaemia, increased amounts of DALA in blood and urine, reticulocytosis, mild anemia. At this stage, an increased incidence of spontaneous abortions, reduced female reproductive capacity and impaired spermatogenesis in men may occur.

Moderate severe stage - with lead colic, anemia, toxic hepatitis and vegetative polyneuropathy. Laboratory characteristics typical of lead poisoning are significant.

Severe stage - with serious neurological symptoms such as toxic polyneuritis, encephalopathy, lead meningitis, toxic epilepsy, spinal hemiparesis, etc. With good modern industrial technologies and prophylaxis, this stage is not observed. With regard to the early diagnosis and prophylaxis of industrial lead poisoning, preclinical conditions of "lead carrier" have been introduced in Bulgaria - low-grade plumbemia, plumburia, elevated urinary DALA values with possible lead stripes or spots and no complaints from the exposed persons.

Clinical-laboratory tests: Contemporary early diagnosis and prophylaxis of poisoning is based on the determination of the amount of lead in biological substrates - blood, urine, saliva, hair, etc. Lead in blood at values above 400 μg/l or above 2,889 mmol/l for professionally exposed individuals are significant for treatment (as well as the amount of lead in the urine above 0.531 mmol/l).

Other important laboratory findings are urinary DALA elevations, urinary uro- and coproporphyrin increased, increased free erythrocyte porphyrins, decreased hemoglobin and erythrocyte count, reticulocytosis of more than 15 ‰ and stipling erythrocytes of more than 0.5 ‰ in peripheral blood. Only the single analysis is not informative enough. It is also necessary to conduct the so-called provocative test with $CaNa_2EDTA$, after which the amount of lead in the urine reflects its mobilization from the depot.

Differential diagnosis. Chronic intoxications with other heavy metals, anemic syndromes, hepatitis, various types of hereditary and symptomatic porphyria, porphyrinuria, etc.

Treatment. The effect of treatment with $CaNa_2EDTA$ is best - amp. 10% 10 cm^3. Apply intravenously in glucose-salt solutions one ampoule for three consecutive days, followed by three days of rest, a total of 9-10 ampoules per treatment course (other regimens are also applicable).

For oral treatment, Penicillamine (Couprenil) can be used, tabl. 250 and 150mg, 0.5 to 1g daily for 7-10 days.

Pectin and pectin products have a good prophylactic and healing effect at doses of 5-10% pectin 100 g for 15-20 days. Balneotherapy with waters containing hydrogen sulphide is recommended.

Lead carriers need to conduct a *provocative test* with $CaNa_2EDTA$ or Penicillamine, and follow the plumb-aemia and plumburia.

Occupational-medical expertise: In case of mild toxicity, after normalization of all laboratory parameters, subjective and objective changes, after balneotherapy usually the working capacity is restored. It is necessary, after medium-heavy intoxications or recurrent minor, to work towards professions without toxic effects.

Prevention. Contraindications to work with lead are liver, kidney, anemic, hypertonic, ulcer, nervous system, and other diseases. The current prophylactic review is important at 3, 6 and 12 months, depending on the nature of the production and the occu-

pational risk. Women of fertile age are not allowed to work.

3.1.8. MANGANESE - V. PETKOVA

Manganese belongs to the heavy metal group. Hygiene significance has its oxides, manganese sulphate and some organic metal compounds (dithiocarbamates) that are used as pesticides.

Possibilities of manganese poisoning exist in the production and processing of manganese-containing ores (pyrolysite, manganite, housmanite, etc.) in metallurgy, using pyrolysis to produce hydroquinones, potassium permanganate and colored glass. High manganese exposure to ferro-alloys for high quality steel. Poisoning was also observed in arc welders using manganese electrodes. Risk exists also in the production of dry alkaline batteries. The replacement of lead tetraethyl with methylcyclopentadienyl-manganese-tricarbonyl (MMT) in unleaded petrol creates prerequisites for exposure to the population as well.

Routes of intake, biotransformation and distribution in the body. *Manganese resp. the oxides of it enter the airways, the gastrointestinal tract and its organic compounds through the skin. In a short time, it circulates in the blood associated with ß$_1$ globulin, spreading and depositing into the brain, liver, kidneys, intestines, muscles, pancreas. The access of manganese to the brain except through the haematoencephalic barrier occurs directly through the olfactory pathways. The main route to eliminating manganese is through bile and intestines with faeces. Separation through the kidneys is negligible.*

Toxic effect. *The toxicity of manganese is partly related to its involvement in metalprotein complexes of some enzymes controlling the oxidation processes in mitochondriases, and its property also in enzyme inhibition pathways to disturb dopamine metabolism. Classical is the affinity of manganese to basal ganglia - n. caudatus, gl. pallidus, corpus striatum, which determines its neurotoxicity.*

Clinical picture.

Acute poisoning. Acute intoxications are rare, incidental and occur with conjunctivitis and upper respiratory tract irritation, and in more severe cases with manganese pneumonia - fever, cough, dyspnea to acute respiratory failure with tachypnea and cyanosis.

Chronic poisoning. They develop in three stages.

Initial stage. It runs with neurozosomal symptomatology, with predominantly hypotensive forms - headaches, easy fatigue, apathy, mood swings, and vasovagal dysfunction of the vagotonic type. It is possible to have the stimulating effect of manganese on hematopoiesis. Diagnosis of manganism at this stage is difficult because neurological symptoms are extremely unspecific and usually go under the mask of neurosis, hypodepressive states, etc.

Second stage. At this stage, these symptoms become more demonstrative and lasting. Drowsiness, emotional lability, gait uncertainty, speech disorders and muscle tone - precursors to parkinsonism. At the same time, clinical and EMG data for vegetosensorial manganese polyneuropathy can be established.

Third stage. Against the background of the preceding symptoms, the typical manganism symptoms of parkinsonism and amnestic-intellectual disorders are outlined.

Laboratory diagnostics. The determination of manganese in blood and urine is not particularly relevant to diagnosis - these indicators are used as biological exposure indicators. For the early diagnosis of chronic manganese poisoning, psychological tests, as well as a relatively specific electromyographic study, which establishes lower bioelectric activity for the lower limbs, a positive strain of reflex that demonstrates increased muscle tone in earlier stages before Parkinson's syndrome develops.

Differential diagnosis. Diagnostic considerations include cerebrasthenia and depression of another nature, Wilson's disease, Parkinson's disease. Especially responsible is the motivation of manganese parkinsonian syndrome. It should take into account young age, proven occupational exposure and exclusion from cranio-cerebral trauma and encephalitis. In Parkinson's disease the tremor is more rough and almost constant. In acute poisoning a differential diagnosis with bronchopneumonia and poisoning with beryllium, antimony, chromium, cadmium.

Treatment. With curative but also for diagnostic purposes chelation therapy with Ca_2Na_2EDTA - 10% - 10 cm^3 - 1 amp. daily in three consecutive days, with a two-fold repetition of the course at three-day intervals. In cases of severe manganism, antidote therapy has no effect and therapy is extremely symptomatic. Symptomatically is treatment of neurasthenic conditions and parkinsonian syndrome. Modern manganese therapy also includes high doses of vitamin E.

Prognosis and labour-medical expertise. In the early stages of treatment and timely cessation of professional contact with manganese, regression of clinical symptoms can be achieved. Diagnosis of chronic manganese intoxication, irrespective of its stage, is indicative of discontinuation of noxa contact and permanent rehabilitation or disability grouping. Clinical symptoms of parkinsonism may also occur after discontinuation of manganese exposure as an

expression of the late evolution of poisoning.

The long-term professional impact of manganese also leads to a decrease in the body's natural immune-biologic resistance and to frequent inflammatory-infectious diseases of the respiratory organs (pneumonia and bronchitis).

Prevention. Medical prophylaxis includes preliminary medical examinations aimed at preventing contact with manganese in persons with organic and pronounced functional CNS diseases, chronic hepatitis, chronic respiratory diseases, anemia. In periodic reviews, besides mandatory manganese determination in biological media, a neurologist is required.

Healing- prophylactic nutrition recommended: Diet B - acidic and pectin-enriched foods and beverages that have a mild antidote effect. Vitaminoprophylaxis.

3.1.9. MERCURY - ST. ANDONOVA

Mercury is the only liquid metal and evaporates at 0 ° C, with its vapors seven times heavier than air. Mercury exists in an elemental state and then the risk of intoxication is its vapor. Toxic are its compounds in which it is found in first and second valence (mercurocompounds and mercury-compounds) such as cinnabar, (mercury sulfide), mercury oxide (yellow and red), mercuric chloride (sublimate), mercury cyanide, etc. Mercury forms with other metals such as gold, silver, zinc, lead, tin alloys, so-called amalgams. There are also organic compounds of mercury. It is used in the pharmaceutical industry, non-ferrous metallurgy, instrumentation, dentistry, paper industry, electronics, military industry and others.

***Biotransformation**. The pathways of penetration are respiratory, digestive and skin. Mercury is a potent inhibitor of enzymes with affinity for free radicals and, most importantly, sulfhydryl groups. It leads to morphological changes of the endoplasmic reticulum in hepatocyte, reduces the cytochrome P-450 and OMF levels and inactivates a number of enzymes in the proximal renal channels. Elimination is mainly through the kidneys.*

Clinical picture. Acute poisoning occurs when inhalation of mercury vapor or oral intake of its salts (mercuric chloride and cyanide mercury are most toxic). Breathing in the airway is caused by:

- pulmonary syndrome characterized by cough, chest pain, shortness of breath, sometimes toxopulmonite and less frequent pulmonary edema and fever. Complications - pneumothorax, subcutaneous emphysema, progressive pneumofibrosis, bronchiectasis;

- neurological syndrome with headache, paraesthesia, tremor, and polyneuritis at more severe intoxications.

Upon orall ingestion of mercury compounds, syndromes are:

- gastrointestinal syndrome that is manifested for a short period of ingestion with abundant salivation, epigastric pain, abundant vomiting, profuse diarrhea. Ulcer-haemorrhagic stomatitis and gingivitis appear in the oral cavity. Severe acute intoxications result in marked dehydration of the body with hypovolaemia, collapse and shock.

- kidney syndrome - diuresis decreases from a few hours to two days, albuminuria occurs and acute renal failure outset for a short time.

Acute poisoning can also occur through the skin when applying mercury-containing ointments or in contact with organic-mercury compounds. Allergic reactions may also occur.

Chronic intoxication with mercury is characterized by:

- neurological syndrome with astenovegetative symptoms, tremor, abnormal handwriting, tachycardia, hypertension, angina pectoris, etc.

- Symptoms from **the digestive system** such as metallic taste in the mouth, abundant salivation, teasing of the teeth, mercury strip, loss of appetite, diarrhea alternating with constipation.

- kidney syndrome. Inorganic mercury compounds primarily cause tubular lesions with the development of mild functional disorders to severe toxic nephropathies. Organic compounds lead to focal extra- or intracapillary glomerulonephritis with moderate tubular damage.

In case of severe chronic poisoning, anemia is observed. In contact with mercury compounds, chronic dermatitis and eczema are observed.

The diagnosis is based on the history of inhalation of mercury vapors or the intake of mercury compounds, the level of mercury in the blood and urine. Differential diagnosis of poisoning with beryllium, arsenic, cadmium, manganese, ethylene glycol and somatic diseases, occurring with the above mentioned syndromes.

Antidote **treatment** with Dimercaptol (amp; 100 mg/ml), Unithiol (amp. 25 mg/ml), 1 mg per 10 kg strictly intramuscularly! For acute poisoning, the dose is administered one to two days, 4-6 hours, depending on the severity of the intoxication, with decreasing doses over the next few days. Vit C and group B vitamins are applied. Adequate water-salt resuscitation. Gastrointestinal depuration. Organoprotective therapy. In renal failure - dialysis methods. In chronic intoxications, the antidote is administered once for 7 days in controlling the level of mercury in the blood and urine.

3.1.10. NICKEL - J. HADJIEVA

Nickel is a silver-white metal with a characteristic luster, malleable and plastic. It is easily melted, dissolved in dilute nitric acid. It reacts easily with halogens, sulfur, phosphorus, arsenic, antimony and others.

Usage. *Nickel is mainly used to produce alloys with other metals having high mechanical, anticorrosive, magnetic, electrical and thermoelectric properties. It is used in the production of alkaline batteries, for chemical apparatus, catalysts, in the reactive technique and construction of nuclear reactors, etc.*

Biotransformation. *Nickel and its compounds fall into the body mainly via the respiratory tract and nickel carbonyl through the skin. Nickel is found in the liver, spleen, kidneys, and nickel carbonyl - and in the lungs, the brain, the heart, and the cortices of the adrenal glands. It is possible to pass it through the placenta.*

*It is **eliminated** by the kidneys and intestines. Nickel carbonyl is also excreted with the exhaled air.*

Pathogenesis. *The pathogenetic mechanism of the toxic action of nickel has not been fully studied. It is also a supposed essential microelement. The toxicity of the compounds varies considerably depending on their solubility. Nickel has a local irritant effect on the skin and mucous membranes. It has the qualities of an allergen and an enzyme effector-suppressing the enzyme activity (arginase, carboxylase, etc.). His carcinogenic effect is unresolved. His teratogenic effect is discussed.*

Clinical picture. Poisoning with nickel is acute and chronic. Under production conditions, **acute poisoning** is predominantly with nickel carbonyl.

Slightly acute poisoning - resembles that of foundry fume fever. Exfoliation, dry cough, chest depression, chills, increased body temperature to 38-40 ° C, flushing, hyperemic mucus, full sweating, followed by a fall in temperature appear several hours after exposure (1 to 5 hours). It is possible that the fever syndrome will recur after 1-3 or more days.

Severe acute poisoning - resembles the clinical picture of carbon monoxide poisoning and occurs with adynamics, headache, apathy, disorientation, loss of consciousness, clonic-tonic convulsions, delirium, hyperthermia, Cheyne-Stokes` breathing, coma. Injuries from other organs and systems most often result in the development of pneumonia, pulmonary edema, varying liver changes, and the like.

Chronic poisoning. Characterized by chronic irritative changes in the upper respiratory tract. Subatrophic and atrophic rhinitis, pharyngitis, sinusitis, nasal bleeding and perforation of the nasal septum are observed. Nickel and its compounds have a sensitizing effect on the skin and mucous membranes (allergic dermatitis, eczema, bronchial asthma). Nervous system disorders are predominantly atheno-vegetative (headache, easy fatigue, vertigo, arterial hypertension). In some cases, organic nervous system, at hearing and visual analyzers are also observed. Kidney damage due to high exposure to its dissolved compounds.

Nickel and its compounds, especially nickel carbonyl, nickel oxide and sulfide, have a carcinogenic effect especially on the respiratory system (carcinoma of the lungs, nose and paranasal cavities).

Clinical-laboratory tests. Haematological indicators - moderate leucocytosis with lympho- and monocytosis, rarely leucopenia and rapid transient reticulocytosis. Clinical and chemical indicators - mostly hyperenzymemy of ASAT, ALAT, GGTP and others. Increased carboxyhemoglobin due to partial decay of nickel carbonyl with CO release.

The content of nickel in the urine is used as an exposure test (standard values 0.1 - 2.5 μg/l).

Differential diagnosis. In acute poisoning a differential diagnosis with acute poisoning of carbon monoxide, nitrogen oxides, with "metal fever" caused by zinc, copper, manganese and others. In the case of chronic poisoning, professional lesions of the lungs, liver, skin.

Treatment. In acute poisoning a complex of therapeutic procedures (respiratory resuscitation, glucocorticoids, calcium preparations, diuretics) are administered. In indications - infusions of glucose and water-salt solutions. Dimercaprole is used as an antidote. The treatment of chronic poisoning is a symptomatic and general strengthening organism.

Labour expertise. Acute poisoning usually causes temporary disability, and chronic - permanent limitation of working capacity to work in contact with toxic substances.

Prevention. Sanitary-technical prophylaxis and medical surveillance of exposed persons. Do not work in contact with nickel persons with respiratory and nervous system diseases, liver, kidney and skin diseases.

Periodic medical examinations are performed once every 12 months with the participation of a therapist, an otorhinolaryngologist, and at indications-dermatologist.

3.1.11. PHOSPHORUS - T. KUNEVA

- Jaw necrosis
- UAP irritation
- Skin burns
- Liver and kidney damage

Phosphorus is an essential microelement for the human body. Phosphorus does not occur naturally in the free state, but only in the form of compounds, the

largest amount being contained in the mineral apatite. There are two allotropic forms: white (yellow) and red phosphorus.

White phosphorus is highly toxic and chemically very active. It is used for the production of red phosphorus and phosphorus compounds, in explosives, fireworks, smoke bombs, for making semiconductors, is alloyed. Phosphoric acid is used in the production of superphosphate fertilizers. Red phosphorus is many sub- toxic because of its water insolubility. It is used for matchmaking, metallurgy and agriculture.

Prolonged ***professional exposure*** *to phosphorus compounds exposed workers in the production of explosives, matches, phosphorous fertilizers, phosphorus compounds, semiconductors. Unprofessional poisoning can occur with accidental or deliberate absorption of phosphorus compounds.*

Absorption, metabolism, excretion. *Phosphorus compounds penetrate the body mainly through the digestive tract, less frequently through the respiratory system, skin and mucous membranes. In the body, most of them metabolize and are eliminated in a significant amount through the kidneys as salts of phosphoric acid.*

In the liver, phosphorus binds to proteins and disrupts the functions of a number of enzyme systems that regulate oxidation processes in cells of the central nervous system, liver, kidneys, damages metabolism - especially carbohydrates by blocking phosphorylation. Chronic exposure to phosphorus causes sub-epiphyseal bone formation with vascular disorders leading to bone necrosis.

Clinical picture.

Acute poisoning. One to two hours after ingestion of phosphorus compounds, the first symptoms of the digestive tract appear - nausea, vomiting, stomach ache, diarrhea. Phosphorescence of the vomited matters and stools is characteristic. Hepato-renal disorders - hepatomegaly, jaundice, oliguria, uremia, haemorrhagic diathesis develop several days later. Hypocalcaemia may develop, leading to tingling of the extremities, paresis, tetany, trizmus, in the most severe cases - cardiac arrhythmia and coma.

Over acute forms of intoxication are known, ending lethally for several hours and showing a severe jaundice, convulsions, delirium, coma.

Inhalation of dust from phosphorus compounds can cause bronchospasm with cough, tightness in the chest, shortness of breath, dry wheezing. Local tissue necrosis is the cause of haemoptoea. In some cases, there is a chemical pneumonia with respiratory failure undergoing at pulmonary edema and cardiac arrhythmia.

Inhalation of phosphine causes headaches, dizziness, tinnitus, fatigue, weakness, nausea, vomiting, strong thirst, dry cough, tingling in the chest, shortness of breath. Neurological symptoms include ataxia, paraesthesias, tremor, diplopia. Death can become by cardiovascular collapse or respiratory failure.

In **contact** with phosphorus compounds on the skin, severe burns occur, most often II and III degree, with ulcerative defects healing very slowly and hard.

Chronic poisoning. For initial symptoms of chronic poisoning, dyspeptic disorders in the form of gastroenteritis are accepted. Typical are the bleeding with the smell of garlic, nausea, vomiting, abdominal pain, sometimes diarrhea. Gradually, anemia, subicterus, mucosal hemorrhage, toxic hepatitis and nephritis develop.

In long-term exposure, changes in the bone system are most characteristic. Typical are the necroses in the upper and lower jaw. Carious teeth facilitate the action of phosphorus on the jaws - complaints of toothache, followed by inflammation of the gingiva, early shaking and falling of the teeth, periostitis, necrosis of the bone with stinging. There is a gray-yellow or brown stripe on the front teeth. Spontaneous fractures of long bones due to decalcification are rare.

Long-term exposure to phosphorus dust causes chronic rhinitis, laryngitis, bronchitis, sometimes pneumonia, and obstructive pulmonary disease.

Laboratory researches. Acute poisoning has been reported to increase liver enzyme activity, hyperbilirubinaemia, leucopenia, haematuria, proteinuria. After acute intoxication, arterial hypoxemia may develop.

In a functional breathing study, obstruction or mixed type ventilator failure occurs. X-ray shows pneumonia, pulmonary edema, jaw necrosis.

Differential diagnosis is made with acute poisoning with phalloides fungi, in chronic poisoning - with tetrachloromethane and arsenic preparations.

Treatment. There is no specific antidote. Upon oral ingestion - gastric lavage with water and potassium permanganate, it may be with a 2% solution of copper sulphate. Following lavage, saline purification (magnesium or sodium sulphate); 100 ml liquid paraffin; 15 ml of 1% copper sulphate solution is applied sequentially; several times a day sodium bicarbonate. Depending on the haemodynamic parameters, intravenous drip infusion of glucose and water-salt solutions is included: vitamin B_1 and C. Hepatoprotective treatment is performed with 10% 500 ml of levulosis. Antibiotics are included to prevent bacterial infection. Upon developing hypocalcaemia, 10% 10 ml of calcium gluconate is administered intravenously. In the case of skin and eye burns - thoroughly washing with water. In case of severe

burns, the skin is smeared with a 3% solution of copper sulfate to neutralize the action of phosphorus.

Prognosis. In severe acute poisonings, the mortality rate is high. In all cases of acute intoxication, treatment is prolonged. Organic injuries following chronic poisoning (hepatitis, nephritis, bronchitis, bone damage) require discontinuation of contact with toxic substances and may cause a disability group.

Medical prophylaxis. Contraindications to work with phosphorus compounds include tooth and jaw diseases, chronic diseases of bone, UAP, lungs, liver, organic CNS diseases, blood and metabolism diseases.

Workers engaged in the production of white and red phosphorus are subject to periodic medical examinations every 6 months, and those from all other industries using phosphorus, phosphorus compounds, superphosphate fertilizers - once a year. Prophylactic examinations are performed by an internist and a dentist, also by a neurologist, an otorhinolaryngologist and an ophthalmologist. Paraclinic studies include: complete blood count, general urine and calcium urine test; at indications to the radiographs of the bones. It is recommended that diet D of the professional protective nutrition.

3.1.12 SELENIUM - S. PAVLOVA

Acute poisoning

- irritation of upper respiratory tract mucosa and inflammation of the skin
- burning of the skin

Chronic poisoning

- fatigue, lassitude
- gastrointestinal complaints
- smell of garlic of exhaled air and sweat
- dermatitis, paronychia, alopecia
- conjunctivitis

Selenium (Se, atomic weight - 78, 96) *occurs in several allotropic forms, most important of which are red amorphous and gray metallic selenium. Selen is less toxic than its compounds - selenium dioxide (SeO_2), selenium trioxide (SeO_3), hydrogen sulfide H_2Se) and the salts of selenious, selenium-hydrogen and selenic acids.*

Usage. *Selenium and its compounds are used in the glass, ceramic, pharmaceutical and rubber industries, in the production of high-quality alloy steel, plastics, pesticides, etc.*

Professional exposure. *Workers involved in the melting and purifying of copper are exposed to dust from Se and SeO_2, as well as H_2Se, formed by the interaction of acids with selenides (Se compounds with metals). The impact of Se is also found in electronics, ceramics, glass, rubber, and plastics. In agriculture, using sodium selenite (Na_2SeO_3) as a pesticide.*

Pathogenesis. *Selenium is an essential microelement. Its toxic effect is related to its ability to block sulfhydryl groups of many enzymes and sulfur-containing amino acids and is expressed in a disorder of the functional state of the CNS, the liver, etc.*

Absorption, metabolism, excretion. *Selenium enters the body via the respiratory tract, the gastrointestinal tract and the skin. In blood, selenium is mainly found in the plasma associated with α_2 and β_1 - globulin fractions and the free amino acids - cysteine, methionine and leucine. Selenium penetrates into erythrocytes, with about three times as high in serum. The biotransformation of selenium is accomplished by the reduction, methylation and binding processes or direct incorporation into proteins. The tissue distribution of Se shows maximum kidney and liver levels, followed by those in the retina, spleen, lungs, myocardium, skeletal muscles, brain and hair. Selenium is mainly emitted in the urine (30-50%), sweat, faeces, hair and exhaled air.*

Clinical picture.

Acute poisoning is rare. Inhalation of selenium vapors, SeO_2, H_2Se, selenium hexafluoride and selenoxyl chloride causes irritation of the upper respiratory tract and eye lining, dry cough, chest pain and pressure, general weakness, headache, dizziness, unpleasant taste in the mouth, Nausea, vomiting, smell of garlic in the exhaled air, and sometimes convulsions. In some cases, there is pulmonary edema, enlargement and soreness of the liver and spleen. In contact with skin selenium compounds cause slow healing burns, as well as dermatitis.

Chronic poisoning. Respiratory organs are affected - dry cough, rhino-pharyngitis, bronchitis, is observed. Exhaled air and sweat are smell of garlic, and pale pinkish-yellow spots appear on the mucous membranes of the gums and the palate. Typical are injuries of skin and skin - adjuncts - nail disorder, dental enamel changes, reddish skin coloring, hair discoloration and alopecia. Gastrointestinal disorders (nausea, vomiting, metallic taste in the mouth), nervous disorders, as well as pulse delays and lower blood pressure without organic changes in the cardiovascular system have been observed. Chronic effects can also cause conjunctivitis - so-called "pink eyes".

Laboratory researches. Biological markers for Se's exposure are used in blood, urine, hair and nails. It is assumed that the increased content of Se in urine (Seu) is a reliable indicator of selenium absorption in the last 24 hours. Depending on the excretion of (Seu), selenium exposure is classified as follows:

acceptable: at (Seu) <0.1 mg/l; **significant**: with a concentration of (Seu) to 0.5 mg/l and **dangerous**:

at (Seu) above 0.5 mg/l. The amount of Se in blood (Se_{bl}) shows longer exposure. The level of (Se_{bl}) to 0.4 mg/l is not associated with signs of poisoning; with an increase of 0.4 to 0.6 mg/l, symptoms of intoxication are observed.

Haematological parameters - decrease in hemoglobin and hematocrit, decreased erythrocyte count and relative lymphocytosis. Changes in the indicators associated with oxidative-reduction processes are also noted - a low level of reduced glutathione in the blood. Enhanced activity of the enzymes characterizing liver function is established.

Differential diagnosis. In acute poisoning, a differential diagnosis with sulfur compounds and nitro-gases intoxications, and in the case of chronic poisoning with sulfur, arsenic and some heavy metals (cadmium, manganese, chromium). Also with unprofessional diseases of the respiratory system, gastrointestinal tract, skin, etc.

Treatment. In acute poisoning, respiratory resuscitation is administered and, in pulmonary edema, glucocorticoids, hypertonic glucose solution, calcium solutions, etc. In the case of burns caused by SeO_2, it is recommended to use a 10% sodium thiosulphate solution (or ointments).

Chronic Poisoning - Symptomatic treatment of respiratory, gastrointestinal, skin, and other diseases. Vitaminotherapy.

Occupational medical expertise. Loss of working capacity in acute poisoning with Se is temporary. Permanent or temporary work without contact with toxic substances is required in case of residual phenomena of acute poisoning and symptoms of chronic intoxication.

Prevention. Sealing of production processes, ensuring effective ventilation and use of personal protective equipment. Performing preliminary and periodic medical examinations (once a year) of the workers. Particular attention should be paid to gastrointestinal and dermatological complaints, as well as to the detection of the smell of garlic in the exhaled air, which implies high selenium absorption. Protective professional nutrition - a diet rich in protein, high in cysteine and methionine.

3.1.13. TELLURIUM - S. PAVLOVA

- Inflammation of the respiratory tract
- exhaled air with garlic odor
- fatigue, drowsiness
- dryness of the mouth and skin
- dehydration
- skin coloration in blue-black color

Tellurium (Te, atomic weight 127,60). *It is encountered as an impurity in different ores. There are two allotropic forms of Te-silver-white with a metallic luster of a hexagonal romboedron crystal and a brown-black amorphous powder.*

Usage. *Tellurium is used for the production of tellurium alloys as semiconductor materials. Tellurium compounds - tellurides, are used in photography, in the ceramic and glass industry, in vulcanization of rubber, in the process of petroleum processing, in lead and copper production, and in electronics.*

Professional exposure. *In the glass, ceramic and rubber industry, as well as those working in electronics and copper, silver, gold, lead and bismuth cleaning.*

Absorption, metabolism, excretion. *Tellurium and its compounds enter the body through the gastrointestinal tract and the lungs. Very rarely, skin penetration is observed. In the body, tellurium compounds are metabolised to methyl telluride and elemental tellurium, which is less toxic. Tellurium is deposited in the bones (up to 90%) and in the liver. Excretion is by lung, faeces and urine.*

Clinical picture.

Acute intoxications: hydrogen telluride(H_2Te), irritates the mucous membranes, but because of its rapid decomposition it is less poisonous than selenium or arsenic (the lining of the upper respiratory tract is colored in black and green). Other tellurium compounds (tellurium dioxide, tellurium acid, tellurium hexafluoride) cause headache, weakness, dizziness, shortness of breath and pulse rate, nausea, vomiting, kidney pain, haematuria, cystitis, cyanosis, comatose. Typical is the smell of garlic of exhaled air and sweat.

Chronic poisoning: accompanied by nausea, vomiting, metallic taste and dry mouth, depressed state, drowsiness. The air exhaled by the mouth is a smell of garlic - from methyltelluride ($(CH_3)_2Te$), which is easily volatile.

Laboratory tests are non-specific. Haemolysis may occur after exposure to hydrogen telluride. It is necessary to test the urine content of Te, which should not exceed 0.05 mg/l.

Differential diagnosis. With poisoning caused by inorganic compounds of arsenic and selenium, also with sulfur, chromium, manganese.

Treatment. The treatment is symptomatic - pulmopoprotection, respiratory resuscitation, vitaminotherapy. At hemolysis - is transfused. Antidote therapy is not administered.

Prevention. Sealing of technological equipment, ventilation of work rooms and control of concentrations of the toxic substance. Use of personal protective equipment. Conducting periodic medical examinations (once a year). Pregnant women are not allowed to work in contact with tellurium compounds.

3.1.14. THALLIUM - V.PETKOVA

Thallium (Tl) refers to the rare elements. It and its compounds are used in semiconductor engineering, instrumentation, glass staining, ooptical glass production, precious stones and pyrotechnics, as a catalyst in some chemical processes. Its salts are comprised of ultraviolet luminophores. Thallium amalgam is used in low temperature thermometers.

Routes of intake, biotransformation and distribution in the body. *In occupational exposure, thallium enters the body through the airways and through the skin. It is possible accidentally taking the metal through the mouth with contaminated drinking water or food. After an even distribution between erythrocytes and plasma in the first hours of administration, the metal is deposited primarily in the kidneys and the liver. It is excreted in the urine and faeces, but also in the sweat glands and the bronchial secretion.*

Toxic effect. *The toxicity of Tl is related to its chemical affinity, respectively inactivation of such biological antioxidant structures such as riboflavin (Vit. B_2), glutathione (sulfhydryl groups) and selenium, which results in an increase in lipid peroxidation. It blocks the SH groups of the mitochondrial enzymes - SCDH, MAO, etc., mainly damaging the nervous, digestive, cardiovascular and skin attachments.*

Clinical picture:

Acute poisoning. Acute poisoning may develop with accidental ingestion of thallium and compounds. It runs with acute gastroenteritis with a similar cholera clinical picture at severe poisoning and rapid development of polyneuric, psycho-organic pathology and hepatorenal syndrome. Hair dripping is a characteristic feature of both acute and chronic thallium poisoning. The cardiovascular system is compromised - tachycardia, transient hypertension, T-wave decline, acute heart failure occurring in severe cases.

Chronic poisoning. Chronic poisoning resembles those of lead and arsenic. The first symptoms of chronic thallium intoxication are increasing asthenia, anorexia and behavioural abnormalities - aggression or depression. When the intoxication process progresses, the full picture of the poisoning with the typical sensemotirial polyneuropathy distal type ("dying back"). Exactly the lower limbs are affected. Distinctive symptom is the atypical pain ("burning feet" - symptom). In the course of poisoning, cranial nerves may also be affected - visual, oculomotor. The development of the neurological symptomatics is accompanied by complete hair removal, the skin is dry, appearance of white semilunar bands on the nail. Psychic disorders (speech disorders, maniacal or depressive states) and hepatorenal manifestations complement the clinical picture of the severe forms of chronic thallium poisoning.

Laboratory diagnostics. In support of the diagnosis, the characteristic EMG -typical for thallium poisoning axonal "dying back" degeneration. Detection of urinary thallium above 10 µg/l, in nails and especially in hairs, confirms the exposure.

Differential diagnosis. It is made with arsenic, lead, mercury, acrylamide poisoning, porphyria and drug polyneuropathy.

Treatment. Symptomatic. Antidote treatment with chelators is not effective and even aggravates the condition.

Prognosis and labour-medical expertise. Restoration of thallic polyneurites is a slow process requiring prolonged therapy and rehabilitation.

Prevention. In view of the easy penetration of thallium through the respiratory tract and the skin, the use of personal protective equipment is mandatory. Preliminary medical examinations aim to prevent individuals with chronic and often recurrent upper respiratory tract diseases, diseases of the peripheral nervous system, kidney and liver. Periodic preventive examinations are performed once a year with the participation of a neurologist and ORL specialist. Exposure control - determination of thallium in urine, nails and hair. Vitaminoprophylaxis.

3.1.15. VANADIUM - T. KUNEVA

- UAP irritation
- bronchial asthma
- green coloring of the tongue

In nature, vanadium is found in a number of minerals, but the largest amount is contained in the crude oil. The main source of vanadium is vanadium ores and vanadium sulphide extracted in Peru and lead-zinc vanadite from South Africa. In other ores such as vanadinite, rosolite and carnotite, Va's content is low. The ferovanadium slag is also the source of this metal.

The largest amount is contained in the crude oil - vanadium oxide is released when burning liquid fuels.

Vanadium belongs to the group of ultra-micro elements. In small amounts, it is found in the adipose tissue and blood.

Usage. *The most important application of vanadium is for the production of ferro-vanadium, cast iron and alloy steels, superconducting alloys for electrical engineering, missile and nuclear engineering and aerospace industry. Vanadium is composed of inks and typographic paints, alloys for magnets and catalysts.*

Professional exposure. *Workers are exposed in grinding and baking of ores, as well as melting and obtaining vanadium alloys. A major source of air pollution with vanadium oxides is the combustion of black*

oil and diesel. Combustion of coal at the thermal power plant also releases harmful emissions that pollute the air of the environment.

Absorption, metabolism, excretion. *Vanadium compounds penetrate the body through the airways and the digestive tract. Absorption in the lungs is higher for miners and persons working and living in close proximity to liquid fuel deposits. The absorption of vanadium from the gastrointestinal tract is negligible (1-2%). It is excreted in urine and faeces. There is poor bone accumulation.*

Vanadium and its compounds inhibit oxidative phosphorylation in cells, have irritant activity, damage the liver, kidneys, nervous system, lipid metabolism.

Clinical picture

Acute poisoning. At high concentrations of vanadium pentoxide dusts and vapors (even at short exposure - 1 hour), there are severe tears and redness of the conjunctiva, serous and haemorrhagic rhinitis, sore throat, cough, chest pain. Significant exposure may result in bronchitis and pneumonia. Neurological manifestations - headache, dizziness, and renal impairment. Normally, in a timely treatment for 1-2 weeks, a full recovery is achieved.

Long-term exposure to vanadium oxide vapors and dusts is associated with respiratory disturbances: snuffle with epistaxis, laryngitis, chronic bronchitis with dry irritative, often spastic cough. The cough sometimes has profuse expectoration and haemoptoea. There is a greater incidence of pneumonia passing as metal-fume fever (beryllium, cadmium, manganese). Workers with more than 10 years of exposure to vanadium develop diffuse pneumosclerosis. Allergic reactions are observed - bronchospasm, seizures of bronchial asthma, dermatitis and eczema. Skin paleness, papular rash of face and hands, greenish-black tongue staining are detected.

Gastrointestinal disorders include loss of appetite, nausea, vomiting, constipation. It may develop toxic nephropathy.

On the nervous system, headaches, dizziness, slight tremor of the fingers, mental disorders are observed. Inflammatory changes in the optic nerve and the retina, severe conjunctivitis are also described.

Metabolic disorders include involve in cysteine and cholesterol biosynthesis, suppression and stimulation of phospholipid synthesis. High concentrations cause serotonin inhibition.

Laboratory researches. At the beginning of chronic vanadium exposure, an increase in erythrocytes and hemoglobin was observed, and then a rapid decrease. Plasma cholesterol is decreasing. Skin-allergy tests to prove sensitization to vanadium compounds.

Differential diagnosis with dimethyl sulfate poisoning, with metal pneumoniae from beryllium, cadmium, manganese, zinc, etc.

The treatment of vanadium poisoning is symptomatic. Chronic bronchitis and pneumonia are treated with antibiotics, broncholitics, high doses of vitamin C. In polyeneuritic events, high doses of B vitamins are administered, and physiotherapeutic procedures.

There are single messages to use Unithiol and Dimercaprol as antidotes.

The prognosis is good for discontinuing contact and treatment.

Labour expertise. In case of intoxication - temporary removal from work. In the development of toxic pneumosclerosis after acute poisoning and in chronic bronchitis without respiratory failure - termination of contact with toxic substances. With frequent recurrent bronchitis and bronchial asthma attacks, patients have permanently reduced working capacity.

Prevention. Basic sanitary-technical measures include mechanization, automation and sealing of production processes combined with ventilation.

Preliminary and periodic medical examinations - contraindications to work with vanadium include atrophic rhinopharyngitis, bronchitis, bronchial asthma, pneumofibrosis, chronic liver, kidney, nervous system, anterior eye, optic and retina diseases, chronic diseases of the skin.

Periodic medical examinations are performed once a year by a therapist and a full blood count is required.

3.1.16. ZINK - J. HADJIEVA

Zinc is a greyish-silver metal of medium hardness. It is easily oxidized. It dissolves in mineral acids, strong bases and ammonia.

Usage. *Zinc is widely used in mechanical engineering (for electrolytic metal coating and corrosion protection), metallurgy (for the production of alloys), chemical, pharmaceutical and textile industries, etc.*

Biotransformation. *Zinc enters the body through the respiratory and digestive tract. Skin and mucous membranes are of secondary importance. The zinc in the gastrointestinal tract is absorbed into the small intestine and is slowly distributed throughout the body. About 35% of the inhaled zinc is permanently bound to blood serum in the form of a metalloprotein, and the rest is labile to a globulin complex. Zinc is found in the pancreas, liver, kidneys, prostate, thyroid gland, bones, muscles, brain, and so on. It is eliminated with faeces, urine and, in lesser quantities, with sweat, hair and nails.*

Pathogenesis. *The biological significance of zinc*

is determined by its involvement in a number of enzyme systems. Its increased intake in the body causes a decrease in serum sulfhydryl groups content, disturbs the electron transport in the mitochondrial respiratory chain, inhibits cytochrome oxidase activity, disrupts secretory function of the pancreas.

The pathogenesis of zinc-induced "metal fever" is not fully elucidated. Its pyrogenic effect is associated with the catalytic activity of the finely dispersed metal particles that denature cellular proteins. An immune pathogenetic mechanism is also allowed.

Clinical picture.

Acute intoxications occur in two forms:

Inhaled form ("zinc fever") - characterized after a latency period of 4-6 hours, mainly with pulmonary syndrome (throat irritation, cough, chest depression), pyrexia syndrome (hard attack from fever and hyperthermia to 40 ° C, which persists for several hours, and then drastically lowering the temperature with abundant sweating) and general toxic syndrome (malaise, fatigue, muscle aches, collapse, etc.). Pulmonary edema may develop (less commonly).

Gastrointestinal - Sweet metallic taste in the mouth, nausea, vomiting, burning and sore throat and epigastric pain, dyspeptic manifestations, collapsing and dehydration phenomena. Consequently, renal syndrome (acute renal failure phenomena) has developed.

Chronic poisoning. The most common manifestations of chronic zinc poisoning are respiratory symptoms (chronic upper respiratory catarrh, chronic rhinitis, perforation of the nasal septum, chronic bronchitis, pneumonia, and, in some cases, pneumosclerosis), the digestive system (chronic gastritis and colitis, ulcerative stomach and duodenal disease), cardiovascular system (development of early atherosclerosis, rhythm disorders), blood and blood-forming system (hypochromic anemia), nervous system (neurasthenia), skin (toxic dermatitis, skin ulcerations in the form of "bird eyes"). The effect of zinc on the immune-biologic reactivity of the organism and the reproduction processes is known. Experimentally, its carcinogenic and mutagenic effects have been proven.

Clinical-laboratory tests. Hematological parameters - reduced hemoglobin and erythrocyte counts, aniso- and poikilocytosis, polychromasia, leukocytosis, eosinophilia. It is possible to detect hyperglycaemia and glucosuria.

Zinc content in blood serum is used in the assessment of exposure of persons in occupational contact with zinc - normal values of 12.0-24.0 μmol/l.

Differential diagnosis. Acute poisoning with zinc requires a differential diagnosis of "metal fever" caused by other metals (copper, arsenic, cadmium, nickel, tin, manganese, etc.), acute respiratory diseases, corrosive and food poisoning. Chronic poisoning should exclude unprofessional diseases of the upper respiratory tract, gastrointestinal tract, skin, etc.

Treatment. Treatment of acute poisoning - inhaled form, includes antipyretics, antidotes ($CaNa_2EDTA$ or dimercaprole), respiratory resuscitation and symptomatic remedies. In the gastrointestinal form, cleansing, gastric lavage, physiological serum and polyelectrolyte solutions, anticollapse agents, etc. Treatment of chronic poisoning is symptomatic.

Labour expertise. In acute poisoning, loss of working capacity is most often temporary. Repeated zinc fever and symptoms of chronic intoxication require temporary or permanent labour without contact with toxic substances.

Prevention. Sanitary and technical prophylaxis (sealing of manufacturing processes, protection of the respiratory tract and skin through the use of individual protective devices, etc.). Medical prophylaxis - are not allowed to work in contact with zinc and its compounds, persons with diseases of the respiratory, digestive, nervous and cardiovascular systems, diseases of the liver, kidneys and skin. Periodic medical examinations are performed once every 12 months with the participation of a therapist, an otorhinolaryngologist, and with a dermatologist.

3.1.17. TIN - J. HADJIEVA

Tin is an supposed essential microelement. Low inactivity is characterized by its inorganic compounds, while its organic compounds are highly toxic.

Usage. *Tin and its compounds are used in the textile industry, galvanoplasty, in glass and enamel production, in the production of alloys, plastic stabilizers, pesticides, etc. In pure form the tin is used for welding, tinning and the like.*

Biotransformation. *Organic compounds of tin come into the body via the respiratory, digestive and skin pathways. The tin lasts for a short time in the blood. It is distributed quickly in the body, with the highest levels found in the lungs, kidneys, liver, spleen, bones. Some of the organic compounds of tin can be found in the brain and muscles.*

The metabolism of tin organic compounds occurs mainly in the liver with mixed function oxidases and by processes of dealkylation and dearylation. Tin and its compounds are ***eliminated*** *by urine and faeces.*

Pathogenesis. *Organic tin compounds have a specific and general-toxic effect. Depending on the chemical characteristics of the compound, the hepatobiliary,*

the excretory and the central nervous systems are predominantly affected. Inorganic tin compounds have a damaging effect on the lungs, skin and mucous membranes. There is evidence of the carcinogenic effect of tin.

Clinical picture.

Acute inhalation poisoning is characterized primarily by irritant changes in the upper respiratory tract and the development of "metal fever". Oral ingestion of inorganic tin compounds causes the development of gastroenterocolitis. Its organic compounds can provoke cerebral symptoms (headaches, convulsions, photophobia, disturbed consciousness, bulbar paralysis, cerebral and spinal cord edema, coma). It is possible to develop toxic hepatitis and toxic nephropathy.

Chronic poisoning is mainly characterized by pulmonary symptoms (cough, tightness in the chest, difficulty in breathing). Chronic bronchitis, early emphysema and moderate respiratory failure are observed. Prolonged exposure to tin can lead to the development of pneumoconiosis (so-called stannosa). Lung carcinoma cases have been reported.

Clinical-laboratory tests. As an exposition test, urine tin (normal values of 0.2-11 μg/l) are used.

Hematological and clinical-chemical studies contribute to the diagnosis.

Differential diagnosis. In acute inhalation poisoning, a differential diagnosis with poisoning causing "metal fever" from other toxic substances. Chronic poisoning with tin is differentiated from diseases of the respiratory system and non-professional genesis.

Treatment. It is imperative to discontinue the contact with the toxic substance in a timely manner and to eliminate it from the body (thorough cleansing of the skin, release of the digestive tract from the poison, etc.). Cerebrovascular (hypertensive glucose solution - 20-40%, pyramem, B and C vitamins, glucocorticoids, mannitol, etc.), hepatoprotective, nephroprotective agents and appropriate symptomatic therapy are used.

Labour expertise. In acute poisoning, workability is temporarily lost. Low chronic poisoning requires temporary rehabilitation without contact with toxic substances. Cases of pronounced, persistent pulmonary and cerebral pathology require rehabilitation.

Prevention. Sanitary and technical prophylaxis - sealing of production processes, use of individual protective devices in order to protect the respiratory tract and the skin.

Medical prophylaxis. Carry out a preliminary medical examination - do not work in contact with tin and its compounds, persons with respiratory and central nervous system diseases, liver, kidney and skin diseases. Periodic medical examinations are performed once every 12 months with the participation of a therapist, neurologist, otorhinolaryngologist, and at indications - dermatologist.

3.1.18. COPPER - T. KUNEVA

- Copper fever;
- allergic dermatitis, green-yellow or green-black skin coloring;
- rhinitis, pharyngitis, gastritis, "copper colic";
- local - corneal ulceration of the eyes, the skin, the nasal septum.

Copper is an essential oligoelement - plays an important role in blood formation, in the activity of a number of enzymes, in the function of some endocrine glands.

It is widely used in metallurgy and machine-building (extraction of copper and copper alloys - brass, bronze); in electronics and radio engineering (wires, details); in the chemical industry (vacuum cleaners, refrigerators, heat exchangers); in the manufacture of mineral paints, artificial silk, microfertilizers, as a catalyst in the tanning industry.

***Occupational exposures** with copper aerosols are exposed to workers involved in the processing of ores containing this metal as well as to the melting and processing of copper and its alloys. Parts of metallic copper or copper alloys may fall into the eyes.*

Relatively lower is the risk for agricultural workers inhalating powders of copper sulphate and carbonate.

***Absorption, metabolism, excretion.** The main ways of penetration of copper in the body are respiratory (vapours and dust) and gastrointestinal (when swallowed). It is resorbed through the intestinal mucosa, circulates in the blood in the form of copper albumins, is mainly deposited in the liver and, to a lesser extent, in the kidneys and pancreas. It is mainly eliminated in urine. Accidental single ingestion of soluble copper salts is generally considered to be harmless to its resorption, as they cause severe vomiting.*

Clinical picture.

Acute poisoning. Inhalation of powders and aerosols of copper salts may cause nasal blockage, mucosal ulceration, and perforation of the nasal septum. A few hours of working with molten metal can cause "copper fever". Prodromal phenomena include burning and dry throat, sweet taste in the mouth, general weakness and headache. Often, the first symptoms are dry paroxysmal cough, sore throat pain, shortness of breath, conjunctival flushing. Four to five hours later a febricity occurred with a sharp rise in temperature to 39°- 40°C, pains throughout the musculature, most pronounced in the lower

limbs. The fever lasted for 1-2 hours, followed by a lytic drop in temperature with abundant sweating. Blood is found in neutrophilic leukocytosis, followed by lymphocytosis, transient hyperglycemia; in urine - leukocytes and cylinders. In the next few days, the patients complain of general weakness and fatigue. Sometimes copper fever attacks recur in a few days. Relatively rarely during an attack the functions of the gastrointestinal tract and the liver are impaired. In severe cases, catarrh of the upper respiratory tract may progress into spastic bronchitis or focal pneumonia. In acute oral intoxications, a gastrointestinal syndrome occurs with nausea, multiple vomiting of greenish-colored stomach contents, abundant salivation, colic-like abdominal pain, diarrheal stools. Hematologic (acute intravascular haemolysis, hemoglobinuria, jaundice, secondary anemia) and hepato-renal syndromes are present in severe poisoning.

Chronic poisoning.

The question of chronic copper poisoning is debatable. Chronical toxic effects of copper have been demonstrated only in people with abnormal autosomal recessive genes inducing the development of hepato-lenticular degeneration (Wilson disease - dysfunction and structural changes in the liver, CNS, kidney, bone and eye). The primary disorder here is the reduced amount with ceruloplasmin - the copper binding protein serum. After ingestion in the intestine, copper becomes unstable with serum albumin instead permanent of ceruloplasmin, and this bond quickly breaks down and copper is deposited in a number of organs. Miners can also increase body absorption when 1-2% of copper ore dust is present in the air in the work environment. However, convincing evidence of primary chronic copper poisoning (observed in patients with Wilson's disease) has not been found in occupational exposure.

A higher percentage of rhinitis, pharyngitis, gastritis has been found in people in contact with copper powder. Gastrointestinal disorders include loss of appetite, nausea, stomach pain ("copper colic"). The liver and kidney functions are relatively rarely affected. Radiologically, copper pulp miners have been found to have a strong lung markings, thickening of the lung roots, basal emphysema. Workers spraying annually vines of copper sulphate can experience pulmonary damage, known as the "lungs of sprayers of the vineyards".

Frequent functional disorders of the nervous system occurring occasionally with vasomotor disorders.

The skin areas in contact with copper preparations are colored greenish-yellow or greenish-black, and a greenish band appears on the gums. Copper and its salts have an irritating effect on the skin and eyes - they can cause corneal, skin, nose septum ulcerations, abscess development, loss of vision. Very common cases of allergic dermatitis occur in contact with copper compounds.

Laboratory researches. Increased hemoglobin, erythrocytes and copper in the blood and bone are detected.

Differential diagnosis of acute poisoning is made with zinc and other metal fevers and colds of the UAP, and in chronic poisoning with gastrointestinal tract and UAP diseases by non-professional factors.

The prognosis is favorable - there are no lasting consequences.

Treatment. Antidote - in acute oral toxicity with copper compounds (copper sulfate) as follows:

1. $CaNa_2$ EDTA - 10% 10 ml/1 g/day intravenous ampoules with 500 ml phys. saline or glucose for three consecutive days with control of urine excretion.

2. D-Penicillamine (Cuprenil) 0.250 g tablets; a daily dose of 2 g, a maximum daily dose of 3 g. Treatment course - 5 days.

3. Dimercaprol (BAL) ampoules 5% 5 ml intramuscularly once a day. Combined with gastric lavage with activated charcoal, infusion resuscitation with glucose and saline solutions, vit. C in large doses, glucocorticoids (against hemolysis).

In copper fever - physical and mental rest, infusions of glucose solutions 20% and 40% with ascorbic acid; antipyretics, alkaline inhalations.

Perforation of the nasal septum - termination of the contact for several months. Local cleaning with subsequent treatment with antibiotic or corticosteroid ointments.

In the eyes - abundant flushing with water, applying a local anesthetic and dropping EDTA collyium at 5 minutes in the first few hours.

In skin ulcerations - mechanical treatment and compress with sodium thiosulphate 5%; corticosteroid ointments.

Labour expertise. In acute poisoning - temporary disability. In case of residual symptoms after acute poisoning and pronounced phenomena of chronic injuries - termination of contact with toxic substances.

Medical prophylaxis. Contraindications to working with copper compounds are chronic diseases of the heart and lungs, the liver, the anterior ocular segment and the skin. Periodic medical examinations are performed once a year by a therapist; At indications-dermatologist and otoneurologist are involved. Laboratory tests include a full blood count.

3.1.19. ALUMINUM - S. PAVLOVA

Aluminum (Al) is a light, silver-white metal insoluble in water. It dissolves in basic and in most acids. Aluminum powder in a mixture with air is explosive.

Usage. *Aluminum is used in the production of various metal products; for the production of alloys used in aircraft and automotive industry; for pyrotechnic aluminum powders and paints. The compounds of Al (oxides, chlorides, formiates) are used in the ceramic, perfume and paper industry.*

Biotransformation. *Aluminum enters the body mainly via the respiratory tract and through the gastrointestinal tract (with food, beverages, water). Simultaneous administration of citric acid can significantly increase its absorption. From blood Al quickly passes into the tissues and is distributed in all organs but in larger amounts in the lungs, bones and muscles. Al concentrations in the brain have been found to increase with age. Elimination is mainly done with urine and faeces. Patients with chronic renal insufficiency represent a risk group due to reduced Al excretion, which results in its accumulation in the tissues. An additional way of excretion can be sweating.*

Pathogenesis. *The mechanism of Al's toxic action is not sufficiently clear. It is assumed that CNS and bones are primarily affected by increased exposure. Aluminum can form complexes with amino acids, nucleotides, organic acids, phosphates, and its toxicokinetics depend on the nature of these complexes.*

Clinical picture.

Acute poisoning is characterized by pronounced increasing dyspnea, severely restricted respiratory movements, cyanosis, tachycardia, decreased blood pressure, electrocardiographic changes and transient atrial fibrillation. Heavier cases of poisoning occur with rapid development of diffuse lung fibrosis, abruptly pronounced peripheral emphysema, spontaneous pneumothorax formation, often with mortal outcome.

Chronic poisoning causes pneumoconiosis (so-called "aluminosis"). There is severe tiredness, shortness of breath, cough, weight loss, dry and wet lung's crepitations. Occasionally, anorexia, nausea, stomach's, and muscles pains of the whole body. Secondarily, some cardiovascular disorders may occur as a result of pulmonary fibrosis and impaired pulmonary function. Possible development of severe encephalopathy with uncoordinated movements, tremor, gradual loss of memory, abnormal thinking and depression. Common skin lesions are dermatoses, telangiectasia and eczema. Conjunctivitis and irritation of mucous membranes of the upper respiratory tract have also been reported.

Clinical-laboratory tests. As a better biomarker of exposure, the determination of Al in urine (biological limit value - 200 μmol/l) is used.

Haematological parameters: Reduced levels of hemoglobin, lymphocytosis and eosinophilia are observed. In persons with insufficient renal function, aluminum may cause microcytosis and hypochromic anemia.

Differential diagnosis. Poisoning with Al and its compounds should be distinguished from unprofessional diseases of the respiratory, cardiovascular, digestive, skin and mucosal diseases.

Differential diagnosis, as well as Alzheimer's disease, suggests that the accumulation of Al in brain tissue is one of the causes of the disease, as well as by persons with chronic renal failure, undergoing prolonged hemodialysis, at them observed hyperaluminaemia and accumulation of Al in tissues.

Treatment. In acute poisonings immediate lead away of the worker is required, respiratory resuscitation, and at severe pulmonary edema - glucocorticoids, calcium preparations, hypertonic glucose solution, etc.

Chronic poisoning requires special treatment of respiratory, gastrointestinal, skin, and other disorders. Antidote therapy with $CaNa_2$ + EDTA is not recommended due to lack of effect.

Labour expertise. Acute poisoning with aluminum is often associated with temporary loss of working capacity. Chronic intoxications impose temporary or permanent labour readjustment under specific work conditions.

Prevention. Sanitary-technical prophylaxis. Strict adherence to the requirements for safe operation (ensuring effective ventilation, mandatory use of personal protective equipment - clothing, glasses, protective creams, ongoing control of aluminum concentrations and its compounds in the air in the working environment, etc.) is required.

Medical prophylaxis. Performing preliminary and periodic medical examinations (once a year). Particular attention should be paid to the condition of the lungs, skin and eyes.

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3.2 ORGANIC SOLVENTS

Solvents - water-soluble (water-based) or organic (hydrocarbon-based) used in manufacturing are predominantly organic substances. In the largest quantities, they are used as intermediates in organic synthesis in the chemical industry, but the greater the number of workers using them as a means of cleaning, degreasing, extraction, dissolution.

Hundreds of organic substances are used to make over 30,000 different types of solvent. These organic substances have their physical, chemical and toxicological characteristics and peculiarity that determine their behaviour, health risk, and make it possible to classify them into separate subgroups, but their classification is mainly their chemical formulation.

The solubility of lipids (lipid solvents) is one of the most important characteristics determining both their effectiveness as production solvents and their health effects. The strength of the narcotic's effect as well as the degreasing agent is proportional to the degree of their lipid solubility (different for the individual organic substances and compounds).

Their penetration through the skin is also related to lipid solubility but also to water solubility - e.g. it is higher for dimethylformamide or glycol esters which are lipid solvents but are also water soluble. The degree of penetration is also related to their volatility (evaporation at lower temperatures) - **Tabl. 3.**

Tab. 3. Brief description of organic solvents

Chemical group	Representatives	Toxic effects	Acute poisoning	Chronic poisoning	Exposure tests
Aliphatic hydrocarbons	Alkenes (Paraffin) methane, ethane, propane, butane, hexane) Olefins (ethylene, propylene, amylene, butylene) Naphthenes (cycloparaffins) (cyclopropane, cyclohexane)	narcotic neurotoxic	rhinopharyngitis nausea, vomiting headache, dizziness drowsiness, fatigue impaired balance disorientation depression impaired consciousness	dermatitis, dry and erythematous skin headache, emotional lability impaired concentration and memory cortical atrophy peripheral neuropathy anemia	EEG - diffuse slow waves psych. tests EMG - slow speed of conduction in peripheral nerves Er ↓ RE ↑
Aromatic hydrocarbons	benzene (benzol) toluene xylene ethylbenzene styrene	irritative neurotoxic myelotoxic	rhinopharyngitis neurasthenic manifestations vasomotor excitement convulsions coma hypotension cardiac arrhythmia	dermatitis distal neuropathy behavioral and cognitive disorders cortical atrophy dementia encephalopathy	Le ↓↓ Thr Er ↓ Hb ↓ Phenol in urine EEG - diffuse slow waves psychological tests CT
Petroleum distillates	gasoline	narcotic neurotoxic	rhinopharyngitis neurological disorders in aspiration - Pneumonia	conjunctivitis dermatitis neurasthenic syndrome anemia	EEG - diffuse slow waves Psycho. tests Er↓ RE↑ Le↓ Ly↑
Amino derivatives of fatty hydrocarbons	ethylenimine ethylenediamine cyclohexylamine	irritants allergens	conjunctivitis corneal edema rhinopharyngitis	allergic contact dermatitis spastic bronchitis asthma	FID AST
Chlorinated hydrocarbons	trichloroethylene perchlorethylene methylenchloride chloroform	narcotic neurotoxic hepatotoxic	rhinopharyngitis neurobehavioral disorders: headache, dizziness drowsiness, fatigue impaired balance disorientation, depression, impaired consciousness jaundice, hepatitis nephropathy	dermatitis impaired of concentration and short-term memory lesion in cerebral nerve cortical atrophy	EEG psychological tests CT

Nitro derivatives of aromatic hydrocarbons	nitrobenzene nitrotoluene trinitrotoluen nitrophenol trinitrophenol (picric acid)	irritative narcotic methemoglobin formers	cyanosis, shortness of breath, arrhythmia, insomnia, agitation, ataxia seizures disorientation impaired consciousness visual disturbances anemia, jaundice hepatitis skin rashes cataract	conjunctivitis dermatitis cerebrasthenia hepatitis nephropathy hyperthyroidism anemia cataract hepatitis	anoxia metHb ↑ Hb ↓ Er↓, cells Heinz urine: albumin ↑ Er nitrophenol> 0,06 mmol/l aminophenol> 0,09 mmol/l
Amino derivatives of aromatic hydrocarbons	aniline diphenylamine p-phenylenediamine (Ursol) α- and β-naphthylamine	methemoglobin formers allergenic carcinogenic	conjunctivitis acrocyanosis, asthenia headache dizziness tottering step convulsions coma hepatitis	conjunctivitis anemia hepatitis vestibulopathy ataxia haemorrhagic cystitis bladder cancer hyperkeratosis, eczema acne vulgaris bronchial asthma	Hb Er ↓ ↓, celles Heinz, and poikilo- and anirzocytosis- MetHb ↑ urina- alb., Er, urobilinogen ↑ ↑
Alcohols	ethyl methyl isopropyl	neurotoxic hepatotoxic	rhinopharyngitis cyanosis, shortness of breath, headache, inebriation, tremors, neuropathies	pharyngitis dermatitis Neuropathia n. optici Parkinson's syndrome hypothalamic syndrome	EEG psychological testing
Glycols	ethylenglycol diethylene glycol propylene glycol	narcotic nephropathy	Headache, dizziness, nausea, vomiting asthenia coordination disorders depression	conjunctivitis pharyngitis nephropathy dermatitis	proteinuria
Phenols	phenol cresol catechol pyrogallol hydroquinone	irritative vasodilatatory necrotic (liver, kidney skin) neurotoxic	rhinopharyngitis hypothermia myocardial ischemia hepatitis nephropathy skin burns neurasthenic manifestations	contact dermatitis gastrointestinal disorders (ptializam, diarrhea, anorexia) cerebrasthenic syndrome vestibulopathy distal neuropathy behavioral and cognitive disorders cortical atrophy, dementia	HB Er ↓ ↓, poikilo- and anizocytosis hemolysis ASAT ALAT ↑ ↑ AP LDH ↑ ↑ proteinuria Phenol in urine, EEG EMG psych. tests
Organic oxides Organic peroxides	ethylene oxide and propylene oxide izopropylbenzol hydroperoxide	neurotoxic, irritant corrosive	headache, dizziness, ataxic step, speech disorder acrocyanosis	tremor polyneuropathy acrocyanosis burning corneas dermatitis, eczema	Tremorogram EMG
Esters	methyl acetate ethyl acetate propyl acetate butyl acetate	irritative allergenic neurotoxic	redness, burns to blindness neuropathy n. optici rhinopharyngitis headache, dizziness, drowsiness palpitations asthma	conjunctivitis rhynopharyngolaringitis dermatitis	EEG psychological tests
Ethers	ethyl-, diethyl-, methyl-dimethyl-, vinyl ether dioxane glycol ethers	irritative narcotic neurotoxic corrosive exp-in/in reproduction	rhinopharyngitis irritability, drowsiness loss of consciousness hepatitis nephropathy anemia, hemolysis encephalopathy	dermatitis headache, dizziness	Er↓ pancytopeny

Organic acids	Formic acetic	corrosive irritants	conjunctivitis rhynopharyngolaringitis	dermatitis, eczema locally-burns	
Aldehydes	formaldehyde acetaldehyde glutaraldehyde dimethylformamide	narcotic irritative allergenic	conjunctivitis, rhinopharyngitis excitation, tremors, visual disturbances paralysis of facial nerve nystagmus ataxia	dermatitis asthma	
Ketones	acetone methyl ethyl ketone cyclohexanone	narcotic irritative	conjunctivitis, rhinopharyngitis salivation, dizziness, headache, flushing impaired consciousness	cathars of UAP anemia dermatitis	Hb↓
Chlorfluoro-karbonates	Chlorfluoro-carbonates (i.e. brand freons - CFCs) trichlorofluoro-methane (F- 11) dichlorodifluoro-methane (F- 12) trichlortrifluormetan (F- 113)	narcotic neurotoxic cardiotoxic	rhinopharyngitis headache, dizziness drowsiness, fatigue impaired balance disorientation, depression, impaired consciousness cardiopathies-rhythm and conduction disorders	dermatitis	EKG EEG

3.2.1. BENZOL - J. HADJIEVA

Pure benzene is a colorless liquid with a pleasant smell, well soluble in organic solvents. Its vapors form with air explosive mixtures at certain volumetric ratios.

Usage. *Benzene is widely used as a solvent for fats, varnishes, paints and more. It is a starting product for the production of a large number of compounds in the chemical industry for the production of styrene, phenol, explosives, detergents, medicines.*

Biotransformation. *Benzol penetrates the body through the respiratory and digestive tract. It is also possible to get it through the skin. In single exposure, it is mainly deposited in the CNS, the adrenal gland, liver and blood, and in prolonged exposure to bones, lungs, kidneys, pancreas and adipose tissue. It is metabolised mainly in the liver with the involvement of the liver microsomal enzyme system. Metabolites of the benzene are phenol, pyrocatechin, muconic acid, and hydroquinone. Elimination is by exhaled air and urine.*

Pathogenesis. *The single effect of high concentrations of benzene causes damage to nerve structures with pronounced narcotic effects. Long-term exposure to low concentrations is dominated by the myelotoxic effect of benzene.*

Clinical picture.

Acute poisoning is rare, mainly in emergency situations. Three degrees of acute poisoning are distinguished:

- mildly - characterized by transient headaches, vertigo, euphoria, ataxia that resolve after a few hours.

- severe degree - psychomotor agitation, somnolence, coma.

- a fast-acting form - there is a rapid loss of consciousness followed by a lethal outcome as a result of paralysis of the respiratory and vasomotor center.

After acute poisoning with benzene, residual phenomena such as headache, vertigo, nystagmus, spastic paresis, epileptiform seizures can be identified. It affects the cardiovascular system - tachycardia, extrasystolic arrhythmia, arterial hypotension. Irritative lesions of the mucous membranes - conjunctivitis, can epistaxis, catarrh of the respiratory tract to pulmonary edema.

Chronic poisoning. Observed with prolonged exposure usually from 3 to 5 years. The main deleterious effects are related to the myelotoxic effect of benzene. The leukopoetic, megakaryocytic and erythroblastic functions of the bone marrow are suppressed. The immunotoxic, neurotoxic and, in some cases, carcinogenic effects of benzene occur. Three degrees of chronic benzol poisoning are distinguished:

- mild degree - vascular symptoms, moderate leucopenia, anemia, initial manifestations of haemorrhagic diathesis are observed. Changes are usually reversible with a timely discontinuation of exposure. In isolated cases, bone marrow changes are progressing. Spontaneous remissions may also occur with persistent exposure.

- moderate degree - more pronounced leucopenia, sometimes thrombocytopenia, deepening of hypochromic anemia and haemorrhagic diathesis, intensification of vegetative-vascular manifestations.

- severely - deepening of bone marrow hypo-

plasia to pancytopenia. There is a tendency to infections and septic conditions.

Under conditions of chronic benzole exposure, acute and chronic leukosis, erythroblastosis, can be diagnosed. They may also develop after the exposure of the toxic substance.

Skin damage occurs in the form of contact dermatitis and eczema.

Clinical-laboratory tests. Leading laboratory tests are the haematological deviations. These are related to leukopoetic, megakaryocytic, and erythroblastic bone marrow lesions. Reduced hemoglobin, decreased red blood cell counts, aniso- and poikilocytosis, increased number of basophil stippling erythrocytes, reticulocytosis, thrombocytopenia, leukocytosis and changes in leukocyte formulations (myelocytes, metameliocytes) or leucopenia, changes in leukocyte quality, chromosomal aberrations in bone marrow and peripheral blood cells.

The contents of the vit. "C" in urine is reduced. Immunological parameters (Ig A and Ig G) show a decrease and the IgM content slightly increased. Determination of urinary muconic acid and phenol is used as exposure tests.

Differential diagnosis. Acute intoxications require a differential diagnosis with alcohol poisoning, benzine poisoning, etc., and chronic - with hemorrhagic fever, systemic blood disorders, septic conditions, etc.

Treatment. In the case of acute poisoning, it is imperative to immediately break the contact with the toxic substance (removal from the gassed environment, when benzene enters the gastrointestinal tract - washing the stomach with water and medicinal charcoal, eye flushing). Antihypoxic and antiarrhythmic preparations, analeptics, nootropic drugs, breathing stimulants, oxygen therapy, and at apnea - intubation and command breathing are used.

The treatment of chronic benzene poisoning is symptomatic. Data on impairment of bone marrow function is a discontinuation of exposure. There are tranquilizers, vitamin therapy (vitamin C and group B vitamins), corticosteroids, anabolic preparations, and others.

Labour expertise. In acute poisoning, workability is most often temporarily lost. Chronic poisoning, depending on the severity of the disability, requires temporary re-employment of 3 to 6 months (with a slight degree of poisoning) and long-term rehabilitation (medium and severe). In many cases, it is necessary to identify a disability group.

Prevention. The high professional risk of prolonged exposure to benzene (carcinogen - group A1) requires that it be placed under special control and, if possible, replacing benzene with another less toxic substance. It is imperative to strictly observe all rules for safe operation, to conduct ongoing monitoring of the benzene content in the air of the working environment and to comply with the MACs.

Medical prophylaxis. Do not work in contact with benzene, people with diseases of the nervous system, blood and haematopoietic apparatus, skin diseases, etc. Periodic medical examinations are performed once every six months with the participation of a therapist, a neurologist, and at indications of a dermatologist and obstetric gynecologist.

Protective professional nutrition - diet "D" is recommended.

3.2.2. ALCOHOLS - R. STEFANOVA

The group of alcohols includes a large number of compounds. They are monoatomic and multiatomic, primary, secondary and tertiary, saturated and unsaturated. Alcohols are widely used as organic solvents and are also used in organic synthesis - the production of plastics, artificial rubber, paints, varnishes, cleaning agents, cosmetics, alcocholic drinks, etc.

Methyl alcohol. *Methyl alcohol (methanol) is a colorless, easy-moving liquid with a specific odor. It is dissolved in water, ethanol, benzene, acetone and other organic solvents. Its vapors form explosive mixtures with air. Oxidation forms formaldehyde, formic acid and carbon dioxide.*

Application. *Methanol is used for the production of formaldehyde, in painting, as an additive in liquid fuels (to increase the octane number), antifreeze and the like.*

Occupational risk exists for workers, in unhermetic work processes and in contact with opened surfaces of the substance. Persons consuming poor alcoholic drinks with a high methanol content are at risk of non-professional poisoning.

Biotransformation. *Methanol penetrates the body mainly in the inhalation route. It is also possible its absorption through the skin. It is distributed in: liver, kidney, gastrointestinal tract, heart, muscle, adipose tissue and brain. A significant portion is oxidized to form formaldehyde and formic acid which lead to severe metabolic acidosis, damage to the nervous system and the optic nerve. At the cellular level, enzymes are blocked, such as hexokinase and the like. Methyl alcohol is eliminated in urine as formic acid and as non-metabolised methanol. Minor amounts are released as beta-glucuronide.*

Methanol has a narcotic effect and has a cumulative effect. The mechanism of its toxic action is associated with its oxidation products (formic acid and formaldehyde). The eye analyzer is damaged due to disturbance in the retinal oxidative phosphorylation.

Clinical picture.

Acute intoxications may occur when inhalation of high concentrations of methanol or ingestion (of life). The lethal dose is 10 to 60 cm^3, depending on the age and individual characteristics of the body. It occurs with headache, fatigue, dizziness, reduced hearing and tinnitus, abdominal pain and vomiting. In severe cases, seizures and coma are observed. Ocular nerve damage is characteristic. Initially, the pupils are enlarged and reduces the pupil's reflex. Later there was blindness, followed by cyanosis, Cheyne-Stokes breathing (due to bulbar paralysis), apnoe and lethal outcome (in over 20% of cases).

Chronic poisoning - headaches, tiredness, dizziness, disturbed sleep rhythm, visual disturbances, vegeto-vascular disorders - changes in Ashner-Dannini reflex and in orthoclinostatic reflex, eyelid and hand tremor, increased tendon reflexes, etc.

Laboratory researches. Strong reduction in blood alkaline reserves (BE), presence of methanol, blood condensed, increased red blood cell count and elevated hemoglobin. Urine contains albumin, hyaline cylinders, methanol and acetone.

Differential diagnosis is made with poisoning from ethyl alcohol, botulism, atropine, ethylene glycol, TEL, toluene, nickel carbonyl.

Treatment. Antidote is ethanol (reduces methanol degradation) - in acute poisoning 10g ethanol/h, 5% p-p, drop, intravenous.

Stomach wash with 2% sodium bicarbonate, 30 g magnesium sulphate in a glass of warm water, infusion therapy with glucose and alkaline water-electrolyte solutions, up to 3 liters per day. If the patient is conscious, alkaline agents can also be taken orally for 2-3 days (correction of metabolic acidosis). The hemodynamic parameters are controlled and caffeine, cardiazole, etc. are administered to maintain the cardiovascular system; Vitamins - C, B_1, B_{12}, against toxic damage to the optic nerve. Later, balneotherapy and physiotherapy were used.

Labour expertise. In the case of mild acute poisoning, the ability to work is temporarily lost, and in the case of severe poisoning, optic nerve damage, the patient is permanently disabled and is defined as a disability group. In case of chronic poisoning - labour recruitment without contact with toxic substances.

Prevention. Sealing during transportation and storage of methyl alcohol. Control of methanol concentrations in air. Use of filtering gas mask. Contraindicated for work with methanol are people with liver, kidney, eye, and nervous system diseases. Periodic preventive examinations are performed once a year, with the participation of a therapist, neurologist and ophthalmologist.

Ethyl alcohol

It is a colorless volatile liquid with a characteristic odor. Ethyl alcohol (ethanol) is slightly toxic, with marked lipotropicity, predominantly damaging the central nervous system and the liver, blocking the functions of a number of enzymes in cellular organelles. It destructed from alcohol dehydrogenase to carbon dioxide and water. Exposure to ethanol vapor or swallowing (**acute poisoning**) is mainly due to CNS changes: initially there are euphoria, hyperthymias, psychomotor agitation. In the case of more severe intoxications, the narcotic effect of ethanol-ataxia, somnolence, sopor, coma, hyporeflexia is manifested. Breathing and haemodynamic inhibition may occur.

Prolonged exposure to ethanol vapour(**chronic poisoning**) at concentrations above 5,000 ppm (10 times the normal) causes mucosal irritation (eyes, nose), headache, infirmity, fatigue, dizziness, drowsiness. Fallen on the skin causes dermatitis. Epidemiological data of carcinogenic effect - an increased incidence of laryngeal cancer, when exposed to synthetically produced ethanol, is associated primarily with its impurities - diethylsulphate and the like.

Laboratory studies show increased alcohol content in the blood, a positive urine ethanol sample. In the third and fourth degrees of acute alcohol poisoning, the excess of blood-bases (BE) is reduced - the presence of metabolic acidosis, the urinary acetone bodies increase.

Differential diagnosis. Intoxications with gasoline, benzene, toluene, acetone, carbon monoxide, other types of coma with non-professional etiology.

Treatment. Cerebroprotective agents are used: if needed, sedative (phenothiazine neuroleptics), or analeptics, respiratory and cardiocircular resuscitation.

Prevention. Effective ventilation of work rooms. Sealing of production processes and storage vessels. Preliminary and periodic medical examinations.

3.2.3. ETHYLENE GLYCOL - D. APOSTOLOVA

Ethylene glycol is a bivalent alcohol with high toxicity of its metabolites. It is a colorless liquid, water-soluble and highly chemically active.

Usage. *Comes into antifreeze (up to 95%), brake fluid, polyester fibers, plastics, resins, varnishes and paints. It is used as a solvent and raw material in many industries - pharmaceuticals, tobacco, leather, textiles, paints and varnishes.*

Toxic action. *Occupational poisonings with ethylene glycol - inhalation of vapors or aerosols, less frequently through the skin, are relatively rare. The toxicity of ethylene glycol itself is low, in the body is me-*

tabolized to formic acid, glycine or oxalic acid, which cause damage to the central nervous system, kidneys and extreme acidosis.

Acute poisoning. Usually, when swallowed after a latency period of 30 minutes to 12 hours, vertigo, headache, somnolence, ataxia, nausea, followed by severe sopor and coma intoxication, often myoclonic seizures, occur. Nystagmus and ophthalmoplegia (with no damage to the optic nerve), tachypnoea, tachycardia, sometimes cardiovascular failure may occur. During the second day, acute renal failure is developing with oliguria anuria and azotemia. The liver damage is infrequent and weaker.

Chronic poisoning. They occur with functional changes in the nervous system, such as headaches, dizziness, ataxia, short-term syncope, nystagmus, somnolence, vegetative dystonia with a tendency to hypotension. Ethylene glycol has a locally irritant effect on mucous membranes, cornea, conjunctivitis. In advanced cases, chronic dermatitis, toxic pneumofibrosis, hepatorenal syndrome with dominant tubulopathy, hepatopathy are noted. Anemia with granulocytopenia is rare.

Laboratory diagnostics. *Metabolic acidosis*, characterized by a reduction of standard bicarbonate (SB) below 20 mmol/l and a decrease in excess of bases (BE); *hypocalcaemia* (Ca below 2 mmol/l), *hyperchloremia* (chlorine above 110 mmol/l); *anemia with macrocytosis* (Hb below 140 g/l for men and below 120 g/l for women, Ery. less than 4.5 million for men and less than 4.2 million for women); segmented nuclear neutrophils (Sg) below 50%.

Exposure test not developed, ethylene glycol in urine may be determined at high exposure. *Oxalic acid in the urine* is also recommended - norme 15-20 mg/24h.

Differential diagnosis. Poisons with other organic solvents (methanol, ethanol, acetone, gasoline, toluene); diseases of general character affecting the nervous system, kidneys, liver and hematopoiesis.

Treatment.

In **acute** poisoning:

- gastric lavage with medicinal charcoal and saline cleanser (for oral admission).

- hemodialysis, acidosis correction and cleansing endotoxin products (nitrogenous bodies).

- infusion of sodium bicarbonate, alkaline and other salt and glucose solutions to maintain abundant diuresis - 3-5 liters per day.

- antidote treatment - ethanol to prevent the degradation of ethylene glycol by alcohol dehydrogenase, calcium gluconate 4x1g daily (as oxalic acid antidote).

- cerebro- and hepatoprotective agents, including B_1 and B_6.

- cardiocirculatory reanimation.

For **chronic** poisoning:

- inclusion of a salt-free diet with a protein restriction of up to 1-2 g/kg bodyweight for 24 hours.

- drip and intravenous infusions - 1-2 l per day of water-electrolytic, 10-20% glucose-levulose and alkaline solutions.

- intravenously 10% Calcium gluconicum 10-20 ml daily and at 5ml. 25% solution of magnesium sulphate.

- vitaminotherapy - vit. B_6, Vit. C.

- hepatoprotective and cerebroprotective agents.

Prognosis and labour-medical expertise. Acute poisoning is serious, and a mortal outcome often occurs. Chronic intoxications can lead to marked disability.

Medical prophylaxis. Do not work with ethylene glycol with CNS diseases, chronic liver and kidney disease, blood disorders and secondary malnutrition. Annual prophylactic examinations with therapist and neurologist and otorhinolaryngologist, with full blood count, urine and ASAT, ALAT, bilirubin, differential blood count, oxalic acid in the urine.

Prophylactic nutrition - diet B (with alkaline predominance), yogurt half a liter daily, vitamins (vitamin C 150 mg daily, vitamin B_6 50 mg daily).

3.2.4. PHENOL - J. HADJIEVA

Phenol is a colorless crystalline substance with a characteristic odor. It dissolves in water, chloroform, ether and oils. It has weak acidic properties.

Usage. *Phenol is one of the most commonly used hydroxyl derivatives of benzene. Used as monomer of phenol formaldehyde resin, in the manufacture of picric acid, dyes, pesticides, adhesives, etc. Phenol is the starting material for the synthesis of a number of aromatic compounds and pharmaceutical preparations.*

Biotransformation. *Phenol penetrates the body through respiratory, skin and gastrointestinal tract. Its respiratory absorption is high and represents 70-80% of the inhaled amount of phenol.*

The phenol resorbed in the body is distributed quickly and unevenly in the tissues in the following order: kidneys, liver, heart, blood, muscles. In the body, the phenol undergoes two oxidation reactions - hydroxylation of the aromatic nucleus and oxidation of pyrocatechin and hydroquinone in the corresponding quinones. The major amount of phenol is excreted in the urine as free and bound phenols in the form of sulphates and glucuronides. Small amounts are eliminated as carbon dioxide. The ultimate release of the phenol from the body occurs after 24 hours.

Pathogenesis. *Phenol belongs to the so-called*

protoplasmic poisons, which have lipotropic activity and the ability to quickly denature the proteins. It suppresses the oxidative-regenerating processes and disrupts the activity of a number of enzyme systems. It damages the nervous system and the parenchymal organs. It has an irritant effect on the mucous membranes and the skin.

Clinical picture. Acute poisoning is rare in professional conditions. Inhalation of phenolic vapors causes acute upper respiratory catarrh, bronchitis, bronchopneumonia, and pulmonary edema. Corrosion changes occur when the phenol enters the body via the gastrointestinal tract.

Nervous system disorders include headache, dizziness, agitation, and in more severe cases - somnolence, convulsions, coma, and paralysis of the center of breath. There is a development of toxic nephropathy and toxic hepatopathy. When phenol enters the skin, burns, necrosis (phenolic gangrene) and, in severe cases, the appearance of general resorptive symptoms.

Chronic poisoning. The clinical picture of chronic poisoning is characterized by a wide variety of symptoms and syndromes from different organs and systems.

- vegeto-asthenic manifestations (irritability, increased fatigue, impaired sleep and memory, unpleasant sensations in the heart, etc.)
- motor disorders (increased tendon reflexes, tremor, coordination disorders);
- cranial-brain dysfunction (insufficient convergence, nasolabial fold asymmetry, exophthalm);
- gastrointestinal discomfort (anorexia, dysphagia, ptyalism, dyspepsia, nausea, vomiting, stomach abdominal pain, epigastric pain);
- biliary-liver disorders (manifold symptoms of mild functional impairment to toxic hepatitis);
- renal impairment (most commonly transient albuminuria);
- skin changes (develop dermatitis or eczema).

Clinical-laboratory tests.

- Hematological parameters (decreased hemoglobin, decreased red blood cell count, aniso- and poikilocytosis, haemolysis);
- Urine (red color to green urine, proteinuria, erythrocyturia, oliguria, sediment of the urine - epithelial, granular and erythrocyte cylinders, abundant kidney epithelial cells);
- Clinical and chemical indicators (increased content of residual nitrogen and residual nitrogen fractions in blood serum, increased serum activity of ASAT, ALAT, LDH, AF, guanase).

Differential diagnosis. Poisoning with other corrosive poisons (dimethylsulfate, chlorine, nitrogen oxides), carbondisulfide, toluene and others. Chronic poisoning is differentiated from diseases of the nervous, urinary, digestive, liver at non-professional origin.

Treatment. In cases of acute poisoning immediate contact with the toxic substance is carried out and procedures for its removal from the body are performed. Calcium gluconate, infusion therapy with glucose and water-salt preparations, respiratory resuscitation, anti-shock therapy, antibiotics, exchange blood transfusion are used. In chronic poisoning, treatment is symptomatic. Hepatoprotectors, nephroprotectors, vitamin therapy and others are used.

Prevention. Sanitary-technical-sealing and automation of technological processes, provision of effective general and local ventilation, use of special work clothes of impermeable tissue and other individual protective means. In emergency situations, it is imperative to use a gas mask.

Medical prophylaxis. Do not work in contact with phenol, people with diseases of nervous, respiratory and urinary system, liver, skin and visual analyzer diseases. Periodic medical examinations are performed once every 12 months with the participation of a therapist, otorhinolaryngologist, and at indications - neurologist, ophthalmologist and dermatologist. Protective professional nutrition - diet "D".

3.2.5. KETONES - D. CHARYKCHIEV, ST. ANDONOVA

Ketones are aliphatic hydrocarbons which are chemically characterized by the presence of a carbonyl group (-C = O). They are colorless, volatile liquids with a specific odor. Representative are the compounds: acetone, methyl acetone (methylethylketone), acetophenone, acetylbromide, acetylchloride, cyclohexane and the like.

Acetone *(dimethylketone, propanone) is used in the manufacture of paints, varnishes, plastics, artificial rubber, explosives, photography, microelectronics and others.*

Methylacetone *(methylethylketone, butanone) also has excellent solvent properties and is used as a component in many organic solvents to denature ethanol and the like.*

Biotransformation. *Entrance hole - breathing path, digestive tract and skin. Breathing in the air rapidly saturates the blood, but is slowly eliminated by the body. They have cumulative properties. Acetone accumulates mainly in the brain, liver, pancreas, kidneys, lungs, muscles, heart. Metabolized by oxidation to carbon dioxide and partly to isopropanol. It is associated with glycogen, fatty acids and amino acids. It is elim-*

inated in urine as unchanged (7-8%), and its metabolites; through the skin - 10% of the body's ingestion; as carbon dioxide and unchanged with exhaled air up to - 50% of the received. Ketones have the ability to induce cytochrome 450. They affect urea and cholesterol metabolism.

Clinical picture. In **acute poisoning**, symptoms of respiratory tract irritation, such as rhinorrhea, cough, chest pain, burning, skin irritation, are observed. In cases of severe intoxication, there are nausea, vomiting, headache, dizziness, disturbance of consciousness to coma. Acidotic breathing occurs. An alkaline acid disorder is impaired with severe metabolic, and later respiratory, acidosis. Acetone and methyl acetone have no axonolytic activity in contrast to other ketones that form a gamma ketone with an axonolytic action in metabolism and lead to ascending polyneurites. Some of the ketones have pronounced hepatotoxic and nephrotoxic effects. Acetone describes the occurrence of cataracts, for methyl acetone phototoxic effect.

Chronic effects are mainly characterized by skin changes - dermatitis. The skin is dry, reddened, itching appear. Affects mucous membranes - conjunctivitis, rhinitis, pharyngitis. Also head gravity and headache, vertigo, salivation, functional neurosis, decreased body mass.

The diagnosis is based on the professional history or household risk, the presence of acetone, methyl acetone in blood and urine, the expressed acidosis. Acetone in urine can not be used as an exposure test.

Differential diagnosis - poisoning with other organic solvents.

Treatment. Breathing resuscitation. Rapid correction of acidosis with sodium bicarbonate administered intravenously. Symptomatic treatment. Upon receipt per os - gastric lavage.

Prevention. Sealing of production processes. Effective ventilation and PPE. People with diseases of the nervous system, liver, anemia are not allowed to work. Periodic medical examinations - once a year.

3.2.6. CHLORINATED HYDROCARBONS - D. APOSTOLOVA, J. HADJIEVA

They are excellent organic solvents with high toxicity. They are used in the manufacture of paints, lacquers, plastics, dry cleaning, fire extinguishers and refrigerators, household cleaning products, etc.

Tetrachloromethane (carbon tetrachloride)

Biotransformation. Pathogenesis. *After entering the body, trichloromethyl is produced and no-chlorine free radicals are formed under enzymatic action, to form complexes of proteins, mainly in hepatocytes. Violated protein and lipoprotein synthesis in the endoplasmic reticulum, energy processes (production of adenosine triphosphate), followed by fatty degeneration and centrolobular necrosis in the liver, tubular necrosis in the kidneys, degenerative changes and narcotic effect in the CNS.*

Clinical picture. Vapor exposure after a latency period of 2-24 hours results in rapid transient irritation of the respiratory mucosa (in very severe intoxications and pulmonary edema). Headaches, dizziness, nausea, ataxia, confusion, somnolence, visual disturbances followed by coma, depression of the breathing and circulation, cardiac dysrhythmia, eventually and clonic-tonic convulsions occur. Damages of parenchymal organs: enlarged, painful liver, jaundice, elevated bilirubin (conjugated) in the blood and urinary incontinence, high values of ASAT, ALAT, LDH and ammonia in the blood, low fibrinogen and prothrombin time, haemorrhagic diathesis, renal failure with oligo-anuria, azotemia, hyperkalaemia. Analogous injuries develop in oral, (with mainly hepatic lesions) and percutaneous intoxication. The latter also has contact dermatitis with erythema, fissures and desquamation.

Trichlorethylene

Trichlorethylene is a colorless liquid with a sweet smell and marked volatility. Dissolve well in organic solvents and slightly in water. Long-term storage of sunlight and contact with an open flame forms phosgene.

Usage. *Trichlorethylene is used as a solvent for lacquers, waxes, rubber, sulfur, phosphorus and the like. It is used for dry cleaning of clothes, extraction of fat from metal parts, etc.*

Biotransformation. Pathogenesis. *Trichlorethylene penetrates the body inhaled. Inhaled vapors are absorbed very rapidly and are distributed predominantly in organs and tissues rich in lipids, mainly in the CNS. Absorption is also possible through the skin. Trichlorethylene passes through the placenta.*

Approximately 10-20% of trichlorethylene absorbed is excreted unchanged and 70-90% is mainly metabolised to trichlorethanol and trichloracetic acid. Trichlorethylene metabolites are excreted in the urine - for long-term exposure, the trichlorethanol / trichloracetic acid ratio is 2:1.

Trichlorethylene has a narcotic and neurotoxic effect. It primarily affects the nervous system. There is a habit of becoming addicted to it. It has a toxic effect on parenchymal organs and has a carcinogenic effect.

Clinical picture. Acute poisoning as a result of inhalation of high concentrations of trichlorethylene causes a state of narcosis and coma. Symptoms of toxic hepatitis, toxic nephropathy and pulmonary edema have been reported. Short-term inhalation

of trichlorethylene vapours at lower concentrations than narcotic, causes irritation of the upper respiratory tract and eye. Vasomotory and dyspeptic events, headaches, vertigo, vomiting, sometimes tremor, increased tendon reflexes, olfactory disorders, mental abnormalities, etc. are observed.

Chronic poisoning with trichlorethylene occurs primarily with nervous system damage.

The mild and moderate degree of chronic poisoning is characterized by astheno-vegetative changes (disturbed sleep, headache, vertigo, increased fatigue) and vegetovascular manifestations (bradycardia, hypotension, extrasystoles). It is also possible to have microorganic symptoms (neuritis and polyneuritis of vegetative-sense type, CNS defeat).

Severe stage with toxic encephalopathy, mental disorders, epileptiform convulsions. The damage to other organs and systems by trichlorethylene is linked to the endocrine system (menstrual-ovarian and male sexual dysfunction) and skin (trophic disturbances of distal phalanxes around the nails, dermatitis, eczema, etc.). The carcinogenic effect of trichlorethylene has only been described in experimental conditions (there is no higher incidence of malignancies in exposed individuals).

Clinical-laboratory tests. Deviations in haematological parameters (polycythemia, lymphocytosis and leucopenia) are observed. Deviations in the clinical and chemical parameters are found with relevant organ damage (liver, kidney, etc.). Excretion tests use the content of trichloroacetic acid and trichloroethanol in urine.

Methylchloride, methylene chloride, chloroform, ethylchloride, dichloroethane, tetrachlorethane, pentachloroethane, dichloroethylene, tetrachlorethylene, dichlorethane, dichlorpropane, ethylenechloride, dichlorethylether, dichlorohydrin, chloroprene, isopropylchloride, propylene chloride, allyl chloride, propylene dichloride, cause carbon tetrachloride and trichlorethylene-like changes.

Chlorinated naphthalenes (CN)

These include all derivatives of mono- to octachlor naphthalene. They are solids with a light yellow, greenish or brownish color. Generally, mixtures of chlorinated naphthalenes are used in industry.

Usage. *Used as a substitute for wax, resin and rubber; in the cable production, production of capacitors and transformers; tissue impregnation; in the ethyl alcohol composition for the etylation of petrol.*

Toxic action. *CN toxicity increases with increasing chlorine atoms in their molecule, primarily damaging the liver and other parenchymal organs - kidney, pancreas, heart. Individual sensitivity is observed. They penetrate the body mainly by inhalation of vapors or dust (by condensation), and possible poisoning through the skin pathway.*

Acute poisoning. Under production conditions, because of the low volatility of CN, acute poisoning is unlikely. They are characterized by hepatic and cerebral syndromes. Nervous-psychical disorders manifest with suppression (vapidity, apathy, drowsiness) followed by excitation (trembling, motor anxiety, manic state), deepening to coma.

Hepatic impairment is manifested by the picture of toxic hepatitis that can pass into acute or subacute yellow dystrophy of the liver, cirrhosis.

Chronic poisoning. In mild cases, it is characterized by common and transient disorders, weakness, headache, dizziness, nervousness, sometimes accompanied by catarrhal phenomena (tearing, coughing, nasal catarrh).

Initial liver changes occurred with right hypochondriac pain, hepatomegaly, subicteric sclera discolouration to skin icterus, bilirubinemia, faecal discolouration, urobilinogen and bile pigments in the urine.

At a more advanced stage, it develops toxic hepatitis, chronic hepatocholescitis, cirrhosis. Gastrointestinal tract disorders - gastritis, duodenitis, stomach and duodenum ulcers often occur. Toxic hepatitis is often accompanied by skin lesions - photodermatitis (erythematous and erythemo-vesicular) with subsequent pigmentation and subjective sensations of itching and burning when working in the sun. It also affects the follicular apparatus with the appearance of comedones, chlorine acne, and secondary infections.

Clinical-laboratory tests. Bilirubinemia up to two to three times the norm, elevation of serum aminotransferases (ASAT and ALAT) and γGTP three to four times the normal; bile pigments in the urine - bilirubinuria and hyperurobilinogenuria; cholesterol over 7 mmol/l, hypoglycaemia and hypoproteinaemia, Hb elevation, gastric hypoacid hyperaciditis. Urine naphthol metabolites can be examined as an exposure test.

Differential diagnosis of chlorinated hydrocarbons - exclusion of diseases of the nervous and secretory systems, the liver and skin with non-professional etiology. Also intoxications with benzene, toluene, acetone, gasoline and others.

Treatment. Acute poisoning requires immediate discontinuation of the poison and its removal (gastric lavage, cleansing agents, skin wash, etc.). When indicated, analgesics, respiratory resuscitation, drip vein infusion of water-soluble and glucose solutions, hepatoprotective agents, The treatment of chronic poisoning is symptomatic. It also uses vitamin therapy, general strengthening agents, balneotherapy

and others.

Labour expertise. Acute intoxications are most often associated with temporary loss of working capacity. In case of residual phenomena due to acute poisoning or in case of pronounced symptoms of chronic poisoning (eg. chronic hepatitis) permanent rehabilitation without contact with toxic substances)

Prevention. Sanitary-technical prophylaxis (sealing and automation of production processes) Current control of concentrations of chlorinated hydrocarbons

Medical prophylaxis - do not work, people with diseases of the nervous, secretory, endocrine and cardiovascular systems, of the liver and skin. Prophylactic medical examinations are performed once every six to twelve months with the participation of a neurologist and therapist, and by indications - a dermatologist and ophthalmologist. Protective professional nutrition - D diet.

3.2.7. AMINO- AND NITRO DERIVATIVES OF BENZENE. TRINITROTOLUENE - J. HADJIEVA

Amino- and nitro derivatives of benzene *represent a large and varied group of compounds. With less volatility and high lipo-solubility.*

Usage. *They are widely used in the chemical industry as a starting product in the manufacture of more complex chemicals, paints, synthetic resins, explosives, in the glass, pharmaceutical, rubber industry and others.*

Biotransformation. *Nitro compounds of benzene fall into the body through the airways, the skin and the digestive tract, and its amino compounds - mainly inhaled and through the skin. A major reaction in the biotransformation of aromatic nitro derivatives is the reduction with the participation of the enzyme nitroreductase. A characteristic metabolic reaction of aromatic amines is acetylation. Using the enzyme arylaminoacetyltransferase, they are converted to aromatic amides.*

Pathogenesis. *The major pathogenetic mechanisms of injuries caused by the amino and nitro derivatives of benzene are related to their action as methaemoglobin-generators, as well as to their allergenic and carcinogenic effects.*

Clinical picture. There are three levels of **acute poisoning**:

Light Grade. Clinical features include headache, general fatigue, disorientation, euphoria, or drowsiness. Cyanosis of the labia, nose, ears and fingers is observed.

Middle grade. Typical are persistent headaches, dizziness, nausea, vomiting, tachycardia, moderate dyspnoea. Cyanosis is getting stronger. Methaemoglobinaemia fluctuates from 30-40%, Heinz's body is found in erythrocytes.

Severe grade. The symptoms described intensify, disorders of sensitivity, visual acuity, speech occur. There may be convulsions and a coma may occur. Blood methaemoglobin content reaches 60%. It is possible to develop a hemolytic process (jaundice, increase of indirect bilirubin, hemoglobinuria, hepatomegaly). Acute kidney failure may also occur.

Chronic poisoning. The clinical picture of chronic poisoning depends on the chemical characteristic of the toxic substance and the duration of exposure. Neurasthenic syndrome (headache, fatigue, dizziness, feeling of fear, etc.) is most often manifested. With prolonged exposure, it is possible to develop degenerative processes in the gray matter of the brain, defeat of some peripheral nerves, obscuration of the lens of the eye, narrowing of the field of vision, central scotomas, etc.

Hepatic damage fluctuates from mild functional disorders to toxic hepatitis. Hepatorenal syndrome is often seen.

Skin disorders most commonly occur as keratosis, dermatitis, eczema, acne vulgaris, etc. Benign and malignant bladder neoplasms have been reported in individuals in contact with aniline, alpha-beta-naphthylamine. The allergic action of dinitrchlorobenzene, p-phenylenediamine (so-called ursolic occupational asthma) is well known.

Clinical-laboratory tests. A pathognomonic sign of poisoning with amino- and nitro compounds of benzole is damage to the blood system - methaemoglobinaemia, haemolytic anemia, Heinz body in erythrocytes, etc. Urinary tests show albuminuria, erythrocytria, increased urobilinogen and porphyrin. Deviations in enzyme, detoxic and other liver functions are observed.

Differential diagnosis. With some acute drug poisoning (phenacetin, levomycetine, etc.), with nitrogen oxides, arsenic acid, and the like. Chronic poisoning should be differentiated from diseases of the nervous system, blood and hematopoietic system, damage to the liver, skin, etc. with non-professional etiology.

Treatment. Acute poisoning includes - gastric lavage, saline cleanser, skin dressing. Breathing resuscitation is performed by indication. Haemoprotective agents (haemotransfusion or exchange blood), glucocorticoids, infusion therapy are used. Antidotic treatment with methyleneblue (1-2 mg/kg body weight, 1% sol. venous for 5 min) - at methaemoglobin above 35-45%. Anti-shock therapy is used in acute circulatory disorders, and in cases of

acute renal failure - dialysis methods. The treatment of chronic poisoning is symptomatic.

Labour expertise. In light poisoning, working efficiency is temporarily reduced. The presence of hemolytic and hepatic changes necessitates labour rehabilitation without contact with toxic substances. In severe cases of poisoning (toxic hepatitis, pronounced anemia, bladder papilloma, etc.) a disability group is defined.

Prevention. Sanitary-technical prophylaxis (sealing, ventilation, use of individual protective equipment, etc.).

Medical prophylaxis - Do not work in contact with benzene amine- and nitrocompounds, people with diseases of the nervous and urinary system, diseases of the blood and the hematopoeitic apparatus, the liver and the skin. Periodic medical examinations are performed once every 12 months with therapist and by indications - a dermatologist. Protective professional nutrition - D diet.

TRINITROTOLUENE (trotyl) *has six isomers (technical trinitrotoluene contains alpha-, beta-, gamma-, and delta-trinitrotoluene). It is a crystal yellow mass. Dissolves in ethanol, benzene, toluene and ether, is difficult to dissolve in water.*

Usage. *It is mainly used in the military industry (explosive material, alone or mixed with other substances). Wide is applied in the production of various dyes.*

Biotransformation. *Trinitrotoluene penetrates the body primarily through the respiratory tract and through the skin. Metabolism occurs in the liver. It is subjected to reduction processes to form aminonitrotoluene. Part of the trinitrotoluene is oxidized to trinitrobenzyl alcohol, which, after reduction, forms dinitraminobenzyl alcohol. The two alcohols bind to glucuronic acid and in the form of glucuronides is eliminated in the urine.*

Pathogenesis. *Trinitrotoluene has a haemotoxic action - it causes methaemoglobinaemia. There is also a pronounced hepatotoxic effect - this effect is associated with its ability to selectively disturb amino acid metabolism. Trinitrotoluene is also known as a carcinogen.*

Clinical picture.

Light acute poisoning occurs with headache, dizziness, general weakness, dyspeptic symptoms, nausea, vomiting.

Severe acute poisoning is characterized by brain manifestations, methemoglobinemia, acrocyanosis, dyspnoea, toxic hepatitis, sometimes aplastic anemia.

Chronic poisoning. Alcoholism, gastrointestinal diseases, obesity, etc. are predisposing to them.

A major characteristic of trinitrotoluene poisoning is the triad: toxic hepatitis, toxic anemia and toxic cataracts. Trinitrotoluene cataract is a sign of toxic injury in its early stages (possibly in isolated cases, the established cataract is also the only criterion indicating retrospective intoxication with trinitrotoluene).

Hepatic impairment may persist from mild functional disorders to toxic hepatitis, cirrhosis and acute yellow atrophy. The observed anemia is of a hypochromic type.

Effects of other organs and systems include: astheno vegetative symptoms, gastrointestinal symptoms (dyspeptic phenomena, gastroenteritis), skin diseases (allergic contact dermatitis, eczema), hair coloration in red, yellowing of the palms and nails, acrocyanosis, menstrual disorders and others. A higher incidence of skin cancer has been reported among trinitrotoluene exposed.

Clinical-laboratory tests. Hematological parameters - decreased hematocrit and hemoglobin, reticulocytosis, methaemoglobinaemia, leucopenia with eosinophilia, lymphocytosis, thrombocytopenia, macrocytosis, increased number of nuclear erythrocytes, Heinz body.

Clinical and chemical parameters - a heterogeneous increase in the activity of serum enzymes - ASAT, ALAT and LDH, increased cholesterol.

Differential diagnosis - with unprofessional diseases of the blood, liver, visual analyzer, skin, etc.

Treatment. The treatment of acute poisoning with trinitrotoluene takes place in the same way as nitrobenzene poisoning, supplemented with hepatoprotective agents. In chronic poisoning, treatment is symptomatic-anti-anemic agents, vitamin therapy, (A, C, etc.), hepatoprotectors and others.

Medical prophylaxis. Do not work in contact with trinitrotoluene, people with diseases of blood and hematopoietic system, liver, visual analyzer, skin, etc. Periodic medical examinations are performed once every 12 months with the participation of therapist, ophthalmologist, and at indications - gynecologist. Protective professional nutrition - diet "D".

3.2.8. GASOLINE - R. STEFANOVA

Benzine is a complex mixture of saturated and unsaturated aromatic hydrocarbons and sulfur compound impurities. It is a colorless or slightly yellowish liquid insoluble in water, well, it dissolves in organic solvents. Its vapour is heavier than the air.

Usage. *It is used in a variety of industries such as solvent for rubber, lacquers, oils, degreasing of metal parts, extraction of fat. Particularly important is its importance as a motor fuel.*

People at risk of intoxication are workers in garag-

es, gas stations, drivers, fitters, workers in the rubber industry, and workers in yielding, processing, transportation and storage.

Biotransformation. *Gasoline enters the body through respiratory, skin and digestive tract. Saturation of the blood and the nervous system with gasoline is happening very fast. It is excreted through the lungs unchanged.*

Gasoline is a lipotropic factor and damages predominantly lipid-rich organs - brain, liver.

Clinical picture. Acute poisoning occurs when large amounts of petrol vapors are inhaled in closed spaces - cleaning cisterns and tanks, spilling gasoline in closed workshops, passing gasoline with suction (inhaled and oral entering).

Benzine has a narcotic effect and damages the functional state of the central nervous system, the extent of its toxicity depends on the percentage of its constituents. Higher levels of octane in gasoline cause rapid deep narcosis, while heptane acts paralytically on the respiratory center.

The following forms of acute poisoning have been observed:

a) a fulminant form - rapid loss of consciousness, and if the injured does not come out of the environment, death occurs from respiratory paralysis.

b) severe intoxication - headache, dizziness, psycho motor woes, unmotivated laughter or crying, hysteroidal seizures. Arterial pressure drops, the pupils are in mydriasis, occurs hyporeflexia, seizures, and can lead to coma.

c) lighter acute poisoning occurs with a condition similar to alcoholic intoxication. Aspiration of petrol vapor may develop toxic pneumonia, manifested by dyspnoea, chest pain, cough with bloody sputum, rapid breathing and tachycardia.

Gasoline pneumonia is accompanied by fever. Radiography shows massive obscurations in the lungs.

When gasoline is ingested, cataracts of the digestive system are observed, along with the typical nervous signs of benzine poisoning.

After acute poisoning, residual phenomena such as emotional instability, neurosis-like conditions, impaired vestibular function, and neuritis are observed.

Chronic intoxication is manifested by neuroses, disturbances in the dark, irritability, frustration, sometimes ataxia, vegetative polyneuritis, dizziness, tinnitus, chest constriction, head gravity and persistent headaches, vascular disorders, accomodative eye asthenopathy, disturbed memory, psychoses, etc. Objectively, vascular disorders, decreased sense of smell, red dermograms, tremor, hyperreflexia, Ashner-Danny's changed reflex were detected. The electroencephalographic study shows reduced alpha waves, the occurrence of beta waves layered on the theta waves, and also paradoxical reactions of light stimuls. At the same time there may be manifestations of the nasopharynx - rhinitis, pharyngitis, laryngitis; by the eyes - conjunctivitis; gastrointestinal manifestations - dyspeptic disorders, toxic liver damage. The menstrual cycle is disturbed, pregnant women may experience spontaneous abortions and stillborns. When it comes to skin, gasoline dissolves fat, skin is dry and cracked, and dermatitis and eczema may develop. In some cases, bronchial asthma is also observed.

Clinical-laboratory tests. Moderate decreases in hemoglobin and erythrocyte count, as well as lymphocytic leukopaenia, are observed. Sometimes anisocytosis and polychromasia and basophilically stippled erythrocytes have been found. Impaired lipid metabolism - an increase in total cholesterol and free fatty acids in the serum. The activity of the ACAT, ALAT enzymes and beta-lipoprotein in the blood serum is increased.

Differential diagnosis. In acute gasoline poisoning, differential diagnosis is made with alcohol and barbiturates poisoning, benzene, acetone, chlorinated hydrocarbons, upper respiratory tract catarrh and pneumonia, and in chronic - for diseases of the nervous system, liver and the haemal system with non-professional etiology.

Treatment. The injured is immediately brought into the fresh air. Oxygen therapy (carbogene), agents which stimulates respiratory and cardiac activity is used. At aspiration of gasoline - gastric lavage with medical coal. Follow-up treatment with antibiotics, against the possible development of pneumonia.

At chronic poisoning uses tranquilizers, vitaminotherapy (vitamin B_1, C), physiotherapeutic procedures, and more.

Occupational medical expertise. In light poisoning, working capacity is temporarily lost. In case of residual phenomena of acute intoxications and in chronic ones, labour is required without contact with toxic substances.

Prevention. Sealing the production process, adherence to all rules of safe operation and dynamic monitoring of gasoline concentrations in the air of the working environment.

Medical Prevention. Do not work in contact with gasoline persons with liver, nervous, digestive and haemal system diseases. Periodic medical examinations are performed once every 12 months with the participation of therapist, neurologist and dermatologist. Protective nutrition, with more proteins and vitamins - diet D.

3.2.9. CARBONDISULFIDE - ST. ANDONOVA, S. PAVLOVA

Acute poisoning

- disorder, manic delirium, hallucinations, paranoia
- upper respiratory tract inflammation

Chronic poisoning

- coronary disease
- neurobehavioural disorders
- retinal micro-aneurism
- peripheral neuropathy with ascending, symmetrical paresthesia and weakness

Physical and chemical properties. Carbondisulfide (CS_2) is a colorless, volatile liquid with unpleasant odor. Evaporates at ordinary temperature. Its vapour is 2.6 times heavier than the air. It is easily flammable and explosive. Good solvent is for fat, wax, rubber, sulfur, phosphorus, iodine, etc.

Usage. *Carbondisulfide is used in the production of artificial fibers (viscose), waterproof adhesives, as a solvent for fat, waxes, in the rubber and chemical industry, for the extraction of essential oils and floral perfumes, for the production of tetrachlormethane, rhodanides, optical glass, pesticides, at flotation of ores, as an insecticide and others. It is separated in coal coke and in distillation of charcoal (it is obtained by interaction with sulfur vapors).*

Biotransformation. *In production conditions it enters the body through the airway and through the skin as one part dissolves in body fluids, so-called free carbondisulfide, and the other part is bound to the amino acids to form thiocarbamates.*

About 70% of the carbondisulfide cumulated. Metabolized to 2-thiothiazolidine-4-carbonic acids. It is eliminated by exhaled air (up to 30%), urine-unchanged (1%) and up to 90% as metabolites: thiourea, thiocarbamic acids, sulphates and others.

Pathogenesis. *Its toxic effect on nerve structures is determined by its lipotropicity, its ability to bind sulfhydryl and amine groups, resulting in cellular metabolism disorders. The genetal toxic effect of CS_2 is related to the inhibition of mixed function oxidases, which leads to increased levels of cholesterol, steroid hormones, prostaglandins, fatty acids with the development of the corresponding pathology - endocrine, cardiovascular and nervous system disorders.*

Clinical picture. Acute poisoning occurs in emergency situations. They are expressed in three stages: **mild**-irritative effects on the mucous membranes of the eyes and the airways, headache, fatigue, psychomotor agitation; **moderate degree** - vomiting, vertigo, visual disturbances; **severe degree** - comatose status. Acute psychoses with delirium are observed in moderate and severe grades.

Chronic poisoning occurs with: **neurological syndrome** characterized by an equilibrium disorder, memory disorders, visual disturbances, retrobulbar toxic neuritis, polyneuritis, toxic encephalopathy and sometimes psychical abnormalities; **cardiovascular syndrome** - increased arterial blood pressure, angina pectoris with conductive and rhythmic disorders, "carbondisulfide atherosclerosis"; **gastrointestinal complaints** - appetite disorders, stomach weight, nausea, vomiting, gastro-intestinal disorders and ulcerative disease; **endocrine disorders** - disturbances in the menstrual cycle and the course of pregnancy, impotence, hypothyroidism, diabetes. Hepatotoxic, nephrotoxic effects, anemic syndrome, dermatitis (erythema, vesicles, chemical burn) can be detected.

Clinical-laboratory tests.

The diagnosis is based on the professional history, clinical manifestations, the amount of carbondisulfide in the exhaled air, the blood, the evidence of urinary metabolites (thiourea, thiocarbamic acids, sulphates), iod-azide test-biological markers for acute and chronic exposure.

Differential diagnosis - poisoning with methanol, toluene, tetraethyl-lead, freons, damages of cardiovascular, nerve and endocrine functions with non-professional etiology.

Treatment. In acute poisoning - inhalation of alkaline aerosol preparations, oxygen-therapy, respiratory resuscitation, forced diuresis, combination of pyramem, vit. B_6, B_{12}, symptomatic drugs, depending on the stage.

In chronic poisoning, treatment is based on the leading symptoms.

Prevention. Sealing of production processes, ventilation, control of the working environment, personal protective equipment. Medical prophylaxis. It is necessary to carry out a strict medical examination at the beginning of the work. The review from an internist, neurologist and ophthalmologist should include a detailed history of past illness. It is desirable to do ECG and laboratory tests (cholesterol, blood sugar, transaminases). As the primary criterion for professional selection, a disulfiram test is used to demonstrate inborn or acquired deviation of mixed function oxidases. Contraindications for entry are: pronounced neurosis, organic diseases of the central and peripheral nervous system, cardiovascular diseases, chronic diseases of the liver and kidneys, diabetes, ulcerative disease. Periodic prophylactic examinations are performed for 12 months by an internist, neurologist, ophthalmologist, obstetrician and gynecologist, and include an iod-azide test, cholesterol, thiourea and urinary protein. Protective nutrition - diet "D", rich in lipotropic factors, vitamins (C, B_1, B_6), copper, zinc.

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3.3 POLYMERS

POLYMERIZATION POLYMERS

Plastics	Monomer	Acute poisoning	Chronic poisoning	Exposure tests
Polyethylene	ethylene	headache asthenia arrhythmia	cerebrasteny autonomic dysfunction IHD	–
Polyvinylchloride	vinyl chloride	>10000 ppm narcotic action hepatitis	Raynaud's phenomenon osteoporosis, acroosteolysis hepatitis, cirrhosis, angiosarcoma scleroderma anemia pulmonary fibrosis, asthma	Le↓ Thr ↓ tiodiglycolic acid in urine> 5ppm
Polystyrene	styrene	conjunctivitis pharyngolaryngitis dermatitis	conjunctivitis, pharyngitis, bronchitis anemia, jaundice, hepatitis myocarditis headache, asthenia uncritically behavior discoordination cognitive and behavioral changes memory loss	Le↓ Thr ↓ ASAT ALAT ↑ ↑ mandelic acid > 200mg/l phenylglioxalic acid > 20mg/l in urine EEG-diffuse slow waves psychological tests CT
Polyacrylonitrile fibers	acrylonitrile	irritation UAP cyanosis dizziness disorientation convulsions coma	conjunctivitis, keratitis atrophic rhinitis vocal cord paresis autonomic dysfunction vestibulopathy IHD anemia dermatitis, necrosis increased risk of Ca pulmo	thiocyanates in urine> 2,5 mg/g creatinine
polymethyl-methacrylate	Methyl-methacrylate	asthenia gastrointestinal disorders headache loss of consciousness, epileptiformic convulsions	conjunctivitis, rhinopharyngitis dermatitis arterial hypotension hepatitis hypochromic anemia autonomic dysfunction polyneuropathy vestibulopathy encephalopathy	formaldehyde blood> 0.2 mmol/l formaldehyde urine > 0,3mmol/l methanol in urine> 15 mg/l formic acid in urine - 80mg/g creatinin

POLYCONDENSATION POLYMERS

Plastics	Monomer	Acute poisoning	Chronic poisoning	Exposure tests
Polyurethanes	polyisocyanates polyvalent alcochols tertiary amines	pneumonia, bronchitis, pulmonary edema burning of the cornea and skin	COPD bronchial asthma allergic dermatitis	–
Phenol-formaldehyde resins	phenol	in splashing of 1/4 to 1/2 of the skin - death fever breathing disorders	pharyngitis, bronchitis gastritis neuroses	phenol in urine >0,16mmol/l
	formaldehyde	mucous membrane and UAP cough, dyspnea, coma rashes, dermatitis	conjunctivitis, pharyngitis bronchitis, asthma contact dermatitis	Formaldehyde blood >0,2 mmol/l formaldehyde urine >0,3mmol/l
Epoxy resin	epichlorohydrin diphenylpropane ethylenediamine	contact dermatitis and eczema	allergic dermatitis and eczema autonomic dysfunction gastritis	phenol in urine

SYNTHETIC RUBBERS

Kinds	Monomers	Acute poisoning	Chronic poisoning
Butadiene	1,3-butadiene (divinyl)	headache, dizziness, asthenia, tachycardia, hypotension, coma	neurasthenia, palpitation, hypotension, dyspepsia conjunctivitis, pharyngitis, leucopenia with lymphocytosis, dermatitis, eczema.
Butadiene-styrolous	butadiene – styrene	conjunctivitis pharyngitis bronchitis hepatitis	autonomic dysfunction myocardiodystrophy toxic hepatitis, dyslipidaemia menstrual disorders
Isoprenous	isoprene + lithium salts	asthenia dyspepsia mucous membrane and UAP irritations	neurasthenia syndrome autonomous distal polyneuropathy increase of thyroid toxic hepatitis allergic dermatitis
Butyl rubbers	isobutylic isoprene	conjunctivitis pharyngitis	asthenia, abnormal function of the ANS and CNS rhinopharyngitis
Chloro-prenous	chloroprene	conjunctivitis, pharyngitis, tachycardia, collapse leukopenia hair loss	neurasthenia syndrome autonomous vasomotor disturbances myocardyodistrophy toxic hepatitis, nephrosonephritis dyspepsia anemia, reticulocytosis dermatitis and eczema

3.3.1. ACRYLAMIDE - V. PETKOVA

Acrylamide is an acrylic acid compound and has a significance as chemical professional factor in the production and processing of polymers (polyacrylamide), when used as a flocculants.

Biotransformation. *Acrylamide enters the body through the airways and skin. Because of its good solubility in water, its distribution to the body's tissues is rapid, with the highest concentrations found in erythrocytes. Biotransformation is via the glutathione system. When conjugated to the latter, nontoxic metabolites are formed, which are released relatively quickly and mainly in the urine.*

Toxic effect. *The toxicity of acrylamide is associated with its ability to disrupt by binding to cellular DNA, the protein synthesis in the proprioceptors, neurons, and axonal endpoints of motor fibers. Its effects on neuromediator functions have also been determined by reducing the concentration of noradrenaline, dopamine etc. in central nervous system.*

Clinical picture.

Acute poisoning. They run through manifesting disorders in the CNS - behavioural abnormalities, auditory and visual hallucinations, seizures. In severe forms there is a loss of consciousness and respiratory distress syndrome. Several weeks after the acute accident are expected to develop late peripheral neuropathy.

Chronic poisoning. Specific symptoms in the early stages of chronic poisoning are abundant sweating, erythema and desquamation of the palms and feet, forming contact or allergic dermatitis and eczema. Typically, these skin changes precede the emergence of classical neurological syndromes in the clinical picture of chronic poisoning. Feeling numb, constant freezing of the fingers, difficulty in fine movements (when writing, for example), muscle pain and weakness in the wrists and feet are the first symptoms of peripheral nervous system involvement. Consequently, the symptoms cover the whole limbs. On the background of toxic cerebrastheny, the typical for moderate and severe stages of poisoning "dying back" neuropathy develops with distal hypesthesia and limb paresis and severe ataxia phenomena - so-called **acrylamide pseudotabes**.

Acrylamide is assigned by IARC to a 2-B group of chemicals - a probable carcinogen of lower probability.

Clinical-laboratory tests. Electromiographic data for axonal lesion with reduced amplitude of induced muscle or nerve potentials as well as delayed conduction in motor fibers in severe cases of peripheral neuropathy. In skin manifestations - positive skin-allergic tests to acrylamide. Exposure tests for acrylamide have not been developed.

Differential diagnosis. Poisoning with acrylonitrile, styrene, vinyl chloride, benzene, methanol.

Treatment. Treatment of acute and chronic poisoning is symptomatic. There is no antidote therapy. High doses of vitamin B_6 are recommended for neurotoxic effects.

Labour expertise. The onset of peripheral neuropathy is an indication of termination of contact with the toxic noxa - full recovery is expected in treatment. In pseudotabes forms, recovery is slow

and sometimes impossible - a permanent loss of working capacity is determined.

Prevention. Process sealing, ventilation, obligatory wearing of personal protective equipment. Do not work with acrylamide persons with neurological, skin and allergic diseases. Periodic examinations are performed once a year with the participation of a neurologist and dermatologist. If possible, EMG monitoring of peripheral nerves is performed. Preventive-professional nutrition - diet D, vitaminoprophylaxis.

3.3.2. ACRYLONITRILE - T. KUNEVA

Acute poisoning

- nervousness, irritability of UAP, nausea, vertigo,
- convulsions, coma, death

Chronic poisoning

- nausea, headache, vertigo, easy fatigue
- increased risk of lung cancer

Acrylonitrile (AN) is a volatile colourless liquid with characteristic smell that can be detected at 20 ppm. It has the property of polymerizing itself (polymerization can be inhibited by the addition of alkaline substances - used to store it). Dissolve well in organic solvents.

AN's vapors are explosive and highly flammable in the range of 3 to 17% - hydrogen cyanide may be released during combustion.

***Usage.** AN is the starting material for the synthesis of polyacrylnitrile fibers. It is used in the production of synthetic rubber, acrylamide, acrylic acid, paints and medicines. Acrylonitrile-containing plastics, especially acrylonitrile-butadiene-styrene rubber and tirenacrylonitrile, are used in fitting tubes for automotive engine parts. Due to the resistance of nitrile elastomers to oils they are used in the petrochemical and automotive industries.*

***Biotransformation. Metabolism.** The primary route of penetration of AN is the respiratory. Liquid acrylonitrile penetrates well through healthy skin and especially through damaged skin and mucous membranes.*

The mechanism of action of acrylonitrile has not yet been elucidated. Based on the observed increased cyanide content in the blood and urine, the presence of cyanemethaemoglobin, the clinical and pathohistological similarities with the cyanide poisoning, it is assumed that acrylonitrile acts to release a cyanide that determines its toxicity. The hypothesis is that the main action of AN is through its whole molecule: blocking the active centers and groups of vital enzymes; cyanide release in response of AN and cytochromosadase; reversible suppression of the activity of the respiratory ferments; total blockage of cell metabolism.

The acute toxicity of AN is believed to be due to the action of cyanide and thiocyanates and its mutagenic effect to glycidonitrile (a reactive metabolite capable of alkylating macromolecules).

Clinical picture. The most common symptoms of **acute poisoning** are upper respiratory tract irritation, respiratory distress, nausea, cyanosis, vertigo, disorientation, weakness in the extremities, collapse, convulsions, cardiac arrest.

Continuous inhalation of acrylonitrile vapors produces atrophic rhinitis, sometimes a vocal cords paresis. The majority of workers exposed to AN have complaints of easy fatigue, weakness, headache, dizziness, drowsiness, irritability, lack of appetite, pain in the heart zone. It is often diagnosed asheno-vegetative or neurasthenic syndrom. There are electrocardiographic changes and, in some cases, myocardial dystrophy. Disorders of the gastrointestinal tract, liver function, vestibular disorders are noted. In contact on liquid acrylonitrile on the skin, chemical burn is developing. Especially serious are the damage to the conjunctivae, sometimes with severe corneal damage. Epidemiological studies have shown that AN is associated with an increase in the risk of lung cancer and colon cancer with a latency period of 20 years and is considered a probable carcinogen for humans - IARC, group A2.

Clinical-laboratory tests. Chromosomal assays at acrylonitrile exposed there are no increase in aberrations. In acute intoxication, an increase in the serum level of cyanide or thiocyanates in the urine may be found. Exposure test - thiocyanates below 2.5 mg/g creatinine.

Differential diagnosis. Poisoning with cyanide compounds, methanol, acrylamide, toluene, styrene, carbon tetrachloride.

Treatment. Bleeding out and oxygen therapy. Upon oral ingestion, a stomach wash with water, medical charcoal and 1% sodium hyposulphate solution.

Depending on the available complications, glucose solutions, calcium preparations, B and C vitamins are administered in large doses. Antidote treatment (CN radicals-rhodanates) does not apply.

Expertise on ability to work. In the case of non-curable skin injuries, it is necessary change of work that there is no contact with allergens.

Prevention. Safe technologies, effective ventilation, PPE.

Medical prophylaxis. Persons with chronic CNS disorders, mental, endocrine, pulmonary, allergic and cardiovascular diseases are not allowed to work with acrylonitrile. Periodic medical examinations are conducted once a year with a therapist and neurologist involved, and include full blood counts, a general urine study, and urinalysis of acrylonitrile and

thiocyanates. Skin-allergy testing with acrylonitrile is desirable.

It is recommended that diet D from the protective professional nutrition.

3.3.3. STYRENE - T. KUNEVA

Acute poisoning

- Irritative manifestations of the eyes, skin and UAP

Chronic poisoning

- Headache, weakness, fatigue, dizziness
- Uncritical behaviour, distorted coordination
- Cognitive and behavioural changes
- Personality changes
- Memory loss
- Hepatic dysfunction - jaundice, hepatomegaly, carcinogen

Styrene is a colorless volatile liquid with a sweet smell.

Usage. *The main application of styrene - about 90% of the total production - is to produce polystyrene (3rd place in the total polymer production after polyethylene and polyvinyl chloride). Styrene is also used as a copolymer in the production of polyester resins and synthetic rubber, as well as paints and varnishes.*

Possibility of poisoning with styrene exists in workers engaged in synthesis and polymerization in the manufacture of plastics, rubber, fiberglass, paints.

Biotransformation. Pathogenesis. *Styrene penetrates into the human body mainly via the airway, less often through the skin. Its distribution in organs is relatively even, accumulation is only seen in fat tissue. Styrene is metabolised in the liver to mandelic acid and phenylglyoxalic acid excreted in the urine.*

The toxic action of styrene is similar to benzene. Styrene has a more irritating effect on the skin and mucous membranes, but has a lesser effect on hematopoiesis and has a lower narcotic effect than benzene.

Clinical picture.

Acute poisoning occurs relatively rarely, due to the highly irritating action of the styrene on the mucous membranes.

Concentrations between 100 ppm and 200 ppm styrene elicit inflammatory changes in eyes and adenocarcinoma. Prolonged contact may cause skin irritation and dermatitis and increase skin absorption.

Inhalation of styrene vapors causes irritation in the throat, eyes, nose, metallic taste in the mouth, nausea, vomiting, dizziness, headache, apathy, drowsiness, and at high concentrations - disturbance in equilibrium, weakened concentration, reduction of reaction time. Parenchymal organs are not damaged.

Chronic intoxications. The most common manifestations are irritation of the mucosal epithelium (chronic pharyngitis and bronchitis), eye (chronic conjunctivitis), intestinal tract (gastritis). Workers exposed to styrene at concentrations of less than 100 ppm experience complaints of weakness, headache, dizziness, tiredness, memory weakness, delayed reaction time. Prolonged exposure may indicate anxiety, emotional lability, increased excitability, irritability, depression. Objectively, there are changes in the electroencephalogram, delayed peripheral nerve conduction, disturbances in the performance of psychological tests.

It is possible for toxic liver damage - hepatomegaly, increased activity of serum enzymes and serum bile acids.

Dystrophic changes in the myocardium and lowering of blood pressure have been described.

Styrene is a mutagen and can cause chromosomal aberrations in humans. Studies on cancer mortality do not indicate an increased risk of exposure - according to the IARC classification, it is in the B2 group and the styrene oxide - group A2.

Clinical-laboratory tests. Moderate leukopenia with relative lymphocytes, reticulocytosis, mild thrombocytopenia is found in the blood. Elevated total fat, cholesterol, triglycerides; total and direct bilirubin; increases the activity of liver enzymes. Exposure tests are: mandelic and phenylglyoxal acids in the urine - above 200 mg/l and above 20 mg/l, respectively.

Differential diagnosis is made by poisoning with benzene, toluene, ethanol and other organic solvents.

Treatment. Quickly remove from the gassed environment, oxygen therapy. Upon swallowing - gastric lavage with water and medicinal charcoal, liquid paraffin is infused at the end of the lavage. If it gets on the skin - thoroughly wash with water. Local dermatoses are used topically to corticosteroids. Cerebro- and pulmoprotective agents.

Chronic obstruction is recommended for hepatoprotective agents and vitaminotherapy. Balneotherapy.

Labour expertise. Depending on the degree of intoxication, temporary or permanent rehabilitation without contact with toxic substances. For pronounced forms of toxic hepatitis and encephalopathy, retirement by determining the disability group.

Prevention. Because of the high risk of ignition, complete automation of production processes, effective ventilation, PPE - with appropriate gloves, shoes, aprons and safety glasses is recommended.

Medical prophylaxis. Contraindications for getting into contact with styrene are chronic diseases of the heart and lungs, gastrointestinal tract, liver

and kidney, organic diseases of the nervous system.

Periodic medical examinations are conducted once a year with therapist, dermatologist and otorhinolaryngologist. Paraclinic studies include the determination of mandelic and phenylglyoxalic acid in urine.

It is recommended that diet D from the protective and professional nutrition.

3.3.4. VINYLCHLORIDE - V. PETKOVA

Vinyl chloride (CH_2 = CHCL) is also known as chloroethane, monochloroethylene, vinyl chloride monomer. Possibilities for poisoning with VC monomers exist in the production and subsequent thermal treatment of polyvinyl chloride - plastic, which is extremely widespread for household and domestic purposes. The highest risk of poisoning is the cleaners of the reactors in which the polymerization of VC is carried out.

Biotransformation. *VC enters the body mainly through the respiratory organs. In the process of biotransformation, metabolites - chloroacetaldehyde, vinylchloridedepoxide, glycolaldehyde, which have significantly higher toxicity than VC, are formed.*

Part of the inhaled VC is unchanged with exhaled air - at high concentrations in the air - up to 90%, at low - 12-15%. Absorbed VC is excreted in the urine, partially unchanged, but mainly in the form of metabolites.

Pathogenesis. *The toxic effect of VC is due to the direct action of its metabolites on cellular proteins and DNA as well as on some redox systems - it reduces the amount of glutathione in the liver, link also with cysteine.*

Clinical picture.

Acute poisoning. At high concentrations, VC has a pronounced narcotic effect.

Chronic poisoning. Chronic vinyl chloride intoxication is demonstrated with peripheral angiodystonia and relative typing manifestations of Rayno's disease-type angiospasm, against the background of neurosis-like conditions. In the subsequent heavier stages, sclerodermic skin changes, osteoporosis and acrosteolysis, as well as bone deformations of distal phalanges of the fingers lung affect and thickening of the nails in the form of a clock glass are observed. The clinical picture is complemented by the development of toxic hepatitis or lung affect - toxic pneumofibrosis, obstructive conditions.

The characteristic combination of syndromes in the unfolded clinical stage of poisoning has given rise to a terminological differentiation of a disease unit - **vinyl chloride disease**.

Vinyl chloride is among the humanly proven carcinogens (IARC classification 1 group). Characteristic of the carcinogenic effect of VC is lung cancer, especially angiosarcoma of the liver, which can develop after a prolonged latency period of 15 to 40 years.

Reproduction effects are also described - an increased incidence of spontaneous abortions and congenital abnormalities of the CNS. A higher incidence of arterial hypertension, headache complaints, and tiredness were also found in workers.

Clinical-laboratory tests. Leukopenia or thrombocytopenia is observed in peripheral blood. In exhaled air, VC can be defined as an exposure test. The content of one of the final metabolites of VC-thiodiglycolic acid in the urine is also an exposure test - above 5 ppm. Elevated ASAT, ALAT, LDH, bound bilirubin in the blood.

Differential diagnosis. Raynaud's disease, scleroderma, vibration sickness, poisonings with trichlorethylene and ketones.

Treatment. Treatment is symptomatic of acute poisoning. At Raynaud syndrome and vascular dystonia syndrome use vasodilators and pain allaying drugs, vitaminotherapy. Physiotherapy.

Prognosis and labour expertise. In the initial stages of poisoning, the clinical symptoms are reversible, but regardless of the stage, vinyl chloride disease is a reason for permanent cessation of professional contact with VC. Clinical symptoms may also progress after discontinuation of exposure.

Prevention. Sealing of production processes, efficient ventilation. Persons with pronounced arterial hypertension, liver pathology, chronic lung disease and diabetes are not allowed to work with VC. Prophylactic examinations are performed once a year with the participation of a neurologist, internist and ophthalmologist and examination of platelet and leukocyte counts as well as the activity of ASAT and ALAT. For monitoring of exposure - periodic determination of thiodiglycolic acid in the urine.

3.3.5. ETHYLENE - T. KUNEVA

Acute poisoning - Headache, asthenia, arrhythmia, coma

Chronic poisoning - Toxic cerebrovascular syndrome, autonomic dysfunction.

Ethylene is a monomer used to make vinyl chloride, polyethylene resin and plastics. It's used in surgeon-for-narcosis.

Ethylene penetrates the body through the airway and eliminates unchanged by breathing air. It is a classic drug, a potential carcinogen.

Clinical picture.

Acute poisoning occurs as a narcosis but lacks the initial stage of excitement. At high concentrations, headaches, asthenia, temporary loss of consciousness, arrhythmia, breathing disturbances,

coma are observed.

Chronic poisoning occurs in the form of neuroso similar (toxical cerebrathenic) syndrome, angiodistonic or angiospastic syndrome and other manifestations of autonomic dysfunction with thermoregulatory, sudomotor and vasomotor disorders, in rare cases hypothalamic syndrome.

Electrocardiographic findings have been reported for ischemic heart disease. Cases of hepatic injury as well as ovarian-menstrual disorders have been observed. There is no proven carcinogenic effect of ethylene and polyethylene in humans and laboratory animals.

Clinical-laboratory tests. Ethylene metabolites have not been identified and there is no specific laboratory finding. A mild anemic syndrome with leucopenia and cytopenia tendency has been reported, increased hepatic enzyme activity and changes in lipid exchange.

Differential diagnosis - Raynaud's syndrome, autonomic poly-neuropathy in vinylchloride intoxications, endocrine diseases, methane, acetone, ethanol, gasoline, etc. intoxications.

Treatment - symptomatic. In toxic hepatitis - hepatoprotective agents, dietary nutrition.

In polyneuropathy - peripheral vasodilators: non-steroid anti-inflammatory agents; analgesics; sedatives and mild antidepressants, vitamin B and vitamin E.

Labour performance. Pregnant women are labour readjustment. In the development of autonomous polyneuropathy, temporary labour is also required without contact with toxic substances and heavy physical labour.

Medical prophylaxis. Contraindications for contact with ethylene are diseases of the nervous, endocrine, cardiovascular system, chronic liver disease, anemia.

Periodic medical examinations are conducted once a year by an internist and a neurologist, at indication - gynecologist. Paraclinic studies include full blood count, liver enzymes, ECG and vegetological tests.

It is recommended that diet D of the professional protective nutrition.

3.3.6. METHYL METHACRYLATE - T. KUNEVA

- Headache, fatigue, irritability
- arterial hypotension, autonomic dysfunction
- rhinitis, bronchitis
- polyneuropathies
- toxic hepatitis

Methyl methacrylate is methyl ester of methacrylic acid. It is a colorless, volatile liquid with an unpleasant odor.

Usage. *Methyl methacrylate is a major monomer in the production of acrylics, also used in the production of organic glass (plexiglas), optical lenses, varnishes and resins, displays ,for the production of light constructions for inscriptions, windows, etc.*

Biotransformation. Pathogenesis. *Enters the body mainly through the respiratory tract and through the skin. Methyl methacrylate is completely hydrolyzed to methyl alcohol and methacrylic acid. In turn, the methyl alcohol is oxidized and form formaldehyde and formic acid. It is eliminated relatively quickly, it has no cumulative effect. Methyl methacrylate and methacrylic acid have irritating and narcotic effects (lipotropic substances), damaging parenchymal organs.*

Clinical picture. The most common symptoms of **acute poisoning** are weakness, nausea, vomiting, headache, tightness in the chest, arterial hypotension, and in more severe cases - loss of consciousness and epileptiform seizures. Usually there are no residual phenomena after acute poisoning.

Leading place in the clinical picture of **chronic poisoning** with methyl methacrylate is the damage to the nervous system. In the initial stage (I), a neurasthenic syndrome with predominance of autonomic disorders is characteristic. Exposed workers complain of fatigue, headache, vertigo, feel faint, irritability, sweating. Polyneuropathies (II) have been diagnosed with polymetacrylate workers over 10 years, as well as functional changes in vision, smell and hearing, vestibular abnormalities, dystrophic bone changes. Patients report memory disorders and character changes. A psycho-organic syndrome may be formed. Possible development of toxic encephalopathy (IIIst).

Dystrophic changes in myocardium and liver (toxic myocarditis and hepatitis) have been reported. Women with disorders in the ovarian-menstrual cycle have been observed.

Methyl methacrylate has a pronounced local action on the skin and mucous membranes. Workers with work experience over 6 months have found subatrophic rhinitis, nasopharyngitis, laryngitis, conjunctivitis, dermatitis, allergic changes (contact dermatitis).

Clinical-laboratory tests. Hypochromic anemia with lymphocytosis and neutropenia, increased hepatic enzyme activity, increased bilirubin are found in the blood. For the exposure test, determination of formaldehyde in the blood - up to 0.2 mmol/l or in the urine: formaldehyde - norm to 0.3 mmol/l; of methane - up to 15 mg/l; of formic acid - up to 80 mg/g creat.

Differential diagnosis. Alcohol poisoning and other organic solvents.

Treatment. Acute poisoning - Immediate removal from the gassed environment, oxygen-therapy, cardiotonics, glucose infusions. In chronic poisoning - vitaminotherapy (B_1, B_6, B_{12}, C); hepatoprotective agents. Symptomatic treatment - balneal physiotherapy. Allergic dermatitis - local corticosteroid ointments.

Labour expertise. In acute and mild chronic poisoning - temporary disability, recovery after treatment. In the diagnosis of polyneuropathies - labour-induced termination of contact with toxic substances. In development of encephalopathy - definition of disability group.

Medical prophylaxis. Contraindications for getting into contact with methyl methacrylate are chronic diseases of the heart and lungs, cardiovascular system, skin, and blood. Periodic medical examinations are performed once every 24 months by a therapist, complete blood count, fluorography. At indications - examinations by neurologist, otolaryngologist and a dermatologist.

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3.4 PESTICIDES

CHEMICAL CLASSIFICATION OF PESTICIDES
(by F. Kaloyanova, 1993)
I. * PHOSPHORRGANIC
II. CHLORORGANIC
IIa. ** MERCURY ORGANIC
III. * CARBAMATES AND TIOCARBAMATES
IV. * DITIO-CARBAMATE
V. TRIASINES
VI. DICHLORPHENOXYACETIC ACID DERIVATIVES
VII. CARBAMIDE DERIVATES
VIII. DERIVATIVES OF ANILIDES AND AMIDES
IX. DINITRO COMPOUNDS
X. * KUMARIN DERIVATIVES
XI. * COPPER PREPARATIONS
XII. SULFUR PREPARATIONS
XIII. BENZOYL-PHENYLUREIC
XIV. DIPYRIDIL DERIVATIVES
XV. PHTALIMIDES
XVI. SULPHATES, SULPHONES AND SULPHONATES
XVII. BENZIMIDAZOLES
XVIII. * SYNTHETIC PYRETROIDES
XIX. TRIAZOLES
XX. PHOSPHIDES
XXI. METHYLBROMIDE
XXII. OTHER DERIVATES

* Pesticides that use antidote therapy

** For mercury pesticides see also mercury intoxications.

3.4.1. CHOLINESTERASE-INHIBITING PESTICIDES - ST. ANDONOVA, R. STEFANOVA

Phosphorus Pesticides

Chemical phosphorus pesticides are derivatives of phosphoric, thiophosphoric, dithiophosphoric and phosphonic acids, which are structurally different compounds with different physicochemical properties and biotransformation. They are poorly resistant to the environment. Depending on the high temperatures, humidity and sunlight, they can be transformed into more toxic substances. They have unpleasant odor and high toxicity to mammals and humans.

Some of the authorized preparations for use in this group are: chlorpyrifosetyl 48%, LD_{50} 135-163 (Argyphos, Dursbane), fenitrothion 50% LD_{50} 250-500 (* Agria-1050), phosalone 35% LD_{50} 135 (* Agria 1060), diacinone 60%, LD_{50} 300-400 (* Agridine, Bazudine), monocotphos 55, 2, LD_{50} 8-23 (* Azodrine), pyrimiphosmethyl 50,5, LD_{50}, 2050 (*Actelic), dimetoat 39%, LD_{50} 320-380 (* Bi58) and many others.*

Usage.

Phosphorus pesticides are mainly used in agriculture as insecticides, some as fungicides and rodenticides. Occupational risk of intoxication exists during production and use in agricultural activities, disinsection and deratization. Acute intoxications with POP are commonly observed in lesions as household or suicidal.

Biotransformation. *They penetrate the respiratory and digestive tracts and through the skin. The major biotransformation of POP occurs in the liver by the monooxidase system (MFO), oxidative desulphurisation, oxidative dealcylation, oxidative dimethylation, nitroreduction, hydrolysis, and other hepatocyte and blood cell enzymes. They are mainly excreted through the kidneys and the intestinal tract. They do not have material accumulation in the human body.*

Pathogenesis. *POP due to structural similarity irreversibly displaces natural substrates in the cholinesterase enzyme, resulting in substantial amounts of acetylcholine being deposited in the synapses of peripheral and central brain structures, effector organs, and blood plasma.*

The clinical picture of acute poisoning is characterized by three major syndromes:

Muscarinic - when reactivating M-cholinoreactive receptors. It is characterized by myosis, bradycardia, increased salivation, sweating, nausea, vomiting, bronchospasm, signs of toxic pulmonary edema, etc.

Nicotine-like - upon reactivation of the N-cholinorenective structures. It occurs with or after muscarinic-like syndrome with muscle weakness, twitching of the eyelids and muscles to severe seizures. Tachycardia, increased blood pressure, and the like may occur.

Comatose syndrome - occurs due to direct effects on the receptors of the central brain structures and occurs with headache, nausea, vomiting and rapid loss of consciousness.

There is a **slight, moderate and severe** degree of acute poisoning with POP, depending on the dose, the physicochemical properties of the compounds, and the degree of blocking of cholinesterase (CE). The lethal outcome occurs as a result of respiratory failure, cardiovascular weakness and toxic encephalopathy. In the course of intoxication, liver damage, toxopulmonites, toxic nephropathy, myocardiopathy, etc. have been observed.

Chronic poisoning. It is observed when the rules for working with production and agricultural activities are no-observed. Neurological symptoms with asheno-vegetative symptoms, neurasthenia, depression and heavier manifestations such as extrapyramidal symptoms, polyneuritis, spastic paralysis, as well as liver damage - from mild functional disorders to chronic hepatitis have been reported. Changes in cardiovascular, digestive and hematopoietic effects can be identified from some POP, irritant and allergic.

Clinical-laboratory tests. Reduction of serum and erythrocyte CE activity, evidence of POP or their metabolites, such as diethylphosphates, dimethylphosphates and others in blood and urine.

There is not always a good correlation between the decrease in CE and the severity of acute poisoning.

Differential diagnosis. It is poisonings with toxic gases, chlorinated hydrocarbons, other pesticides (carbamate, pyrethroids), medications, mushroom intoxications, stroke.

Treatment. It starts with depurjration events and detoxical agents to the front door. An agent of choice in the treatment of acute poisoning is the administration of Atropinum sulfurium (ampoules of 1 mg 1 ml). For severe and moderate poisoning, start with 1-3 mg intravenously and then 15 minutes or 30 minutes until an atropine effect is obtained, then dose is reduced accordingly and the increase in CE in the following days. After atropine is used primarily in the first 24 hours of acute poisoning so-called CE reactivators (Toxogonin 250 mg 1 ml of Obidoxim 500 mg 5 ml in a dose of 1 ampoule intravenously over 2 hours in severe intoxications or 4x2 amps, 3x2 amps intravenously in water-saline solutions Also using early carbohemorperfusion, complex detoxical, resuscitational and organoprotective therapy

The prognosis is unfavorable for severe acute poisoning and chronic with organic damage

Prevention. Use of PPE No labour with POP for persons with CNS, endocrine, liver and kidney diseases, chronic skin injures Periodic medical examinations - once every 12 months with the participation of a therapist and neurologist, if necessary a dermatologist and ophthalmologist and mandatory control of CEA.

CARBAMATE PESTICIDES

These are widely used plant protection agents such as fungicides, insecticides, herbicides, nematodes and oth. They are derived from carbamic acid - carbaryl 85%, LD_{50} 850 (Sevin, Dicarbam NP 85), pyrimicarb 50%, LD_{50} 147 (pyrimor 50 BP), propamocarbhydro chloride, LD_{50} 8600 (*Previcur), benomyl 50%, LD_{50} 9000 (* Benileat 50 NP, Fundazol 50 NP), Etiofencarb 0.50%, LD_{50} 411 (* Kronstone), metomyl 90% 20% , LD_{50} 17-24 (* Lanat 20 EC, Lanat 90 NP, Nudrin 20 EC) and the like, of thiocarbamic acid - cyclost 720 g, LD_{50} 2710 (* Ronit), ethyl dipropylthiocarbamate 720 g, LD_{50} 2550, (*Bradican 6E, Eradican-extra, Bitam EC), molinate 720 g, LD_{50} 369 (ORDAM 6E, Hydram, Yalan), and others, and dithiocarbamic acid-tiram 50.85%, LD_{50} 865 (* TMTD, Wolffentiuram), 35% aluminum ethylene-bis-dithiocarbamate, LD_{50} 5200 (* AMBIS), zinc ethylenebis dithiocarbamate 75%, LD_{50} 5200 (*Cineb, perocin), mancoceb 80%, LD_{50} 8000 (* Dithane M45, Pencoceb 80 BP), methyl thiofanate 40%, 70%, LD_{50} 600, (*Metyl-topsin ULV, Metyl topsin 70VP) thiodicarb 37.5% , LD_{50} 66 (* Larwin), and others. Most of them are highly toxic to insects and low and middle toxic to humans. They have environmental sustainability and only some have material accumulation in the animal and human body.*

Occupational risk. *Agricultural workers working with pesticides of this group in the production of carbamates, tranquilizers, hypnotics, etc.*

Biotransformation. *The primary route of penetration is the airways, less skin, and household intoxications - the digestive tract. Biotransformation occurs in the liver directly from esterases or by oxidative metabolism of the carbamate group with the phenol group release. Excretion from the body is through the urine. In some of them at the kidneys, mainly a-naphthol and sulphates are excreted.*

Pathogenesis. *Toxic action of carbamates is based on the binding of enzyme systems based on cholinesterase. Unlike POP, it is a competitive reversible inhibition of CE by carbamic acid and inhibition of DFS from dithiocarbamates. The latter are degraded to ethylenethiourea.*

The clinical picture of acute poisoning with carbamate pesticides is characterized by signs of muscarin-like and nicotine-like syndrome, as with poisoning with POP. Cholinesterase activity is less pronounced and rapidly recovers after treatment. At intoxications with thio- and dithio carbamic acid derivatives, headache, nausea, ataxia, irritation of the respiratory tract mucosa, and severe constipation with severe intoxications are observed. In alcohol use, an antabuse effect appears - carbamates reduce tolerance to alcohol. Various clinical manifestations of allergy have also been observed.

Chronic intoxications are rare and occur with neurotoxic effects, thyroid dysfunction, hematopoietic and hepatic function disorders. Experimentally, mutagenic, embryotoxic, teratogenic and possibly carcinogenic effects of carbamates have been demonstrated - mainly due to ethylene thiourea.

Clinical-laboratory tests. Proving of carbamates, their metabolites - α-naphthol and sulphates, in

blood and urine, anemia, thrombocytopenia, methaemoglobinaemia. **Differential diagnosis** is made with POP poisoning, synthetic pyrethroid contact dermatitis of other origins (drugs, toxic substances).

Treatment. Carbamate compounds that block cholinesterase carry out with Atropinum sulfurium (1 mg, 1 ml) intravenously, intramuscularly or subcutaneously in CEA monitoring, symptomatic therapy, general detoxical and depourging means. No cholinesterase activators are administered! The prognosis is usually favorable.

Prevention. Use of PPE. Medical prophylaxis - as with POP.

3.4.2. CHLORORGANIC PESTICIDES
V. PETKOVA, ST. ANDONOVA

Chlorinated pesticides (COPs) are chlorine derivatives of hydrocarbons - predominantly cycloparaffins (hexachlorocyclohexane and its gamma isomer - lindane, hexachlorocyclopentane) and multi-nuclear (dichlorodiphenyltrichloroethane - DDT, chlorandan, heptachlor, dieldrin, endrin).

These are a large group of plant protection agents that are used as acaricides (celtane, tetranidide, pentak, milball), such as insecticides (thiodane, thionex) and others.

They have an adverse environmental effect because they are persistent in the environment (more than 20 years) and also accumulate in the human body. These unfavorable properties warranted the use of very effective and widely used preparations such as DDT, aldrin, lindane, heptachlor and others. Poisoning can be expected during production, packaging and use, as well as in constant contact with materials treated with COP.

***Biotransformations:** COPs enter the body through the respiratory organs, the skin and the gastrointestinal tract. Due to their affinity for lipids, chlor-organic compounds, respectively their metabolites accumulate in organs and tissues rich in lipids - adipose tissue, hemato-poietic and nerve structures. They also damage the liver and kidneys. Removed from the body unchanged (metabolized hard) and a little like metabolites - with faeces and less commonly with urine, the mammary glands. They pass through the hepato-encephalic and hepato-placental barriers.*

***Pathogenesis.** Most of the COPs currently used are moderately toxic. Their cumulative effect determines their chronic toxic action, as well as some remote biological effects (DDT - carcinogenesis, embryotropic action). Toxicity of COP is associated with a cellular metabolism disorder by inhibiting the activity of certain respiratory enzymes, as well as disturbances in the cell membrane lipid structure.*

Clinical picture.

Acute poisoning with COP has a higher significance in case of household accidents and represents a synthesized type of symptom of chronic poisoning. Upon oral ingestion, poisoning may occur with severe gastroenteritis and subsequent general resorbtion action of the drug with severe neurological symptoms - seizures, ascending bulbar paralysis and coma.

Chronic poisoning. Chronic poisoning is often occupational, but it is also seen in the household (especially sensitive to the toxic effects of COPs are children and adults).

COPs are classical polytropic poisons with marked polymorphism of organ damage.

In its initial **mild stages**, the poisoning is manifested by functional disorders of the central nervous system with vegetative dystonia - neurosis similar conditions with headache, insomnia, vertigo, adynamia, irritability, emotional lability, functional liver disorders, mild haematological abnormalities - leukocytosis or leucopenia, eosinopenia.

In the more advanced - **medium and severe forms** of poisoning, there is a worsening of the symptomatology with a clear outline of the organ pathology - toxic hepatitis, toxic vegeto-sensory polyneuritis, toxic encephalopathy and encephalo-polyradiculoneuropathy in severe cases. The latter manifests the characteristic myelotoxic activity, which is most commonly associated with diene COP - aldrin, dieldrin and others. With prolonged exposition or severe poisoning due to the high cumulativity of COP, hypo- and aplastic anemia, agranulocytosis and pancytopenia may occur.

The clinical picture of poisoning is supplemented by respiratory tract damage: chronic rhinopharyngitis, tracheobronchitis, pneumofibrosis. A characteristic feature is the frequent complication of asthmatic conditions. There is also a kidney syndrome - oligo-anuria, acute renal failure. Allergic dermatitis and eczema are also characteristic of the chronic effects of chlor-organic compounds.

Clinical-laboratory tests. Blood leukopenia, thrombocytopenia and hypochromic anemia (respectively the degree of involvement of the haematopoietic apparatus) are detected in the blood. Essential is the COP gas chromatography exam and metabolites in blood and urine.

Differential diagnosis: It is done with other aetiology hemopathies, most often myelodysplastic syndrome, drug leukemia thrombocytopenia, alcoholic, diabetic or para-neoplastic polyneuropathies. Also with poisoning with POP, chlorinated hydrocarbons, toluene and others.

Treatment. There is no antidote therapy, but an-

tioxidants may be recommended, e.g. Tocopherol 0.5% - 2 ml. i.m. Treatment course - 10-20 days.

Per os poisoning - gastric lavage, saline cleansing, hemodialysis.

Organoprotective therapy: glucose solutions, cocarboxylase, nootropic (pyramem 2.4 g/d), central vasodilators (cavinton - 20 mg/d). Treatment course - 30 days. In polyneuritis - pain-allayings, peripheral vasodilators.

In haematological disorders - haemostimulating. Treatment course 15-20 days. Corticosteroids - by scheme, and haemotransfusion - in severe cases.

Labour expertise. Initial and mild forms of poisoning are reversible after treatment and do not require change of work. The prognosis is unfavorable in aplastic states and in the presence of myelopolyneuropathies - they determine longer-term disability and long-term retirement or disability group.

Prevention. Sealing, effective ventilation in the production and preparation of solutions. PPE when in use.

Medical prophylaxis requires that persons with allergic, chronic pulmonary and often recurrent upper respiratory tract inflammation, chronic hepatitis, recurrent inflammatory diseases of the bile and biliary tract, anemia, organic and pronounced functional diseases of the central nervous system.

Periodic medical examinations - once a year with the participation of internist, neurologist and ORL specialist and with tracking the content of COPs in the blood of the working people.

Prophylactic intake of vitamin C (0.5 - 1.0 g daily) or vitamin C treat drinks is recommended, and professional protective nutrition-diet D.

3.4.3. MERCURY PESTICIDES
V. PETKOVA

The organic compounds of mercury - alkyl-, aryl, alkooxy- compounds are the active ingredient of a large and varied group of fungicides (methylmercuryiodinide, ethylmercuric chloride, ethylmercuryamide, methoxymethylmercuric chloride, phenylmercuric chloride, etc.)

Due to the high risk of poisoning among the population and in the workers and the accumulation (and the biological chain) of mercury and methylmercury in the environment, food and in organisms, their use as fungicides for seed treatment is very limited.

Biotransformation. *Mercury compounds enter into the body through the respiratory organs, the skin and the gastrointestinal tract. The availability of a hydrocarbon radical in the molecule determines their lipotropicity and favors the penetration of poison into various tissues of the organism, and the passage of the haematoencephalic barrier, which is associated with their high neurotoxicity. They are found to circulate in the body for some time, then metabolise and as metallic mercury are deposited in some organs (liver, kidney, endocrine glands, etc.) and/or separated from the body.*

Pathogenesis. *The toxicity of these compounds is due to the combination of their particular toxokinetics, remembered that of organic solvents and the affinity of mercury (thiol poison) for sulfhydryl groups of cell proteins and some enzymes.*

Clinical picture.

Acute poisoning. Clinical symptoms of acute poisoning occur rapidly after a short latency period with manifestations of general resorption - toxic cerebrasthenia, and in severe forms - severe disseminated damage of the nervous system with cerebellum (ataxia), diencephal and extrapyramidal symptoms, optic nerve atrophy. Oral administration - metallic taste, hypersalivation, erosive gastritis, and in severe cases - haemorrhagic gastroenteritis with collapse, shock and uremia.

Chronic poisoning. It may occur after a relatively short exposure of 3-4 months. In the early stages of poisoning neurosis-like symptoms with marked vegeto-vascular dysfunction and marked disturbances in the emotional sphere are observed. Neurological pathology is usually accompanied by changes in the oral cavity - gingivitis and hypersalivation as with mercury poisoning. In advanced cases, neurological symptoms have been exacerbated with generalized vegeto-vascular disorders of the diencephal type, vegetosensory polyneuritis, psycho-organic symptomatology and psychoses. The most commonly reported mass poisoning with mercury compounds in the Minimata in 1950 to 1,000 japanese fishermen consuming fish with a high methyl mercury content, and that in Iraq in 1972, affected about 6,000 people (500 deaths) after eating bread, made from seeds treated with mercury pesticides. In both incidents, the poisoning has occurred with the above described disseminated nervous system damage and severe motor and sensory polyneuritis (ataxia, dysarthria, deafness, visual disturbances) and hepatorenal syndrome.

On contact with the skin, they develop allergic and contact dermatitis and eczema.

Differential diagnosis. Myelopolyneuritis with other etiology (infectious, poisoning with arsenic, lead, tetraethyl lead), diencephal syndrome, other types of psychoses.

Treatment. Specific antidote therapy with Unithiol 5% - 5 cm^3. In case of acute poisoning on the 1st day, 1 amp. at 6 hours, and in the next few days at 1 amps. 12 hours to resolve the most acute symptoms. In case of chronic poisoning - 1 amp. daily in a

course of 5 -10 days.

Symptomatic therapy - neuroleptics and tranquilizers, antidepressants with soothing effect. Total strengthening and vitaminotherapy.

Prognosis and labour-expertise. The prognosis is unfavorable for severe forms of poisoning. Exceeding the contact with the noxa does not exclude the progression of clinical symptoms.

Profilaxis. Organic mercury compounds are not allowed for persons with chronic skin diseases, functional and organic diseases of the nervous system, kidney pathology. Periodic medical examinations are conducted once a year and include internal and neurological status and urinary mercury determination. Vitaminoprophylaxis. Protective prefessional nutrition - diet B. Prevention of high skin resorption - use of personal protective equipment.

3.4.4. DIPYRIDYLS - ST. ANDONOVA

The main pesticides in this group are paraquat dichloride 20%, LD_{50} 150 (Gramoxon 20) and diquat 20%, LD_{50} 440 (* Reglone). They are used as herbicide defoliants and disinfectants. They are easily degraded and do not represent environmental pollutants. They have a pronounced toxicity to animals and humans, and therefore the use of Gramoxon 20 is prohibited for use in our country. They enter the body through the respiratory tract, the skin and the digestive tract. They are metabolised in the liver and emitted through the lungs and urine. They have a pronounced irritant effect on the mucous membranes and the skin, causing severe destructive changes especially in the lungs, also in the liver, kidneys and myocardium.*

Clinic of acute poisoning. Inhalation occurs with bronchospasm, toxic pneumonitis, after latency - toxic pulmonary edema. These changes are followed by progressive pulmonary fibrosis, making the prognosis extremely unfavorable. Accepted by digestive tract dipyridyls cause mucosal ulceration, hyperemia and sore throat and abdominal pain. In the coming days, toxic hepatitis and hepatorenal syndrome develop. In cases of severe intoxications, central brain structures were impaired. Poisoning with Reglon can take place with the ileus picture with severe collapse. The skin is hyperemic with changes that resemble burns and blistering.

Chronic effects are not well studied.

Differential diagnosis is done with acute exposure to POP, chlorine, nitrogen oxides, explosive gases, etc.

Treatment - there is no specific therapy, but removal of dipyridyls should be done very quickly, as well as oxygen therapy, bronchodilators, glucocorticosteroids, vitamins, cytostatics (against pneumofibrosis). In case of oral intoxication, stomach lavage, saline laxative, forced diuresis (3-6 l/daily glucose solutions), hemoperfusion.

3.4.5. NITROPHENOLS (DINITRO COMPOUNDS) - V. PETKOVA

The compounds of this group - nitrophenols, 2,4-dinitrophenol, 2,4,6-tri-nitrophenol (picric acid), dinitroorthocresols, etc., are nitrooxy derivatives of aromatic hydrocarbons. They are used in the manufacture of dyes, such as explosives (picric acid) but mainly as insectfungicides in agriculture (dinitroortocresol, dinitrophenol).

***Biotransformation.** Nitrophenol pesticides are introduced into the body through the airway and, to a great extent, through the skin. Metabolized in the liver to aminophenol compounds and excreted through the kidneys. These processes are slow, which determines the significant coefficient of cumulation of these compounds. Daily toxic intake of small quantities in the body of the worker for one week is reached toxic concentrations in the blood (about 40 µg / g for DNOC).*

***PATHOGENESIS.** The high toxicity of nitrophenol compounds is due to their ability to block oxidative phosphorylation in mitochondria, which prevents the use of highly energetic ATP compounds. There is a sharp increase in aerobic glycolysis, hyperglycemia, glycogen depletion in the liver and muscles, heavily increased heat production.*

Clinical picture.

Acute intoxications occur with the clinical picture of heat stroke with hyperthermia up to 40 °, thirst, headache, skin hyperemia and conjunctivitis, and in severe cases - hepatorenal and pulmonocardial syndrome and pronounced neurological symptoms - delirium, convulsions, coma.

Chronic poisoning occurs with progressive adynamia, vertigo, headache, and anorexia, characteristic for toxic cerebrovascular disease. Distinctive signs are manifestations of hyperthyroidism - weight reduction, subfebrility, hyperhidrosis, rhythm disorders. In severe forms of poisoning, toxic hepatitis and toxic nephropathy are observed. Particularly for the chronic effects of nitrophenol compounds is the yellow coloration of the skin, nails, hair and conjunctiva. Other skin changes are also known - contact dermatitis with papulo-vesicular rash.

An isolated occurrence of chronic poisoning is the nitrophenol cataract.

Clinical-laboratory tests. Acute poisons in the blood reveal non-specific changes - decreased activity of catalase and peroxidase, increased lactic acid content. Heinz and methaemoglobin bodies are found. In more severe forms, electrolyte disor-

ders, hyperglycemia, albuminuria, acetonuria are observed.

Chronic intoxications - increased thyroid hormone activity, hyperchromic anemia, Heinz body and methemoglobin, hyperglycemia.

The presence of nitrophenol compounds and their metabolites in the blood and less frequently in the urine (there is also functional accumulation where pronounced symptoms of poisoning can also be observed with low NF content).

Differential diagnosis. Consider hyperthyroidism, diencephalosis, viral hepatitis, neurasthenia with pronounced vegetative disorders and sympathicotonia.

Treatment. The first medical assistance in acute poisoning is like a heat stroke. To suppress pathologically enhanced catabolic processes, thiouracil (thymidazole) preparations are administered in small doses with concomitant cardiocircular and infusion resuscitation with glucose and saline solutions.

Organoprotective treatment - cardiotonics, peripheral vasotonics, hepatoprotectives. Barbiturates and acetyzal are contraindicated.

The treatment of chronic poisoning is symptomatic when the noxe is discontinued.

Labour expertise. Chronic poisoning with NF compounds is reversible and does not require long-term rehabilitation except in the case of toxic hepatitis and cataracts. In acute poisoning (severe cases) the prognosis is bad.

Prevention. The use of PPE is mandatory (high dermal absorption).

People with skin diseases, chronic respiratory pathology, blood, liver, and thyroid diseases are not allowed to work. Periodic medical examinations are aimed at timely detection of toxic nitrophenol cataracts and toxic hepatitis. Exposure control is monitored (blood NF monitoring). Vitaminoprophylaxis - vit. C - 0.5-1 g daily.

3.4.6. PYRETHROIDS - ST. ANDONOVA

Known for centuries as a healing herb, the camomile and its native species contain substances with very good insecticidal action - pyrethrins I-VI (esters of chrysanthemum and pyrethric acid) and others. Although their biological activity is many times less than that of DDT, after the ban on the latter, pyrethrin I became important as a strong natural insecticide. Difficulties in the production of industrial quantities, its instability in light and access to oxygen have led to the synthesis of new species with a stabilized molecule with strong insecticidal activity and low toxicity to mammals and humans. These are cyclopropanecarbonic acid analogs: cyflotrin 2,5%, LD_{50} 500 (Byterode), alphamethrin 10%, LD_{50} 200-858 (* Vaztak 10EK), phenpropratrin 10%, LD_{50} 164 (* Dannitol 10 EK) lambda-diholothrin 2.5% LD_{50} 60 (* Karate 2.5 EK), cypermethrin 40%, LD_{50} 251 (* Ripcord 40 EK, Simbush 25 EK) They degrade quickly in the environment. Slightly volatile and almost insoluble in water.*

***Endangered professional groups** are working in greenhouses, processing of fruit orchards, vineyards, production and storage of pyrethroids.*

***Biotransformation.** The entrance door is the respiratory tract, the digestive system and the skin.*

They have acute oral medium toxicity and a mild stage acute percutaneous. They do not cumulate. Practically non-toxic for birds and very toxic for fish and bees. Toxicity is significantly affected by solvents. Biotransformation in the body primarily destroys the ester bond, as the acids are released as free or bound to glycine or glucuronic acid for 2-4 days. Metabolites with a cyano group are released more slowly, to form thiocyanates.

***Pathogenesis.** It depends on the chemical structure of the preparation. Direct neurotoxic effect, blocking of enzymes such as monoamine oxidase, mild demyelination, activation of nerve mediators (M-cholinorective effects) have been demonstrated.*

Clinic. Acute poisoning occurs with irritative changes of the mucous membranes, such as tearing, sneezing, rhinorrhea, burning sensation of skin and mucose. Severe intoxications are characterized by psychomotor agitation, headaches, numbness of tongue and lips, ataxia, gastroenteritis. For those with muscarinic-like action (Ripcord, Decis) there is increased salivation, myosis, shivering of the limbs, tonic and clonic seizures. In dermal contact - dermatitis with erythema, vesicles, bulls, paresthesias. The outcome of poisoning is usually favorable.

Experimentally, a slight sensitizing effect of pyrethroids has been documented.

Chronic toxicity is rarely seen with irritant effects on the mucous membranes of the eyes and the airways, damage to the peripheral nerves.

Clinical-laboratory tests. Chemical analysis for the presence of pyrethroids and their metabolites in blood and urine at normal serum CE levels.

Treatment. Clean the mucous membranes and skin areas with plenty of water; If ingestion - is oral - gastric lavage. Medication treatment is symptomatic. If necessary (hypersalivation) - moderate atropinization. Detoxic therapy by conventional methods. For paraesthesias - vit.E local.

Prevention. Use of PPE and workwear. Medical prophylaxis. People with organic CNS diseases, endocrine diseases, chronic diseases of the liver and kidneys, chronic skin injuries are not allowed to work. Periodic examinations - once a year, attended by a therapist, neurologist, dermatologist.

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3.5 TOXIC GASES

These are a group of different irritant gases and particles that have a local irritant effect and cause systemic toxicity. They are distinguished in the following groups:

Group	Chemical affiliation
1. Irritating gases	chlorine, ammonia, formaldehyde, sulfur dioxide, ozone, phosgene, nitrogen dioxide, fluorine, hydrogen fluoride
2. Simple asfiksanti	acetylene, argon, ethane, helium, hydrogen, nitrogen, methane, butane, neon, carbon dioxide, combustion gas, propane
3. Systemic toxic	carbon monoxide, hydrogen cyanide, hydrogen sulfide, inhalants, phosphorus organic gases, hydrogen fluoride, fluorine
4. Smoke gases	oxides of sulfur and nitrogen, chlorine, hydrogen chloride, phosgene, hydrogen cyanide, carbon monoxide, isocyanate and the like.

IRRITATING GASES - HALOGEN

	Irritation	Acute poisoning	Late consequences	Chronic poisoning
Chlorine	1-6 mg/m³	laryngitis, tracheobronchitis pneumonia bronchiolitis pulmonary edema	pulmonary fibrosis	conjunctivitis ulceration and perforation of the nasal septum, dermatitis, eczema, "chlorine acne" teeth destruction
Fluorine	30-40 mg/ m³	perf. and ulcers of the nasal septum pharyngolaryngitis, bronchitis bronchiolitis, pulmonary edema rhythm disorders gastroenterocolitis dermatitis, keratoconjunctivitis	pulmonary fibrosis hepatitis	rhyno-pharyngolaryngitis bronchitis, pneumosclerosis, asthma hypotension, heart rhythm and conduction disturbances bone fluorosis-deposition of calcium fluoride dental fluorosis
Bromine	5-23 mg/ m³	conjunctivitis dermatitis pneumonia pulmonary edema	pulmonary fibrosis	conjunctivitis, pharyngitis, bronchitis, dermatitis autonomic dysfunction abnormal thyroid gland function
Iodine	over 1,5 mg/m³	conjunctivitis, dermatitis bronchopneumonia pulmonary edema	pulmonary fibrosis	COPD autonomic dysfunction anemia, methemoglobinemia

BLASTING GAS

	Pathogenesis	Irritation effect	Acute poisoning	Late consequences	Chronic poisoning
Carbone oxide	CoHb hypoxia	100-150 mg/ m³	headache (10 % COHb) dispnea (20% COHb) visual disturbances (30% COHb) collapse, convulsions (40 % COHb) myocardiopathy (50 % COHb) coma myocardial infarction	autonomic dysfunction vestibulopathy hemiparesis epilepsy Parkinson's syndrome psychoses	violation in vision, hearing, balance autonomic dysfunction neuritis, polyneuritis, BVDs angina pectoris, arrhythmia myocardial infarction early atherosclerosis psychoses hypohromic anemia
Nitrogen oxides	methemo-globinemia hypoxemia	100- 250 mg/m³	conjunctivitis cough, dispnea pulmonary edema	pneumosclerosis	rhinopharyngitis chronic bronchitis, pulmonary emphysema psychoses

Sulphur oxides	irritative effects	20-50 mg/m³	conjunctivitis pharyngitis laryngeal edema epistaxis, dyspnea, cyanosis pulmonary edema coma	pneumosclerosis	rhinopharyngitis, epistaxis ulcers in the septum bronchitis with asthma components pneumosclerosis autonomic dysfunction brittleness of teeth, yellow colour, striae on enamel dermatitis, eczema thyroid hypofunction
Hydrogen sulfide	tissue hypoxia	over 6 mg/m³	conjunctivitis, pharyngitis headache, vestibulopathy tachycardia, hypotension pneumonia, pulmonary edema, ataxia, polyneuritis convulsions, coma	paresis psychoses	keratoconjunctivitis rhinopharyngitis, laryngotracheitis bronchitis

3.5.1. CARBON MONOXIDE - ST. ANDONOVA, D. CHARAKCHIEV

Carbon monoxide is released in a number of production processes that are associated with incomplete combustion of carbon-containing substances. These include heavy metallurgy, the chemical industry, blasting activities in the mining, construction, electrical wiring and others. Accumulates in poorly ventilated rooms when working on engines (garages, truck cabs, airplanes, cars, etc.)

Biotransformation. Pathogenesis. *The pathway of penetration in the body is the respiratory system. Carbon monoxide is indifferent to the structures of the bronchopulmonary system. Passed through the alveolar capillary membrane, carbon monoxide quickly and easily binds to the iron of hemoglobin and forms carboxyhemoglobin: $HbO_2 + CO$-$HbCO + O_2$. The dissociation of carboxyhemoglobin is slow, and with these toxic effects the dissociation of oxyhemoglobin is delayed. Resorbed CO also binds to plasma non-hemoglobin iron and affects other iron-containing structures: myoglobin, peroxidases, catalases, cytochromosoxidase, cytochrome C, cytochrome P-450 and others. As a result, severe hypoxic changes occurring direct and indirect to which nerve structures are most sensitive. At CO concentrations in the inhaled air of 4000 mg/m³ and HbCO 60-80%, a sudden death occurs. The skin of the affected persons has a pink to light red color (the color of HbCO). There are punctual haemorrhages, thrombotic changes, dystrophic and necrotic changes in the brain, lung, myocardium, liver, and so on.*

Elimination of CO is primarily by the airways.

Clinical picture.

Acute symptoms occur in three stages:

- mildly - with fatigue, headache, dizziness, noise or deaf ears, nausea, drowsiness, rapid cardiac action, angina pectoris. In the blood, the carboxyhemoglobin content is 20-30%. Complaints continue for 5-6 days.

- moderate-severity - when, in addition to the symptoms described above, there is a loss of consciousness for about 3-4 hours, convulsions and development of pneumonia. Disorders of health status are about 10-15 days. Saturation with HbCO is up to 40%.

- severe stage - coma for several days with breathing disturbance, cardiac activity (tachycardia, extrasystoles), changes in blood pressure with a tendency to increase in the beginning and decrease in deterioration. ECG data on rhythm, conduction disturbances and hypoxia. HbCO in blood is 50-60%. Toxical hepatosis, nephrosis, pancreatic lesions (hyperglycemia) are observed. Restoration is slow with residual encephalopathy.

Chronic intoxication. Mild intoxications occur with astheno vegetative manifestations such as fatigue, dizziness, headache, increased blood pressure.

In moderate poisoning, astheno-vegetative manifestations are more pronounced with evidence of toxic encephalopathy and myocardiopathy with changes in EEG and ECG, after a different period of discontinuation of exposure pass over, whereas in severe cases the symptoms persist and even progress.

Clinical-laboratory tests. Higher amount of carboxyhemoglobin over (5%), compensatory increase in erythrocyte count, hemoglobin count, changes in alkaline-acid balance, and blood-gas analysis parameters with a typical reduction in oxygen in arterial blood.

The differential diagnosis of acute poisoning in impaired consciousness is done with acute intoxications of opiates, cyanide, alcohol, etc. In chronic

intoxications - with lead, freons, chlorinated hydrocarbons, porphyria, ischemic heart disease, etc.

Treatment - in case of acute poisoning, it is necessary to immediately remove from the gassed environment and start oxygen therapy (a mixture of oxygen and carbon dioxide). If possible, hyperbaric oxygenation is performed. Pyramem amp. 20% 5 cm3 i.v. are applied, Centrophenoxin amp. 250 mg i.v. In doses of 4x2 amps. for 24 hours or more depending on the severity of intoxication, group B vitamins, vitamin C, blood transfusion, balneo-, and climato- treatments.

Prevention is mainly related to technical and sanitary-hygienic activities.

Labour expertise. The ability to work is impaired after severe acute and chronic CO poisoning and recurrent acute intoxications.

3.5.2. HYDROGEN CYANIDE AND CYANIDES - V. PETKOVA

Hydrogen cyanide (HCN) may have its own application as a pesticide (rodenticide and insecticide - fumigant), as well as a professional chemical agent as an intermediate in the production of synthetic resins and plastics (polyurethane, acrylonitrile) in coke chemistries, in the production and application of cyanate salts in galvanotechnics and steel, in hardening of metals, gold and silver extraction, etc.

Biotransformation. *The risk of cyanide compounds is due to cyanide, which is released in almost all processes associated with their use. Hydrogen cyanide enters the body through the airways and, more rarely, through the skin. It is partly exhaled unchanged through the lungs. Biotransformation of hydrogen cyanide by binding to SH - groups of glutathione and other proteins results in the formation of classical metabolites of HCN - thiocyanates (rhodanides). They are excreted in urine and faeces.*

Patogenesis. *The high toxicity of cyanide resp. its compounds are due to inhibition of cellular cytochromosadase due to their affinity for the trivalent iron of the enzyme. The toxic effect can also be manifested by the specific thiocyanate inhibitory action on the thyroid gland.*

Clinical picture.

Acute poisoning. In mild cases, the irritant effect of cyanide on the conjunctiva and upper respiratory tract, with signs of muscle weakness, vertigo, headache, nausea, vomiting, is observed. In severe poisoning, clinical symptoms expand with dyspnoea and neurological symptoms - seizures and coma resulting from severe respiratory distress and asphyxia. Also myocardial damage with heart failure, decompensated metabolic acidosis.

Different, so-called - lightning forms of acute poisoning with massive inhalation of HCN with a fatal outcome from momentary breathing paralysis. Distinctive diagnostic features of acute hydrogen cyanide poisoning and its compounds are bright rose skin coloring, reddish venous blood with a specific smell of bitter almonds, and severe metabolic acidosis.

Chronic poisoning. They are rarely observed. Neuro so-similar conditions with pronounced vegeto-vascular dystonia (a tendency to hypotension) are a characteristic clinical manifestation of chronic poisoning. Distinctive symptoms include persistent headache, heart heaviness, insomnia, varying degrees of hypothyroidism with diffuse thyroid enlargement. Toxic hepatitis and cognitive-intellectual disorders at cortex atrophy mark a transition to a heavier stage of chronic poisoning.

Characteristic of the chronic effects of HCN on the skin are erythema, eczema, contact and allergic dermatitis.

Clinical-laboratory tests. In the blood in acute poisoning cyan and thiocyanates above 0.169 μmol/l can be detected. In severe cases - metabolic acidosis. Thiocyanates in the urine are the appropriate test for occupational exposure, but smokers also expect an increased level of thiocyanates, which may lead to a false positive result.

Differential diagnosis. Carbonoxide, hydrogen sulfide and freon poisoning, latent brain circulation deficiency, diencephal syndrome, neurosis, etc.

Treatment. In acute poisoning - cardiocircular and respiratory resuscitation. Hyperbaric oxygenation, Vit. C in large doses, alkalizing solutions with sodium bicarbonate. Antidote treatment: Inhalation of amyl nitrate - 5-8 drops every three minutes; methylene blue 1% in 20-25% Sol. glucosae i.v. or per os; sodium nitrite 1-3% resp. 30-10 cm^3 slow i.v. (forms a methemoglobin that binds and disposes cyanide), with subsequent application in the same needle of sodium hyposulphate - 10% - 10-50 cm^3. $CaNa_2$ EDTA - 2 x 300 mg in Sol. glucosae - 10% i.v. can also be used.

In chronic poisoning, treatment is symptomatic.

Labour expertise. The prognosis for mild forms of acute poisoning is favorable. Severe poisoning can lead to irreversible and disability disorders such as encephalopathy, conduction and rhythm disturbances. With marked manifestations of chronic poisoning such as hypothyroidism, persistent hypotension, as well as toxic dermatitis and eczema, the patients are recruited.

Prevention. Mandatory use of PPE and sanitary-technical measures to limit exposure. HCN is controlled in the air in the working environment and in thiocyanates in the blood and urine.

Medical prophylaxis. Do not allow to work HCN people with skin, respiratory, blood, and expressive vegetative disorders. Periodic medical examinations are performed once a year with the participation of an otorhinolaryngologist, dermatologist and neurologist.

3.5.3. HYDROGEN SULPHIDE - ST. ANDONOVA

Hydrogen sulphide is a colorless gas with the smell of rotten eggs, soluble in water and heavier than air. It is a common toxic factor in the working environment in the production of artificial silk, plastics, petroleum and petroleum production. It is found in sewers, pits, caves, where rotting organic matter.

Biotransformation. *The primary route of penetration is respiratory and less skin. It is rapidly oxidized to sulfuric acid. It is absorbed in the blood. Inhibits cytochrome C-oxidase and other enzyme structures. Compensatory anaerobic metabolism is enhanced - lactic acid accumulates, metabolic acidosis develops. Metabolized by oxidation to thiosulphates and sulphates. There is local and general toxic action. A small portion is eliminated through the lungs, and the rest is metabolites by the urine.*

Clinic of acute poisoning. Mild intoxications are characterized by the irritating effect of the formed sulfuric acid on the mucous membranes of the eyes, the upper and middle airways with the onset of tears, rhinorrhoea, dry cough, burning. Neurotoxic effects such as headache, vomiting, ataxia, polyneuritis are added to the **moderate** ones. Rhythm and conduction myocardial lesions, tightness in the cardiac area, hypotension, tachycardia are observed.

Severe stage-occurring with severe damage to lung structures such as pulmonary edema, toxopulmonite, pneumonia. Neurotoxic manifestations are loss of consciousness, coma with convulsions. Also described is a fulminant form in which death occurs for a few minutes from respiratory center paralysis.

Chronic intoxications are caused by the symptoms described in sulfur dioxide poisoning - chronic toxic bronchitis, chronic gastritis, skin lesions, etc.

Clinical laboratory tests - presence of thiosulfates in the urine and sulfhemoglobin in the blood.

Differential diagnosis - Intoxication with chlorine, nitrous gases, carbon monoxide, hydrogen cyanide.

Treatment. Respiratory and cardiovascular resuscitation in severe cases. Antidote treatment - sodium nitrite 1-3%, 30 or 10 cm3, slowly, intravenously. For neurotoxic effects - Pyramem, Centrophenoxin, B vitamins. Symptomatic treatment.

Prevention. Sanitary and technical prophylaxis and PPE. Medical prophylaxis. Non-admission to work of persons with chronic diseases of the respiratory, digestive and central nervous systems, chronic diseases of the skin and the anterior ocular segment. Reviews once a year, attended by a therapist, ophthalmologist, neurologist.

3.5.4. ARSINE (HYDROGEN ARSENIDE) - T. KUNEVA

Arsine is a colorless gas, odorless, heavier than air, poorly soluble in water. At room temperature it slowly decomposes (oxidized) to garlic-smelling products that characterize its presence.

Arsine gas is released into the air in the working environment in all cases of metal arsenides interaction with water, humid air or acids. It is engaged in non-ferrous metallurgy (enrichment, melting, electrolysis), galvanizing, microelectronics and others.

Domestic intoxications occur in damp rooms painted with arsenic-containing paints.

Biotransformation. Pathogenesis. *Arsine gas enters the body through the airway, dissolves in plasma and is fixed in erythrocytes. It causes hemolysis of erythrocytes and forms methaemoglobin. At erythrocytes destrsction arsenic acid formation occurs, after chronic poisoning. Two forms are present in the body - colloidal arsenic and arsenic acid. Arsenic is found in the kidneys, lungs and liver, in prolonged contact - and in hair and nails. It is separated into urine.*

Clinical picture. In case of **acute poisoning** with arsine, the most characteristic is the development of hemolytic syndrome, methemoglobinemia and extrahepatic jaundice. Some hours after inhalation of high concentrations of arsine gas, unwell, fever, abdominal pain, nausea and eruction of garlic, hematemesis, cyanosis on the face and extremities appear; the skin and skleras turn yellow, later turn red. Hemoglobinemia (the blood turns dark-brown to black) and hemoglobinuria (urine is a wine-red) is observed. Hepatosplenomegaly and acute renal failure (oligo-to anuria and uremia) develop.

In modern working conditions, the most common forms of acute poisoning with arsine are the mild forms, with headaches, general weakness, joint and muscle pain, sub-icterus, mild anemia and haematuria.

Chronic arsine poisoning occurs with headache, vertigo, nausea, eruction with garlic odor, dry mouth, thirst, sometimes vomiting, diarrhea, sore throat, hoarse voice, cough, shortness of breath, edema of the face and eyes, tingling fingers of the toes.

Disorders of the nervous system of arsine are similar to those of chronic arsenic poisoning - pol-

yneuropathy develops. Initially, decreased fingers-sensitivity and weakening of tendon reflexes were observed; in the case of progression-paresis and paralysis with atrophy of musculature. Relatively rare occurrence of taste and smell disturbances, bulbar neuritis; in single cases, acrocynosis and obliterating endarteritis are described.

With chronic effects of arsine, hepatitis, melanodermia and hyperkeratosis are more common.

Clinical-laboratory tests. In the blood there is a decrease in hemoglobin and erythrocytes, there is methemoglobin, in urine - albumin. Exposure test uses urinary arsenic determination - norm to 0.13 μmol/l.

Differential diagnosis is made by poisoning with hydrogen cyanide, hydrogen phosphorus, nitrogen oxydes, nitro- and amino derivatives of benzene.

Treatment. In acute poisoning to limit acute haemolysis, immediate administration of Urbason 2 ampoules of 80 mg intravenous is required. In case of haemolysis, a partial transfusion with fresh (24 hour) blood is shown. At the onset of intoxication, preserved diuresis, with very good effect, the infusions of hydro-electrolyte solutions, 20% glucose and 20% levulous solutions - 1000 ml/day are well known. In respiratory failure - respiratory and cardiocirculatory resuscitation; in anuria - haemo- and peritoneal dialysis.

For chronic intoxications, antidote treatment with BAL (selective agent) or Unithiol - 5% ampoules 5 ml, 1 ampoule daily intramuscularly, 5-10 days. Succimer (CHEMET) - capsules 0.350 g - 3x2 daily; course of treatment 5-7 days. $CaNa_2$ EDTA - 10% 10 ml (because of the weak antidote effect only applies in the absence of the above mentioned preparations); one ampoule daily, intravenously with glucose or saline - three days.

Symptomatic treatment.

Vitaminotherapy - vit B_1, B_6 and B_{12}, vit C, vit. A

Labour expertise. In acute poisoning the course is severe, with high lethality. Chronic toxic hepatitis or polyneuritis requires continued treatment and discontinuation of contact with toxic substances. Depending on the lost working capacity, a disability group is defined.

Prevention. Sanitary-technical prophylaxis. Contraindications to work are chronic diseases of the lungs, liver, kidneys, blood, nervous system, optic nerve.

Periodic medical examinations are performed once a year by a therapist, an otorhinolaryngologist, a dermatologist; at indications - neurologist. Paraclinic studies include full blood counts and urinary arsenic determination.

Diet D is recommended from a preventive professional nutrition.

3.5.5. CHLORINE - ST. ANDONOVA

Chlorine is a yellow-green gas with a specific sharp irritating odor. It is heavier than air, easily water-soluble, with pronounced chemical activity.

Usage. *It is widely used in various branches of industry such as plastics, pesticides, petroleum refining, hydrochloric acid, paper-pulp industry, household chemistry, water chlorination, etc.*

Biotransformation. *The entrance door has breathing paths and damage to the skin in the production and digestive tract as of life toxications. With water and biological fluids, it quickly forms hydrochloric acid and hypochlorite, at resulting in the dissociation of them latter to give of nascent oxygen (O). Disorders, from irritant to necrotic action on mucous membranes (ocular, respiratory and alveoli) and skin are in most cases severe and determine the clinic.*

Clinical picture.

Acute poisoning - mildness. It is characterized by burning in the eyes, tearing, nasal and throat irritation with rhinorrhea, dry cough, mild bronchospasm. Objectively, conjunctival hyperaemia, nasal mucosa and throat, larynx, separate dry whistling wheezes are detected.

Moderate-severity - Pain and burning of damaged mucous membranes are more pronounced, coughing is painful with severe dyspnoea. Extremely dry whistling wheezes. Very quickly, bronchopneumonia and cardiac weakness may develop. Mucosal hyperaemia is very pronounced.

Severe stage - which can be characterized by acute asphyxia and death, or severe toxic bronchitis, bronchiolitis, pulmonary edema with subsequent inflammatory changes.

Diagnosis is based on data on industrial accident and leakage of chlorine or household use of chlorine-containing preparations, clinical picture, ABB data for acidosis, hyper lung markings, lung congestion, and inflammatory changes in X-ray examination. After acute poisoning of moderate or sever stage, chronic rhinitis, pharyngitis, tracheobronchitis, bronchiectasis, pulmofibrosis, pulmonary emphysema may occur.

Differential diagnosis. Poisoning with paraquat, dimethylsulfate, ammonia, hydrogen fluoride

Chronic poisoning. Cough, fatigue, burning in the eyes, nose, throat, clinical data on chronic bronchitis and pulmonary fibrosis, chest tightness are characterized. There are chronic dermatitis and eczema, sometimes chlorine acne. On the side of the oral cavity, the teeth and the cavity - hyperemia,

cariousness, periodontal disease, anemic states are described.

Treatment. In case of acute poisoning, ensure airway passage, oxygen therapy and, if necessary, mechanical ventilation. Medication with bronchodilators, broncholitics, cough soothing remedies. In inflammation - chemotherapeutics, antibiotics. Wash eyes thoroughly with water or physiological solution (consultation with an ophthalmologist). In the case of skin burns, the clothes are removed, the patient bathes with plenty of water, treatment of damaged skin and TAP insertion.

Treatment for chronic poisoning is dependent on organ damage and its severity. Depending on the severity of the injuries, the working capacity may be distorted to varying degrees.

Prevention. Sanitary-technical prophylaxis. Medical prophylaxis - persons with chronic diseases of the respiratory, cardiovascular and central nervous systems, the skin and the anterior ocular segment are not allowed to work. Periodical medical examinations once a year with the participation of a therapist, dentist, ophthalmologist and ORL specialist.

3.5.6. FLUORINE, HYDROGEN FLUORIDE - ST. ANDONOVA

Fluorine is a pale yellowish gas with strong odor, a chemically very active metalloid, a strong oxidant.

Usage. *Hydrogen fluoride (HF), hydrofluoric acid and its compounds (fluorides), fluoroacetic acid and its compounds (fluoroacetates), sodium fluoride for the treatment of dental caries, fluorocarbon (freon) in the refrigeration industry as solvent, production of plastics. Fluorine is also used as a rocket fuel oxidant, atomic industry, uranium production, pesticides, plastics, deratization specimens, dental practice, electro-welding, etc.*

Biotransformation. *The main routes of penetration of the body are respiratory and digestive and, more rarely, through the skin. The damage effect is due to the formation of hydrogen fluoride. In the stomach, hydrofluoric acid salts form with hydrochloric acid also fluorine, which has a rapid resorption. It binds large amounts of calcium and blocks many enzymes (glucose 6-phosphatase, cholinesterase, maltase, etc.), suppresses the release of phosphoric acid, thereby increasing the amount of dextrose diphosphate, pyruvate and lactic acid. Impaired metabolism of fluoroacetic acid leads to the accumulation of fluorcitric acid. These metabolites have a pronounced damaging effect on the central brain structures, the digestive system, the lung, the heart. Fallen on the skin and mucous membranes can cause mild irritation to severe necrotic changes.*

Clinical picture. Acute intoxication - the mild degree of fluorine and hydrogen fluoride intoxication, depending on the entrance door, occurs with coughing, chest pain, mild breathlessness. Per osnausea, vomiting, burning in the stomach, moderate diarrhea occur. The affected skin areas and mucous membranes are red and painful.

Moderate grade - Clinical finding of the lung - pronounced bronchospasm, dyspnoea that occur after a latency period. From the digestive system - abundant vomiting and profuse diarrhea, leading to risky dehydration, hyponatremia, hypokalaemia, hypocalcaemia. Muscle pains and cramps occur. From heart-tachycardia, extrasystolia. Skin lesions are heavier.

Severe degree - From the lung - data on bronchiolitis, pulmonary edema, acute respiratory failure. Deepening the clinical manifestations of the digestive system. Expressed cardiac rhythm disorders, camera fibrillation, shock. By inhalation of large amounts, the death from fluorine and hydrogen fluoride intoxication occurs for a short period of time with clonic seizures, respiratory and cardiovascular failure. Skin lesions are presented with severe necrosis. In cases of severe and moderate intoxications persons, marked pulmonofibrosis, renal and hepatic impairment were observed.

Clinical-laboratory tests. Electrolyte imbalance with elevated hypocalcaemia, toxo-chemical analysis for the presence of fluorine and metabolites.

Differential diagnosis. Acute poisoning with heavy metals, chlorine, phaloid fungal intoxication and others.

Treatment. Depending on the entrance door with depurging and non-specific detoxic combinations, calcium preparations, symptomatic treatment, specific dermatological and ophthalmic treatment.

Chronic poisoning with hydrofluoric acid compounds occurs with a prolonged use of water containing fluoride salts and in industrial exposure.

Chronic intoxication. It runs with significant osteoporosis due to calcium binding. It is more pronounced on the bones of the limbs, but can also be generalized. Yellow-brown teeth coloring, brittleness and shaking of the teeth are observed. From the respiratory system - chronic rhinopharingitis, tracheitis, tracheobronchitis.

From the digestive system - stomach and abdominal pain, nausea and less frequent vomiting.

Symptoms of the central nervous system (headache, vertigo, etc.) and cardiovascular system-hypotension, conduction and rhythm disturbances can be detected. Liver damage is also described.

Diagnosis is based on the occupational history, the presence of fluorine in the toxo-chemical analysis of blood, radiographs for the presence of pro-

nounced osteoporosis, dental examination, and data on hypocalcaemia. Treatment as with acute poisoning with dosage adjustment.

Prevention. Sanitary-technical. Medical prophylaxis - persons with chronic diseases of the CNS, cardiovascular and respiratory systems, skin, liver, oral cavity and bones, the anterior ocular segment, chronic gastritis and ulcerative disease are not allowed to work. Medical examinations - 6-12 months with the participation of therapist, otorhinolaryngologist, a dentist and radiologist. Control of Hb and urinary calcium.

3.5.7. AMMONIA - ST. ANDONOVA

Ammonia is a colorless gas with a strong specific odor and strong alkaline taste. Boils at 33-35 ° C. Easily dissolves in ether, water, alcohol, fat.

Usage. *Ammonia is obtained synthetically and is a major source in the production of explosives and synthetic fibers, soda, nitric acid and nitrogen fertilizers. Used in refrigeration, oil refining, the sugar industry, the textile industry, in the production of mirrors (silver amalgam). It accumulates in septic tanks, meeting in the air of chemical laboratories. Major industrial accidents can also be affected the population close to the industrial site. Along with other industrial gases, toxic pollutants cause a serious ecotoxicological problem.*

Biotransformation. *Ammonia is a typical toxic irritant. It penetrates the body primarily through the respiratory system. Depending on the concentration and exposure, it determines injuries of varying distribution and depth.*

Clinical picture.

Mild acute poisoning manifests acute irritative lesions in the upper respiratory tract, laryngitis.

Moderate forms occur with the clinical picture of toxic tracheobronchitis, toxic pneumonia or pulmonary edema.

High concentrations cause laryngeal spasm, reflexive breathing stoppage, acute hypoxia, convulsions, death.

When an ammonia is applied in high concentrations on the eyes for 5-10 minutes, burning, corneal breakage, blinding can occur. When liquid ammonia falls on the skin, second degree burns develop. Combined lesions are heavier.

Chronic exposure to ammonia is characterized by reduced respiratory mucosal resistance, manifested by frequent bacterial infections, chronic bronchitis, pneumonia - in a significantly higher percentage than those in unexposed. Chronic conjunctivitis, keratitis and dermatitis are also more common.

Differential diagnosis, treatment and prophylaxis - as with chlorine poisoning. The smell of ammonia is equal to or lower than its hygienic norm (MAC - 18 mg/m^3), which greatly reduces the risk of noxious poisoning in the production.

3.5.8. SULFUR GASES - ST. ANDONOVA

Due to the high chemical activity of sulfur, its compounds are widely distributed, as sulfur dioxide, trioxide and hydrogen sulphide are most commonly found in the production environment.

SULFUR DIOXIDE

It is a colorless gas with a sharp, suffocating odor, easily soluble in water. Manufactures where workers most often exposed to sulfur dioxide are non-ferrous and ferrous metallurgy, gas production, machine building, refining of oil, pulp and rubber products, sulfuric acid production, etc.

Biotransformation. *In industrial contact, the primary route of penetration is the airways, less skin and the digestive tract. With biological fluids it forms rapidly sulphurous and later sulfuric acids, therefore irritating effects on the mucous membranes of the eyes, airways manifest themselves early, and at high concentrations, toxopulmonites rapidly develop. Quantities of them passing in blood determines cumulative changes, metabolic acidosis. Biotransformed into sulfates, part of which forms S-cysteine, S-sulfo-glutathione and S-sulfonates, mercapturic acids and their derivatives. Elimination of sulfur dioxide is via the lungs and metabolites of urine.*

Clinical picture of acute poisoning. Inhalation of small amounts results in tearing, rhinorrhoea, dry cough, pain in throat and sternum. In severe poisoning, spasms of laryngeal edema, bronchospasm, toxopulmonite development and pulmonary edema are observed. Running is unfavorable. Liquid sulfur dioxide fallen on the skin has an irritant to a necrotic effect. Inhalation of large quantities leads to a rapid lethal outcome.

Chronic intoxication. It is characterized by burning, pain, secretion from the eyes, upper respiratory tract and esophagus. Astheno-vegetative manifestation is observed. In more pronounced forms, there are chronic toxic bronchitis, often with asthmatic expression. The development of toxic pneumocosclerosis in parallel with bronchitis or alone. Chronic oesophagitis, gastritis is detected. From the oral cavity - increased cariousness, yellowish tooth coloring, stomatitis. On the skin side - dermatitis, eczema.

Clinical and laboratory investigations. Presence of sulphates and other metabolites in the urine, ABB, functional breathing study, X-ray examination of the lungs.

Differential diagnosis. Poisoning with paraquat, dimethylsulfate, ammonia, hydrogen fluoride.

Treatment. In acute poisoning - oxygen therapy

to mechanical ventilation in severe bronchospastic conditions and lung edema. Symptomatic treatment. In the presence of toxic pneumonia the inclusion of antibiotics.

Prevention. Sanitary-technical prophylaxis. Medical prophylaxis. Do not allow persons with chronic diseases of the respiratory and cardiovascular systems, of the skin, of the gastrointestinal tract, of the liver, of the anterior ocular segment. Periodic reviews of 24 months (dentist at 6 months) with the participation of therapist, dermatologist, otorhinolaryngologist, ophthalmologist. A "B" diet of protective professional nutrition is recommended.

3.5.9. NITROGEN OXIDES - T. KUNEVA

- In acute poisoning - conjunctivitis, cough, shortness of breath, pulmonary edema

- In chronic poisoning - bronchitis, emphysema, toxic pneumosclerosis.

Nitrogen oxides are: ***NO, NO_2, N_2O, N_2O_3, N_2O_4 and N_2O_5.*** *The most common in manufacturing and with the greatest medical significance is NO_2.*

Usage. *NO and NO_2 are involved as intermediates in the preparation of nitric acid; NO_2 finds application as a nitrating agent; N_2O is used for narcosis ("laughing gas").*

Nitric gases are released during the synthesis of nitric acid and nitrites as well as in all processes in the chemical industry where nitric acid, nitrites and nitrates are used. Nitrogen oxides are formed in the blasting of explosives, in electrical and acetylene welds, in the celluloid burning, in the production of artificial fertilizers, in the ensilage and storage of cereals, in the operation of internal combustion engines.

Pathogenesis. *Nitric gases penetrate the body through the respiratory tract. They have a pronounced local irritant effect on the mucous membranes of the UAP, vasodilator and narcotic action. They fall into the group of methaemoglobin-forming poisons.*

Clinical picture. Acute poisoning is characterized by several periods:

- period of initial events - a few minutes after contact with high concentrations occurs irritation of the mucous membranes of the eyes and the upper respiratory tract, head-pain and fatigue. Symptoms are usually mildly and rapidly transient.

- a latency period of several hours to one day.

- period of development of bronchial edema - irritable cough and wheezing, shortness of breath. In case of worsening, a high level of dyspnea with cyanosis and coughing of foamy sputum, eventually haemoptoea, occurs. Ausculatory in the lung areas is heard from small to large wet wheezes. Sinus tachycardia, systolic blood pressure is often elevated, at cardiogenic shock-reduced to 100 mm Hg.

- a period of reversal. After severe pulmonary edema, pneumosclerosis may develop as a residual phenomenon.

Chronic poisoning. Occupational contact workers often experience inflammatory changes in the mucosal membranes of UAP, chronic bronchitis, early lung-emphysema. Dyspeptic disorders, metabolic disorders, muscle and heart weakness, hypotension are less common. Single cases of "toxic psychoses" with depressions and hallucinations have been described. At high concentrations, yellow coloration of the hair, nose and fingers, greenish accretions with metallic tint of the teeth, partial tooth decay can be observed.

Clinical-laboratory tests. There is a slight increase in hemoglobin, erythrocyte and reticulocyte counts, leukopenia with relative lymphocytosis, methemoglobinemia.

Differential diagnosis. Intoxication with other irritating gases. In chronic poisoning - with viral and bacterial inflammation of the lungs.

Treatment. In case of acute poisoning, oxygen through a nasal tube or mask to normal arterial oxygen partial pressure, in restless patients sedative agents; diuretics; secretory agents; corticosteroids, antibiotics (to prevent pneumonia); symptomatic means.

In chronic poisoning, treatment is symptomatic; vitaminotherapy.

Expertise on employment. After acute and chronic poisoning, people are temporarily incapacitated. Suspended of contact with toxic substances, dust and unfavorable microclimate are workers after acute contact with pulmonary edema or pneumonia and in recurrent chronic bronchitis, pulmonary emphysema or pneumosclerosis lesions.

Prevention. Sanitary-technical prophylaxis. Medical prophylaxis. Contraindications for exposure to nitrogen oxides include atrophic rhinopharyngitis, chronic lung diseases, the cardiovascular system, the gastrointestinal tract, the liver, the skin and the anterior ocular segment disorders. Periodic medical examinations - once every two years, with the participation of a therapist, an otorhinolaryngologist and a dentist (6 months) - on indications dermatologist, ophthalmologist and a blood sample is investigated. It is recommended that diet B be taken from the preventive professional nutrition.

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3.6 OTHER CHEMICAL COMPOUNDS

3.6.1. ACIDS - S. PAVLOVA

Acute poisoning

- Irritative dermatitis, burning of the skin;
- Upper respiratory tract inflammation, lung edema.

Chronic poisoning

- With hydrofluoric acid-osteosclerosis;
- with nitric acid (nitric oxides) - fibrous obliterating bronchiolitis;
- for chromic acid - nasal ulceration, perforation, skin ulceration.

Usage.

Inorganic acids

Sulfuric acid (H_2SO_4) - *used in the production of fertilizers (superphosphate, ammonium sulphate, etc.); Of soap; In metallurgy of non-ferrous and rare metals; In the manufacture of dyestuffs; in the leather industry (leather tanning); in the metalworking industry (metal grinding); for charging batteries; in the production of a large number of organic substances (acetic acid and tartaric acid, phenols, ethyl ether, various types of esters, etc.); in the production of nitrocellulose, nitroglycerine, nitrobenzene; for the purification of various petroleum fractions and others.*

Nitric acid (HNO_3) - *used in the production of nitrogen fertilizers, explosives, nitrocellulose lacquers, cinema films, in the manufacture of synthetic silk and celluloid, aromatic nitro compounds and a large number of synthetic dyes; in galvanoplasty (gold plating copper and brass); for engraving of metals; in the polygraphic industry. HNO_3 easily forms nitrogen oxides. Its vapours have a similar effect but are more toxic than pure nitrogen dioxide.*

Hydrochloric acid (HCl) - *used in the production of fertilizer; in galvanoplasty; for the production of sugar, organic dyes; in the refining of fats and oils; by tanning of fur and their dyeing; In the textile industry; in photography and more.*

Phosphoric acid (H_3PO_4) - *finds application in the production of phosphorous fertilizers; in the food industry; in the production of cinema films and photoreagents; to obtain tissues with fireproof impregnation and the like.*

Hydrofluoric acid (HF) - *the main use is in the manufacture of creolet, for the production of aluminum; in the production of beryllium, manganese, uranium; when welding with electrodes containing fluorine compounds; in the manufacture of matches, etc.*

Organic acids

Formic acid (H.COOH) - *mainly used in textiles (for wool, silk, cotton) and in the leather industry; in the production of perfumes, soaps, varnishes, etc. H.COOH has the most pronounced toxic action of organic acids.*

Acetic acid (CH_3COOH) - *used in the textile industry; painting; in the manufacture of medicines, paints; in many organic syntheses and more.*

Aliphatic dicarboxylic acids - *maleic, fumaric and adipic are used in the production of synthetic resins, paints, inks, etc.*

Glycolic acid ($CH_2(OH)COOH$) and lactic acid ($CH_3(OH)COOH$) *are widely used in the textile, leather, plastic and food industries.*

Pathogenesis. *In contact with tissues, acids cause coagulation necrosis due to their "drying" effect on proteins. Hydrofluoric acid alone induces liquable necrosis by binding to calcium and magnesium.*

Clinical picture. Acute poisoning in vapor exposure develops irritative to corrosive lesions of the conjunctiva and the mucous membrane of the respiratory tract and chemical pneumonitis. These are manifested by keratoconjunctivitis, sometimes laryngeal edema, tracheobronchitis, bronchopneumonia, pulmonary edema. When on the skin, acids cause chemical burns of varying degrees (I-III), with the development of erythema, vesicles, bulls, ulcerations that reach even the bones when damaged by hydrofluoric acid. All injuries are accompanied by severe pain, eventual shock.

Chronic poisoning

Inorganic acidic

Sulfuric acid. Chronic poisoning is usually combined with the action of sulfur dioxide, hydrochloric acid and nitrogen oxides. Diseases of mucous membranes of the mouth, tooth decay, atrophic changes of upper respiratory tract mucosa, bronchitis, pneumocystis, in some cases bronchial asthma, gastritis, ulcerative disease, etc. are observed. Also skin diseases - dermatitis, paronychia, etc., functional changes of the CNS and of the cardiovascular system are also observed.

Nitric acid. In contact with skin, concentrated HNO_3 causes severe burns, colored in a characteristic yellow color (xantoprotein reaction). Diluted solutions can cause eczema. Inhalation of vapours-chronic bronchitis and relatively mild bronchiolitis with headache, dizziness, tinnitus, drowsiness.

Hydrochloric acid. Chronic exposure causes der-

matitis (to a lesser extent than those in the action of sulfuric and nitric acids); airway catarrhs; tooth decay, which begins with the appearance of brownish spots on their surface; corrosive lesions of the lining of the nose (it is possible to pierce the nasal septum); gastrointestinal disorders.

Phosphoric acid. In contact with skin - dermatitis. The vapours cause atrophic processes of the nasal mucous membranes, which can also lead to a break in the nasal septum. Typical haemorrhages of the nose, nasal and swallow dryness, shaking of the teeth.

Hydrofluoric acid. It acts highly corrosively on the skin, with dermatitis formation II-III degree (even weak solution of HF - 0.03%, acts destructively on the epithelium).

Organic acids

Formic acid - most strongly acting from organic acids. Bleeding, nasal cold, sneezing, sore throat, cough, pain and tightness in the chest, dry mouth and throat, hyperacid gastritis are observed.

Acetic acid - the action of her vapours irritates mucous membranes. Chronic exposure causes diseases of the nose, the nasopharynx, the larynx, as well as conjunctivites and bronchitis.

Clinical-laboratory tests. Non-specific changes in some haematological parameters (hemoglobin, erythrocytes) have been observed. Chronic exposure to HNO_3 increases the level of methaemoglobin in the blood. As a biological marker for the effects of HF, determination of F in urine (mean reference values - 0.5 mg/l) is used. X-ray exploration for interstitial or alveolar edema; blood-gas analysis hypoxia is detected.

Differential diagnosis. Intoxications with dimethyl sulphate, ammonia, chlorine, bromine, sulfur dioxide, ozone, powders, bases, etc. Dermatites degrade from those due to bases, halohydrogens, salts of halohydrogen acids, chromium, arsenic and others.

Treatment. Respiratory and cardiocirculatory resuscitation; pulmoprotective treatment; analgesics and alkali (alkaline bicarbonate) in acidosis; eye and skin wash with physiological serum and ointment, surgical treatment.

Prevention. Sanitary-technical prophylaxis. Personal protective equipment. Medical prophylaxis. Contraindications for admission include: chronic bronchitis, pulmonary emphysema, bronchial asthma, pronounced atrophic rhinopharyngitis, laryngitis, chronic diseases of the anterior ocular segment, skin, liver, caries, disposition, chronic gastritis, ulcerative stomach and duodenum, diseases of the cardiovascular system. Periodic examinations of 24 months by a therapist, an otorhinolaryngologist, a dentist (6 months) - as indicated by a dermatologist and ophthalmologist and includes the following paraclinical studies: blood alkaline-acid state, hemoglobin, urine - total study, ammonia determination, titration acidity and vitamin C in the urine.

It is recommended diet C of the protective professional nutrition.

3.6.2. BASES - T. KUNEVA

- Acute effects - eye and skin burns, irritant changes in the UAP;
- chronic effects - ocular blindness.

The most common inorganic bases are ammonium, sodium and potassium. Many oxides and salts dissolved in water cause toxic lesions similar to inorganic bases-calcium, strontium and barium oxide, ammonium and potassium carbonate (potash), sodium silicate (water glass).

The bases are solid - amorphous or crystalline substances or liquids with strong corrosive action. It is stronger than concentrated acids. By removing water from the tissues, the bases cause denaturation of the proteins and cause deep liquable necrosis.

Usage.

Sodium hydroxide (NaOH - caustic soda)*. In production of caustic soda, chlorine, soap, cellulose and synthetic fibers, explosives, disinfectants, plastics, black pitch distillation products (phenol, naphthalene, etc.). The most common use of NaOH is to neutralize strong acids and dissolve water-insoluble chemicals.*

Calcined soda ($Na_2CO_3.10H_2O$). *Used in the manufacture of glass, paper, laundry powders, soap, ultramarine, bicarbonate and other sodium salts, disinfectants, for bleaching tissues, for the purification of naphtha, it is composed of cooling alkaline solutions for cutting metals as well and in the form of mixtures for the cleaning of deposit from boilers.*

Potassium base (KOH). *It is used for the production of potassium compounds, bases, soap, in laboratory practice.*

Potassium carbonate ($K_2CO_3.2H_2O$ - potash). *It is used in glassware, faience and pharmaceuticals, for saponing, wool dyeing, etc.*

Calcium oxide (CaO - quick lime). *It is used in construction, agriculture, metallurgy, leather and textiles, in the production of synthetic paints, soda and others.*

Pathogenesis. *Bases penetrate most often through the skin, less frequently through the respiratory tracts and through the digestive tract (when used by mistake).*

In production conditions, skin lesions are most often observed. Oral sodium (even minor) administration may result in fatal burns, perforation of the esophagus and stomach.

Clinical picture

Acute poisoning develops upon oral administration of the base. Symptoms are severe, similar to those seen with acid poisoning. Immediately after ingestion of the base, the lips swell, there is burning in the mouth and the gulp, accompanied by plenty of salivation, followed by severe pain, disintegration in swallowing to inability to swallow. A profuse and continuous vomiting begins. Severe bleeding may occur as a result of deep necrosis of affected tissues. Urine volume decreases. There are also general toxical symptoms - somnolence, adynamia, dehydration, hypovolaemia with disorders of the water-electrolyte and alkaline-acid balance (alkalosis). Shock conditions and complications are similar to those with acid poisoning, but they occur more frequently and occur much more severely. *Early complications* are perforation of the esophagus with acute mediastenitis, stomach with acute peritonitis. *Late complications* include scarring and obstruction of the esophagus and pylorus, cachexia, hypoproteinemia and hypovitaminosis.

When inhalation of alkali, laryngeal edema is observed, tracheobronchitis, bronchiolitis, sometimes pneumonia develop.

If there is a direct contact with the skin on a concentrated basis, there are signs of severe chemical burns. Secondary infection is often overlapped. After cure of ulcers, scars are often.

Chronic skin lesions are often. In contact with unconcentrated bases, the skin is irritated and degreased, becomes stiff, dry, gruff, cracks. Dermatitis and eczema develop. Nails darken, break easily and separate from the nail-bed.

Base-to-eye fall causes ulcerative corneal damage, sometimes lesion the iris, vitreous, and retina. Possible occurrence of the blindness.

The prognosis is always serious.

Clinical-laboratory tests. No specific laboratory-diagnostic tests for risk assessment have been developed.

Differential diagnosis. Differentiation from other corrosive chemicals - acids, formalin, mercury and other compounds.

Treatment. Respiratory resuscitation and pulmoprotective treatment; administration of analgesics, antibiotics and glucocorticoids (against scarring); eye and skin wash with physiological serum and spread with corticosteroid and antibiotic creams and ointments; surgical treatment, incl. necrectomy and plasty. Parenteral nutrition with concentrated glucose, plasma, plasma substitutes and mixed nutrition (liquid feed).

Labour expertise. Mostly sicks are temporarily incapacitated - from a few days to several weeks. In the case of severe corrosive lesions with partial or complete obstruction of the esophagus, continuous treatment is required and the persons have a reduced working capacity for a long time. In rare cases (corneal blindness), a disability group is defined.

Prevention. Sanitary-technical prophylaxis and personal protective equipment. Medical prophylaxis. Contra-indications for contact with bases are atrophic rhinopharyngitis and laryngitis, chronic lung diseases (bronchitis, emphysema, asthma, fibrosis, bronchiectasis), chronic liver, heart, skin, antero-ocular segment diseases. Periodic medical examinations are performed once a year, with the participation of therapist and otorhinolaryngologist, at indications - ophthalmologist and dermatologist.

3.6.3. CHLOROMETHYL ETHERS - R. STEFANOVA

Halogenated ethers (monochloromethyl ether - CH_3-O-CH_2Cl and dichloromethyl ether $ClCH_2$-O-CH_2Cl) are colorless liquids, easily volatile at room temperature, with suffocating odor. They are easy to mix with all organic solvents. They are alkylating agents. In water they decompose with separation of formic aldehyde, hydrogen chloride and alcohol.

Usage. *They are used for chloromethylation, such as nitrocellulose paint solvents, for the production of antioxidants, plasticizers and stabilizers in the textile industry, organic synthesis and the production of ion-exchange resins. The thermostable emulsion of polymers containing methyl acrylamide as a binder, can release formaldehyde which, in the presence of chloride, forms chloromethylethers, endangering workers in the production of certain resins and plastics.*

Clinical picture.

Acute poisoning. Chloromethyl ether vapors irritate the eyes, upper respiratory tract and skin. Even low concentrations can cause pulmonary edema and fatal outcome. It is believed that the probable cause is the formation of formaldehyde from chloromethylethers.

Chronic poisoning is characterized by headache, irritation of the eyes and upper respiratory tract, shortness of breath and cough. Affected people can develop chronic bronchitis and emphysema. There have been cases of mass hysteria, bronchial asthma and vestibular disorders.

In the chemical industry, where chloromethylethers are used, three times more frequent lung carcinomas (latent period of 5-25 years) are reported in exposed workers. Mortality is high over 10 years of service.

Clinical-laboratory examinations. Specific exposure tests have not been established. Lung X-ray

and cytological examination of the sputum (screening method) are required to establish pulmonary carcinoma.

Differential diagnosis. In acute poisoning, a differential diagnosis is made with damage from irritating gases (chlorformraldehyde, sulfur dioxide, acetone, acids, etc.). In diagnosed lung carcinoma a differential diagnosis is made with malignancies caused by asbestos, arsenic, chromium, uranium, etc.

Treatment. Eye and skin wash with saline, antibiotic ointments; pulpoprotective treatment; respiratory resuscitation (if necessary).

Labour expertise. In cases of acute poisoning, the incapacity is temporarily lost, and in chronic, with the development of pulmonary damage, the patient is subject to rehabilitation.

Prevention. Sealing of chemical processes, and effective ventilation. Regularly control the concentration of substances in the work environment. Use of personal protective equipment.

Medical prophylaxis. Opposites for work are diseases of the skin, respiratory and nervous systems. The medical examinations should be performed once a year with the participation of a therapist, neurologist and dermatologist. A cytological study of sputum is desirable.

3.6.4. FORMALDEHYDE - R. STEFANOVA

Formaldehyde is a gas with an intense suffocating odor. With air and oxygen it forms explosive mixtures. Polymerizes in paraformaldehyde. Formalin is a 35-40% aqueous solution of formaldehyde.

Usage. *Formaldehyde is used as a feedstock in the chemical industry for the production of phenol formaldehyde resins in the textile, paper, leather and furniture industries. It is an antiseptic and deodorizing agent. Upon heating of the polymers, rapid depolymerization occurs and gaseous formaldehyde is released (even at room temperature).*

Biotransformation. *Formaldehyde penetrates the body through the airway and through the skin. Metabolized to methyl alcohol and formic acid, which are eliminated by the body with exhaled air and urine.*

Clinical picture

Acute poisoning. Formaldehyde causes a highly irritating effect on the eyes, the skin and the respiratory tract. It occurs with tears, thickness, loss of voice, cough, pain and tightness in the chest, difficulty in breathing. At concentrations of 20-50 ppm formaldehyde may cause pulmonary edema. As a residual phenomenon of acute toxicity, toxic neuropathy and toxic optic neuritis may be observed.

Chronic poisoning. They develop with eczematous skin reactions - eyelids, hands and neck. It is possible to provoke allergic contact dermatitis and asthma. Prolonged occupational exposure is associated with astheno vegetative syndrome, facial nerve neuritis, impaired vision, damage to the parenchymal organs and myocardium.

Clinical and laboratory studies. Exposure test is the concentration of formaldehyde in the blood - up to 0.6 mg% (0.198 mmol/l), urine - 10 mg/l.

Indicators for functional status of the liver and kidneys increased activity of the serum enzymes ASAT, ALAT, elevated creatine, etc.) are altered. Partial oxygen pressure in the blood is reduced and acidosis is observed.

Differential diagnosis. In acute poisoning - with intoxications of irritating gases, acids, phenol, chloromethylethers, ethylene oxide, methanol, and in chronic - with non-professional diseases of the skin, nervous, respiratory and digestive system.

Treatment. Therapy is symptomatic, aimed at bronchopulmonary and skin manifestations. Inhalation of alkaline solutions, oxygen therapy, respiratory and cardiovascular agents. Specialized ophthalmic treatment.

Labour expertise. In acute poisoning, working capacity is temporarily lost. In organic changes after acute poisoning and chronic intoxication, the ability to work is permanently limited.

Prevention. Sealing of production processes, effective ventilation and use of individual means of protection. Control of formaldehyde concentrations.

Medical prophylaxis. Conducting medical examinations, once a year, with the participation of an internist, ophthalmologist, neurologist and dermatologist and, if necessary, an allergist. Do not allow persons with disabilities of the respiratory system, skin, nervous system, anterior eye segment, liver and kidneys to work.

3.6.5. SURFACTANTS. R. STEFANOVA

Surfactants are polar organic compounds with an asymmetric molecule. The hydrophilic part may be a sulfate, a carboxyl, a sulfonate, a polyoxyethylene chain, or a nitrogen-containing dipolar group. The hydrophobic part is a branched or straight aliphatic chain of 10-20 carbon atoms, a benzene or naphthalene ring with substituents.

The aqueous solutions of surfactants are of different ionic nature: anionic (a-surfactants) which dissociate with the formation of a negatively charged organic ion and a positive ion of a metallic nature; cationic c-surfactants) - compounds that are dissociated by separation of a positively charged organic ion

and a negatively charged inorganic ion; amphoteric surfactants - with different functional groups (in acid environment they react as cationic and in alkaline - as anionic), non-ionic surfactants - which do not dissociate to ions.

Surfactants alter the surface tension of aqueous solutions, some of them also in oil solutions.

Usage. *They are used as detergents, emulsifiers, ionizers. They come in the form of lotions, shampoos, toothpastes and others. Anionic and non-ionic surfactants are mainly used as washable and soap powder (liquid) detergents. Amphoteric surfactants are used in cosmetics for emulsifiers, children's shampoos and others. Cationic surfactants have utility as bactericidal and insecticidal disinsectants and antistatic agents. Productive exposure is the workers involved in the production, mixing, packaging and storage of surfactants, as well as those using them in auto cosmetics. All who are using them in the household are also at risk.*

Biotransformation. *They most often penetrate the skin through long contact (especially cationic surfactants) and, to a lesser extent, by the respiratory tract. It is possible to penetrate, albeit rarely, through the digestive tract. They accumulate mainly in the liver. They are degraded by beta-oxidation to simpler chemical compounds or non-toxic products, eliminated for 48 to 72 hours, mainly urine and bile, and some of them with exhaled breath. They do not have cumulative properties.*

Pathogenesis. *The toxic effects of surfactants are highly varied. They have a local and general resorption effect. Their irritating and sensitizing effect on the skin has been well studied. This effect is due to some of their ingredients, such as bleach, preservatives and antimicrobials. Some surfactants also have a photo-sensitizing action. Surfactants disrupt lipid balance in the skin, damaging cell membranes and enzyme systems in the epidermis. Anionic, non-ionic and amphoteric surfactants have a less deleterious effect on the skin.*

They have an irritant and allergenic effect on the respiratory system as well. In experimental conditions, their teratogenic and carcinogenic effect has been proven.

Clinical picture.

In case of accidental swallowing of cationic or anionic surfactants, toxic inflammation of the gastrointestinal tract (nausea, vomiting, diarrhea, dull abdominal pain, etc.) is observed.

Irritative changes in the mucous membranes of the eyes and the upper respiratory tract of breath, coughing and burning in the eyes may occur with inhalation of finely divided powders. Skin contact with anionic or cationic surfactants leads to skin erythema, swelling, burning and pain. In skin contact, allergic dermatitis and eczema may occur in some cases.

Workers exposed to high concentrations of surfactants containing proteolytic and other enzymes, under adverse working conditions, suffer from shortness of breath, and some asthma attacks. Dyspnoea occurs a few hours after the end of the day or night. Accompanied by red rashes on the face, neck and hands, also from burning in the eyes and throat, swelling on the eyelids, intolerance to strong light. Allergic reactions of the respiratory system are of a fast type. Direct falling into the eyes of c-surfactants leads to severe inflammatory lesions.

Clinical-laboratory tests. Increased absolute number of eosinophils in the blood, increase in specific antibodies of the type of immunoglobulins (IgG, IgE, etc.) in the blood serum.

In bronchospastic conditions, the external breathing values changes. Skin allergy samples are positive for surfactant.

Differential diagnosis. In acute oral poisoning - with intoxications from medications, from other toxic substances, as well as with unprofessional gastroenterocolitis.

In cases of bronchopulmonary apparatus, skin and eyes - with non-professional diseases of these organs.

Treatment. Upon entry of surfactants in the digestive system, stomach washes and alkalizing beverages are given.

In the case of skin injuries - corticosteroid ointments and others. Irritative and allergic manifestations of the respiratory system are treated symptomatically-bronchodilators, vit. C, calcium preparations, expectorants, and in asthmatic attacks novphilin, ephedrine, dehydrocortisone and aerosol preparations (Alupent, Ventolin, Isoprenaline, etc.) are used. Non-specific desensibilization occurs.

In case of eye damage - plenty of physiological saline, ophthalmic treatment.

Labour expertise. Injuries caused by surfactants are most often related to temporary loss of employment. In case of allergic reactions - interruption of contact with professional allergens.

Prevention. Automation and sealing of production processes, effective ventilation, personal protective equipments, control of surfactant concentrations in the air. Medical prophylaxis includes medical examinations once a year, with the participation of therapist, dermatologist and, if necessary, ophthalmologist and allergist. Contra-indications for work with surfactants are chronic skin, ocular, allergic diseases, respiratory and digestive diseases.

3.6.6. ETHYLENE OXIDE - J. HADJIEVA

Ethylene oxide is a gas which, at a low temperature, turns into a colorless liquid with the smell of ether and bitter taste. Dissolve well in water, ethanol and diethyl ether. It tends to polymerize and to interact with many organic and inorganic substances (alcohols, amines, inorganic chlorides, etc.). Upon heating with water, it forms ethylene glycol.

Usage. *Ethylene oxide is used as an intermediate in the production of various chemical substances (ethylene glycol, diethylene glycol, triethylene glycol, ethanolamine, choline, choline chloride, etc.) as a fungicide, in the medical and dental practice for sterilizing dressings materials, systems and oth.*

Biotransformation. *It penetrates the body through the inhalation route and to a lesser degree through the skin. It is deposited in the spleen, less in the liver, lungs and testicles. Its metabolism is accomplished by hydrolysis processes and by binding to glutathione.*

Pathogenesis. *Protoplasmic poison. It has a strong narcotic effect and expressed general and specific toxicity. It has an allergic and irritant effect. It affects reproduction, has mutagenic and carcinogenic effects.*

Clinical picture.

Acute poisoning is characterized by disorders in the nervous and mental sphere (asthenia, drowsiness, vertigo, heavy pulsatile headache, bradypsychia, delayed movements, hypomimia), irritative manifestations of the upper respiratory tract and lungs, severe dyspnoea and pulmonary edema. In some cases there is a brief loss of consciousness and persistent dyspeptic manifestations. In case of contact with the skin - chemical burn with erythema, edema, bulls. Iritative lesions of the conjunctivae - burning in the eyes, ferythema.

Chronic poisoning. Observed in persons with work experience over 5 years. The lesions of the central and peripheral nervous system are leading. Symptoms develop gradually with initial neurasthenic and vertigo-vascular manifestations. Intentional tremor, dysarthric speech, hypomimia. Changes in surface sensitivity of the limbs by type "gloves and socks" are characteristic. Possible development of encephalopathy and polyneuropathy. There have been cases of hypertonic reactions, sometimes with myocardial disorders and coronary insufficiency symptoms. Various functional abnormalities of the liver have been reported.

Ethylene oxide has a highly irritating and sensitizing effect on the skin. Severe dermatitis and skin burns are possible. Ethylene oxide damaged male and female reproduction. There is an increased incidence of spontaneous abortions with nurses using it for sterilization. Ethylene oxide is carcinogenic for humans - IARC group I. Higher rates of stomach cancer, peritoneal mesothelioma, brain tumors, leukemia in subjects exposed to ethylene oxide have been found.

Clinical-laboratory tests. Hematological parameters - reduced hemoglobin and lymphocytosis are observed. Cases of chronic lympholeukosis and chronic myeloleukosis are described.

Differential diagnosis. Poisoning with acids, chloromethyl ethers, formaldehyde.

Treatment. There is no specific treatment. Nephroprotectors, cerebro- and pulpoprotectors, hepatoprotectors and other symptomatic therapy are used. Eye and skin wash with physiological serum, ointment application.

Prevention. Sanitary-technical prophylaxis. Adherence to regulations for safe operation, control of ethylene oxide concentrations in the working environment.

Medical prophylaxis. Do not work in contact with ethylene oxide, people with diseases of the nervous and cardiovascular systems, diseases of the liver and skin. Periodic medical examinations - 12 months, with the participation of dermatologist and at indications - ophthalmologist.

Protective professional nutrition - diet "D" is recommended.

3.6.7. TETRAETHYL-LEAD - J. HADJIEVA

Organic compound of lead. It is an oily liquid with a specific odor and high volatility. Practically, tetraethyl lead is insoluble in water, dissolves well in fats and organic solvents.

Usage. *It is mainly used as an anti-detonator for gasoline and a lubricant for internal combustion engines. It is a constituent (up to 75%) of the ethyl liquid that is added to the gasoline.*

Exposed to ethylated gasoline are persons employed in the production, transport, storage and use of the gasoline (air mechanics, airplane engineers, motor vehicle drivers, etc.).

Biotransformation. *The main way of getting into the body is breathing. It is relatively easy to penetrate through the skin and the digestive tract. Tetraethyl-lead circulates in the blood unchanged from 8 hours to 2-3 days, and in individual cases up to 3 months. After its degradation, part of the lead is deposited in the CNS and the parenchymal organs.*

Pathogenesis. *Tetraethyl-lead is a neurotropic poison with a pronounced cumulative effect. It changes the functional state of the hypothalamus by the reflector pathway by irritating the vasoreceptors. Tetraethyl-lead is an enzyme effector. It increases the content of pyruvic acid and acetylcholine in tissues and impacts*

oxidative phosphorylation. Deviations in other organs and systems are predominantly secondary and are related to disorders of their CNS-regulated functions.

Clinical picture.

Acute intoxications are most commonly caused by the inhalation of large amounts of vapor, by pouring of the skin or by sucking and swallowing high doses of ethylated petrol. After a latency period of 10-12 hours to several days, the clinical picture of poisoning unfolds, forming three stages of leakage:

- Mild. It is predominantly characterized by vegeo-asthenic events - headache, fatigue, restlessness, chest tightness, sometimes euphoria. The triad is established - bradycardia, hypotension and hypothermia. Parallel with this, other vegetative manifestations are observed - hyperhidrosis, increased salivation and red dermograms.

- Medium (pre-cumulative). Characterized by hallucinations (hearing, olfactory, tactile), depersonalisation, tonic and clonic seizures, dysarthria. Paralysis of the facial nerve, the ptosis of the eyelids, diplopia and the appearance of pathological reflexes are observed.

- Severe grade (cumulative). Rich psychotic finding - complete disorientation, psychomotor agitation, hyperkinetic disorders, epileptiform seizures, etc. Hypothermia and hypotension change with hyperthermia (up to 40 ° C) and hypertension. Spontaneous bleeding and loss of consciousness. The exit is often lethal.

Chronic poisoning. Characterized with a slow and unclear beginning. Major clinical manifestations are vegeto-asthenic syndrome (early sign of intoxication) and toxic encephalopathy. The triad (hypothermia, hypotension and bradycardia) is optional. In some cases there is a tendency for hypertension combined with bradycardia. Increased tendon and superficial reflexes, sometimes asymmetric, pronounced small tremor of the eyelids, tongue and hands, diffuse sensory disorders, asymmetry in facial inertia, hypothermia, etc. There have been cases, though very rarely, of pronounced parkinsonic syndrome.

Clinical-laboratory tests. There are no characteristic deviations in the haematological parameters. Increased numbers of basophilically stippled erythrocytes and reticulocytes is a rare finding. The enzyme and protein function of the liver has been compromised. Increased urinary excretion of 5-ALA and coproporphyrin. Urine content of lead is used as an exposure test.

Treatment. Antidote therapy with $CaNa_2EDTA$, hepatoprotectors, neuroprotectors, vitaminotherapy (vitamin C and vitamin B group), etc. Physiotherapeutic procedures have a good effect. The use of morphine and bromine is contraindicated.

Labour expertise. In acute poisoning (mild degree), loss of working capacity is temporary. In the case of an medium degree of poisoning, labour is required without contact with toxic substances, and at severe grade defined a group of disability. Chronic poisoning requires temporary or permanent rehabilitation.

Prevention. Sanitary-technical prophylaxis. Respect for personal hygiene and use of personal protective equipment.

Medical prophylaxis. Do not work in contact with tetraethyl-lead individuals with CNS, liver, kidney, cardiovascular, digestive, endocrine, skin and visual analyzers diseases. Periodic medical examinations - once every 6 months in the production of tetraethyl-lead and ethyl liquid and once every 12 months with the use of ethylated benzine, with the participation of therapist, neurologist and at indications - dermatologist and psychiatrist.

Protective professional nutrition - diet "B" is recommended.

3.6.8. POLYCYCLIC AROMATIC HYDROCARBONS - D. APOSTOLOVA

Polycyclic aromatic hydrocarbons (PAHs) - with significant toxicity and carcinogenicity, are: naphthalene, antracene, benz (e) pyrene, benz (a) anthracene, dibenzanthracene, benz (a) pyrene, chrysene, dibenzcarbazole and the like.

Usage. Occupational risk. *They are obtained by the high-temperature processing of organic raw materials (petroleum, resins, oils, bitumen, coal, etc.) by dry distillation, cracking, coking, incomplete combustion. Contained in coal tar, asphalt, coke, car gases, smoke, soot, petroleum products, asbestos ore and treated asbestos. Naphthalene is used as an insecticide, in the production of chemicals, plastics, paints; anthracene - in the manufacture of anthraquinone and dyes. Workers from the mining, chemical, transport and electrical industries are at risk.*

Biotransformation. Pathogenesis. *The primary route of penetration in the body is respiratory, with slow absorption. It is also possible in the case of skin contact, and ingestion (food, water). They are deposited in fat-rich tissues. In non-cancerous effects, the target organ is the respiratory system. Liver disorders, myelosuppressive effects, and the pathogenesis of atherosclerosis are also observed.*

The carcinogenic effect of PAHs was demonstrated indirectly after a study of occupational tumors in workers (where there was a PAHs group effect) and experimentally on animals. PAHs with proven experimental carcinogenic activity include over 480 substances. In

animal experiments, their carcinogenic activity has been shown to depend on the 1,2-benzapyren content, since among the inherent PAHs 1,2-benzapyrene has the highest carcinogenic activity, IARC group 2a. Cancer cases in humans as a result of direct contact with pure 1,2-benzapyrene have not been reported, but a marked correlation has been observed between the incidence of occupational tumors in contact with thermal fuel treatment products, the temperature regimen of their preparation and the content of 1,2-benzpyrene in them.

It is assumed that the toxic effect and the carcinogenic effect of benz/a/pyrene are associated with its metabolism. Its metabolic products (reactive epoxides) are genotoxic agents that covalently bind to DNA and lead to mutations and initiation of tumors.

Carcinogenic PAHs cause skin cancer at the site of the contact, but tumors are not excluded in individual organs. It is believed that carcinogenic PAHs affect the incidence of lung, stomach, liver cancer.

Increased risk for smokers, excessive exposure to UV radiation, malnutrition (vitamin C, iron, riboflavin loss), concomitant liver and skin diseases, women of childbearing age. Passing through the placenta benz (a) pyrene increases embryo - fetolethality and leads to premature birth and malformations.

Clinical picture.

Acute poisoning. Inhalation irritative lesions of the respiratory tract lining develop with sneezing, harassing of throat and behind the sternum, cough, also headaches, nausea and vomiting. Naphthalene can cause methaemoglobinaemia and haemolysis. In massive skin contact, erythema with itch or moderate chemical burning is observed.

Chronic effects. Benign skin changes - folliculitis, comedones, arteficial dermatitis and hyperpigmentation, leukoplakia, precancerous degeneration (keratoses, papillomas, melanoses, oil acne, torpid ulcerations) more on the head, neck, limbs and genitals. After prolonged skin contact with benz/a/pyrene (10-30 years), spinocellular carcinoma with slow evolution developed. It is possible to develop lung cancer and urinary bladder carcinoma. Liver lesions with hepatomegaly and elevations of γGTP, stimulation of hepatic regeneration (proliferative effect), rarely hepatic carcinoma are also observed. Myelosuppressive activity is manifested by leucopenia, rarely thrombocytopenia and aplastic anemia.

Clinical-laboratory tests. Increased γGTP (over 30 U/l), leucopenia (less than 3 G/l), thrombocytopenia (below 100 G/l), aplastic anemia (Hb below 100 g/l and Er less 2.10^{12}/l), decrease to lack of reticulocytes in peripheral blood.

Specific biomarkers for exposure are not available (noxe and its metabolites in urine - e-hydroxypyrene can be investigated). Biomarkers for the toxic effect are suitable in vitro tests for effects on DNA with manifestations of induced chromosomal aberrations and daughters chromatin exchanges.

Differential diagnosis. In acute poisoning - with irritating gases, amino- and nitro derivatives of benzene and toluene. In chronic - with impairment of other carcinogens in the working environment.

Treatment. In acute poisoning, cerebral and pulmoprotective agents are administered; treatment of available methaemoglobinaemia and haemolysis in the usual ways; washing the skin with water and soap and ointment. Photodermates are treated with cortisone-containing preparations, protective or exposure-reducing creams. The manifestations of the carcinomas are surgically treated with chemotherapy radiotherapy.

Prognosis and expertise of working capacity. In acute poisoning, the prognosis is good. When developing a professional carcinoma, the prognosis is pessimistic. Patients are permanently removed from the work environment and directed to WEMC. In case of skin precanceros, they are permanently removed from the work environment and have temporary limited working capacity.

Prevention. Sanitary-technical prophylaxis and personal protective equipment. Medical prophylaxis. Do not work people with skin diseases with increased photosensitivity and skin precancerous. Workers in contact with surfactants are subject to a 6-month prophylactic examination with the participation of therapist and dermatologist with Hb, leukocytes; on indications BSR, DBC, γGTP, cystoscopy. It is recommended diet B of protective professional nutrition.

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3.7 MASS ACUTE POISONING - TOXOCHEMICAL TRAUMATISM. MASS POISONING FROM INDUSTRIAL POISONS

Mass acute poisonings, also called toxo-chemical traumatism, are disastrous conditions in a particular area due to industrial, household, natural accidents, where a large amount of poisonous substances are released or by the inclusion of toxic compounds in special conditions. Many people are affected by the action of mono- or poly toxical agents. The conditions of accidental incidents and the mass nature of aggression determine availability of certain features in the clinical characteristic and treatment, which distinguish them from single toxic injuries of the same type of noxae.

Mass acute poisoning is, by its nature, one of the most aggressive pathologies in modern conditions. This is due to the fact that:

- At the same time, a large number of people are struck in a very short time (minutes and hours), in which way another damaging cause (such as earthquake, flood) can rarely occur;
- The striking agent causes such injuries in the body of the injured that, in inadequate combat, the same causes great mortality and disability.

Group and mass acute poisonings can be classified in the following classification (Al. Monov) according to the type and conditions of occurrence **(Figure 1).**

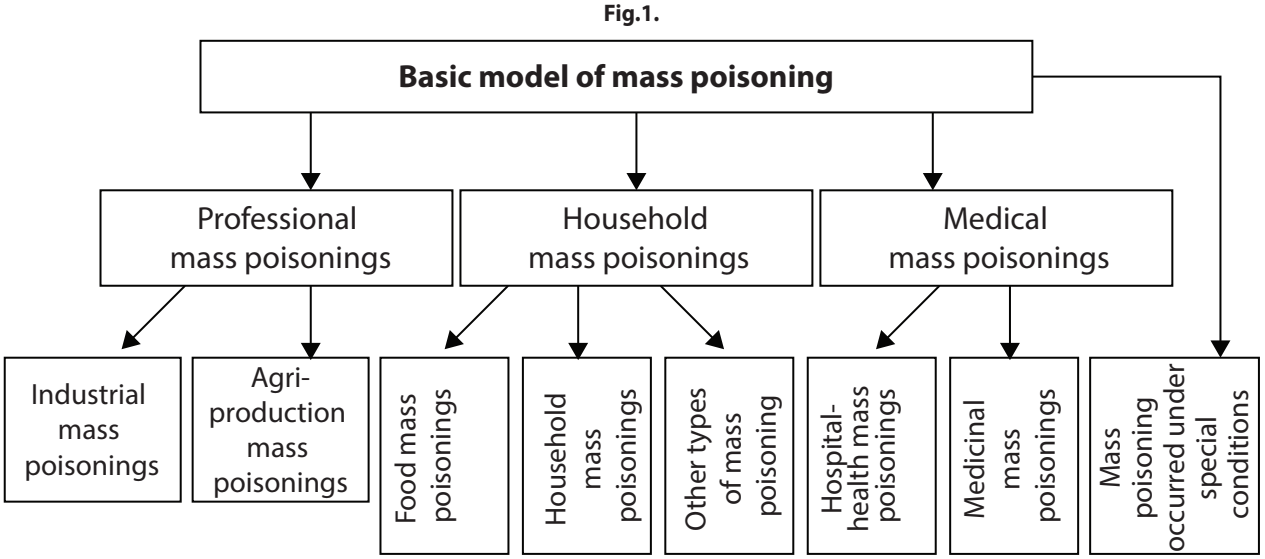
The type of damage from acute intoxications in these groups is determined by the toxic noxa that has different physicochemical properties. Due to the massive nature of the injuries of the victims of massive poisoning and their simultaneous occurrence, they are also defined as toxochemical traumatism - the most aggressive form of mass traumatism. It grows annually and is presented in the following forms (Al. Monov):

1. Monotoxic forms.
(a) gas-inhalation;
(b) nutritional;
2. Polytoxical forms;
3. Coupled forms: toxic-mechanical-baro-thermal species.

Etiology. Mass poisoning is most commonly caused by two types of agents: poisonous gases penetrating the body mainly through the respiratory system and less frequently through the skin and mucous membranes and other types of poisons penetrating the digestive tract predominantly with different types of food.

In some cases, the damaging agent is monotoxic - only one toxic substance, in other cases it is mono- or poly toxic, combined with other striking agents: dusts, glowing of the gas dust mixture, exposure to the latter under pressure on the human body. These peculiarities of the etiological nature of mass poisoning determine the much heavier and wider possibilities of their aggression. The conditions in which

Fig.1.



poisonous gases are formed are mostly technical accidents, fires, explosions. Hence, smoke or explosive gases, silage gases, etc. are formed as agents of mass poisoning. In terms of their chemical nature, they are most often carbon monoxide, chlorine, fluorine, nitrobenzene and phenol vapors, hydrogen cyanide and others. The complex composition of toxic gas agents contributes to the potentiation of their damaging effects on injured persons. Also:

- In the gas mixtures mentioned above, the formation of new toxic substances (eg carbon monoxide and chlorine - phosgene is formed at this temperature).
- Simultaneous combination of single and combined poisonous gases with the above-mentioned non-toxic damaging agents (thermal, dust, etc.).

Chemical food poisoning is caused by poisons that do not at most exhibit their specific odor qualities or are poisoned by food stuffs and food technology. Mass poisoning caused by toxic polluted sources of drinking water is also the cause of these lesions.

Pathogenetic mechanisms of gas-induced acute poisoning.

In the case of mass poisoning by mono- or poly toxic gas combined and uncombined poisons, local and general mechanisms of damage are developed. Local damages mainly affect the respiratory system and are manifested by:

- irritative or necrotic lesions of the lining and the walls of the airways with edema and increased secretion;
- blockage of different parts of the bronchial tree - as from the lining edema and the lining secretions, and under corresponding conditions of mechanical particles;
- multiple atelectatic areas - in connection with obstruction and the occurrence of bronchospasm;
- inflammatory processes in the changed lung areas;
- pulmonary edema and bleeding in pulmonary parenchyma.

When a baro- factor is present, microrupturesare formed in the wall of the bronchi and bronchioli and, in the presence of a thermofactor, burns occur on the surfaces of the respiratory tract. To a lesser extent, there are various types of toxic damage on contact areas of the skin and ocular mucous membranes.

These damage to the respiratory system results in the formation of a broncho-alveolar capillary block - disturbing the absorption of oxygen, which results in severe general damages.

The general lesions are varied depending on the chemical characteristics of the poison and the conditions of the intoxication. The main mechanisms here refer to:

- ***Hypoxia*** - affecting particularly the brain and myocardium cells;
- ***A shock condition*** - due to blood circulation disorders;
- ***Direct damage to enzyme systems.***

In this way, severe damage to the parenchymal organs, blood, disorders of the main balances in the body, such as acid-alkali with severe acidosis, hydroelectrolyte - with severe dehydration and hemoconcentration; protein, carbohydrate, lipid balance, and so on.

Clinical characteristics of gas-inhalation mass acute poisoning. The following major syndromes are formed:

- *general toxic;*
- *pulmonotoxic;*
- *ophthalmotoxic and rhinotoxic;*
- *chemotoxic;*
- *hepato- nephrotoxic.*

The prognosis of these mass poisonings is too often unfavorable due to complications, irreversibility of severe forms, rapid lethal outcome in improper treatment.

Mechanisms of impairment and clinical features of mass food poisoning. They also have local and general disabilities. Local include varying degrees of damage to the lining of the digestive tract (most commonly hyperemia and increased secretion) and rapid disruption of bacterial equilibrium in the intestinal tract with a strong increase in pathogenic forms.

General disorders are manifested by a variety of pathogenetic mechanisms and include: severe dehydration; impaired acid-base equilibrium; acute circulatory disorders; syndromes by CNS, liver, kidney, blood, and oth. (depending on the chemical characteristics of the poison).

Clinical picture. In the case of mass poisoning, it is manifested by:

General gastrointestinal syndrome - vomiting, diarrhea, abdominal pain.

Other syndromes from different organs and systems depending on the type of intoxication. Forms of this type of mass poisoning are aggravated by shock conditions, respiratory disturbances, severe metabolic acidosis, etc., which determines severe prognosis and inappropriate treatment - lethal outcome.

General healing tactics for massive acute poisoning

Preparatory section. The uniform preparatory program includes the following groups of events:

- Preliminary information from the various re-

gions of possible toxic noxae in industrial enterprises, farms, households, etc., which may cause mass poisoning, under favorable conditions.

- Determination and estimation of medical and other means of transport.
- Determination of the bed fund in the hospitals.
- Identification and preparation of the medical staff who will serve the incoming contingent.
- Designation of reserve buildings and spaces for further set up of hospitals at an extremely massive nature of poisoning.

Therapeutic section. It identifies two types of data: criteria for assessing the type of intoxication and the extent of its damage to poisoning; the amount of medical assistance in the three stages of service to the victims - *at the place of the accident, in the transport vehicle and in the hospital*.

Basic actions in the treatment of mass poisoning of the gas-inhalation type.

1. Removing the victims from the gassed environment.

2. Reanimation methods - apply to the severe and extreme severe stadium of injuries to the victims in the three stages of their care. These are: respiratory and cardiovascular resuscitation and correctional-replacement therapy.

Breathing resuscitation. It is performed with portable or stationary respiratory equipment (if necessary with portable and stationary barocameras);

- cardiovascular resuscitation and correctional-replacement therapy. It is performed with drip infusion therapy intravenously - with hydroelectrolyte, monosaccharide and plasma-substitution solutions combined at shock with glucocorticoids and vasopressor agents; cardiotonic drugs (such as strophantine, cardiac glycosides, etc.) are added.

3. Pulmoprotective agents - include aerosol and parenteral forms of various drugs (glucocorticoids, beta-stimulants, novphilin, acetylcysteine, etc.) to overcome the broncho-alveolar capillary block.

4. Antidotal agents.

5. Anti-hypoxants (Pyramem, etc.).

6. Protective drugs and combinations to the affected organs and systems.

7. Detoxic cleansing methods of blood - exchange Transfusion and Dialysis.

8. Antibiotics and immunoprotective agents.

9. Symptomatic remedies.

Basic Methods of treatment of mass food poisoning.

1. Detoxic cleansing of the gastrointestinal tract - applied at the onset of intoxication in the absence of contraindications.

2. Drip infusion of hydroelectrolyte, monosaccharide, amino acid solutions and protein preparations, plasma substitutes with corrective and replacement target of the three service stages.

3. Resuscitation - in extremely severe and severe forms of blood circulation and breathing damages in the three stages of service.

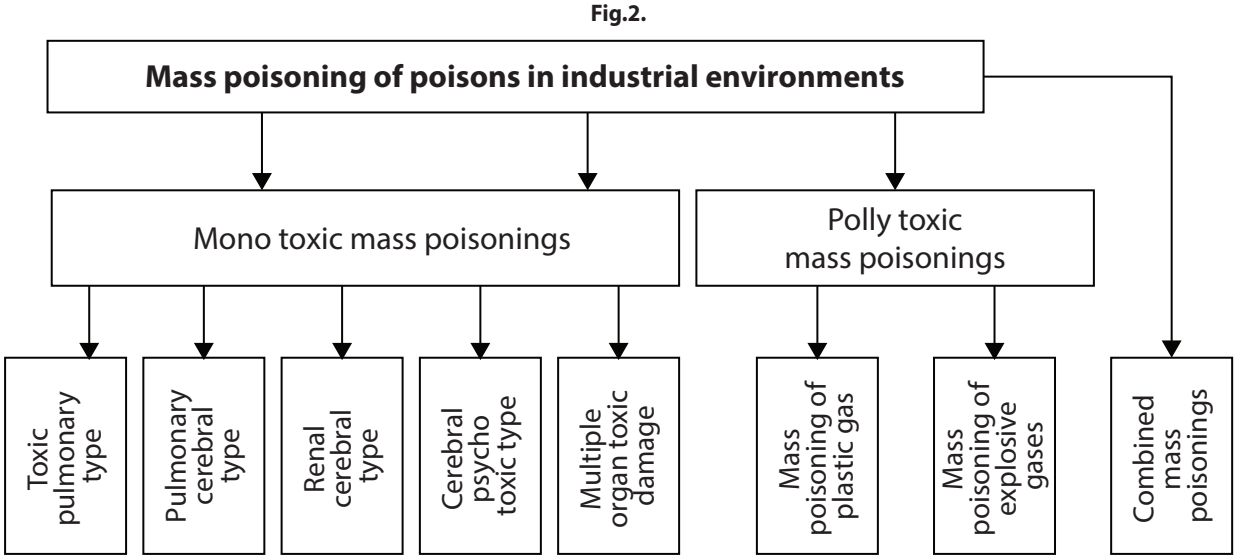
4. Antidotal agents.

5. Organoprotective remedies at the hospitals.

6. Antibiotics and symptomatic agents.

An important meaning for the damages of mass poisoning is the fact that a number of non-toxic substances, under certain conditions become a source of poisons causing severe poly-toxic mass poisoning. Such substances are plastics - at high temperatures (fires) the different synthetic materials form highly toxic gases and vapors: carbon monoxide, nitrogen oxides, sulfur oxides, phenol, nitrobenzene, aminobenzene, phosgene, formaldehyde, toluene and others.

Fig.2.



Too often toxic factors in mass poisoning are combined with other factors: thermal agent - high temperature in fires; baro- factor - high pressure of the toxic mass, most often in an explosion. This combined etiological factor in mass intoxications causes much more severe and varied lesions on the affected contingent, and it is most common in industrial mass intoxication damage.

Mass poisoning of industrial poisons

They occur in emergency situations in industrial plants and large laboratories with the most frequent reasons:

1. Incident in technical equipment: gas pipe cracking, tank breakage or explosion.

2. Incident in the technology of the production process: in case of deviations in the temperature regime and the pressure of the gas components.

3. Fires in production and storage premises.

4. Explosion of waterhousing pressurized poisonous substances.

5. Special conditions: terrorist actions, military conflicts, riot stuggle, etc.

In the case of mass poisoning by industrial poisons the most common causes are gas poisons. The following types of industrial mass poisoning occur **(Figure 2)**:

1. Monotoxic mass poisoning - caused by only one type of toxic agent.

2. Polytoxic mass poisoning - caused by a mixture of more than one toxic agent.

3. Combined mass poisoning - caused by the combination of a mono- polytoxic agent combined with other factors (thermo- baro factors, etc.).

The most common mass poisoning is with leading *pulmo-toxic* lesions, mass intoxications with leading *pulmocerebral* lesions, leading *renal-cerebral* and *cerebro-psychical injuries* and mass poisoning with *polyorganic damages* to the injured.

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4 OCCUPATIONAL DISEASES BY PHYSICAL FACTORS

Zl. Stoineva

4.1 PROFESSIONAL CRYOPATHOLOGY

Subject of cryopathology is the person in conditions of cold impact with his changed life activity, altered reactions of the organism as a whole and of the individual functional systems, changes in the morphology of cells, tissues and organs.

Risky professions *are workers in refrigeration chambers and repositories for food and other products; meat and fish processing industry; fishermen and sailors; storage and processing of dairy products; artificial ice production; fermentation rooms for sugar and beer production; chemical and biochemical laboratories operating at low temperatures; sellers of frozen fish and meat; launders with non-mechanized work; retouchers of porcelain; workers in winter outdoors in construction, logging, agriculture and others. Artificial cold applied to more than 250 branches of industry, science, technology, food industry.*

All living tissues react to the effects of cold, but the most sensitive and injured are the nervous system and the vascular system. Diseases of the peripheral nervous system - neuralgia, neuritis, radiculitis occur 1.5 to 2 times more often among workers in general or local cold conditions and occupy second place after cold airway diseases.

Under the influence of low temperatures, the peripheral blood vessels of the skin and the subcutaneous tissues shrink, which improves the thermal insulation of the tissues and reduces the heat transfer. Peripheral vascular spasm is due to the direct action of cold on peripheral neurovegetative structures and blood vessels, to the excitatory catecholamine mechanism of stress response with stimulation of the sympatho-adrenal system. Centrally, hypothalamic efferency is caused by hypothermia. Compensator stimulates metabolic heat production through arbitrary muscle contractions and "cold tremor". The cold conditioned vasodilator response is more pronounced in acclimated patients. Tissue metabolism is disrupted, vascular permeability is disturbed, blood viscosity is increased, stasis and microtrombosis are detected by shutting off various parts of the vascular line while preserving blood circulation in arteriovenous anastomoses, and in capillaries, venules and other vessels gradually ceases. Pathogenesis is associated with tissue frost, hypoxia and the release of inflammatory mediators with excessive production of thromboxane A2, which disrupts the normal balance between prostacyclin (prostaglandin I2) and thromboxane A2, followed by progressive necrosis. Systemic hypothermia disturbs physiological vital functions - blood circulation, tissue metabolism, respiration. Oxygen consumption is reduced by about 7% at 1 ° C. Myocardial repolarisation is delayed, heart defibrillation occurs.

External factors that increase the risk of cryopathology include: increased humidity and speed of air movement, vibrations, sharp temperature drops, narrow, moisture-tight and wet clothing and shoes, altitude, geographical specificity. Endogenous factors contributing to cold pathology include: exhaustion, overwork, avataminosis, hunger, smoking, alcoholism, reconvalescence conditions after severe illness, physical disabilities, mental disorders, reduced motor activity, impaired adaptation and acclimatization, pre-existing cold traumas.

Crush damage can be generalized or local; temporary (transient) or permanent. Depending on the cooling rate and the duration of the cold effect, acute, subacute or progressive and chronic cold trauma are distinguished. Progressive cold-induced effects are characterized by a gradual decrease in temperature in peripheral and central body tissues, expressed in pain, neuro-muscular insufficiency, loss of sensation and resp. cold-induced damage. Chronic effects affect peripheral nerves and vessels as well as cardio-pulmonary functions.

Generalized hypothermia, as a concept, involves the influence of cold on the whole organism and is the result of ineffective thermogenesis. It occurs not only at minus temperature but is also possible at + 10 ° C to -12 ° C at exposure over 12 hours and additional endogenous predisposing factors.

The initial phase of total cooling is a common and easily reversible when warming condition. Body temperature remains above 35 ° C, but frostbites of varying degrees may be present on the acral parts of the body. Initial symptoms are often unobservable without specific clinical features. There is fatigue, adynamia, apathy, drowsiness, dizziness, memory disturbance. The thermoregulatory "cold tremor" disappears, the speech becomes blurry, obscure, the coordination of movements is disturbed. Increased irritability, hallucinations. Disagreeable pupil reactions, hypo- to anesthesia for pain, touch and temperature, hyporeflexia, attenuated or absent pulse, reduced respiratory rate, arrhythmia, arterial hypotension, increased blood viscosity have been reported. The skin is pale, cold, the fingers pale or cyanotic.

In a *mild degree* of generalized hypothermia, a rectal temperature of 25°C-33°C, adynamia, bradypsychia, bradylalia, apathy - movements are sluggish, slow, speech is descant, slow. There is indifference. The pulse is slightly slow. Local frosts of I-II degree are possible.

At *moderate degree* of generalized hypothermia, body temperature falls to 32°C-29°C. Stupidity, somnolence, pale cold skin, bradycardia, arterial hypotension, reduced to 7-12 per minute respiratory rate, breathing is superficial. Nerve conduction is delayed. Possible complications with local frosts from I to IV degree.

The severe degree of generalized hypothermia is characterized by loss of consciousness, constant, usually flexionary muscle contracture. The body temperature is below 28°C. The pulse is slower than 32 beats per minute, poorly filled, does not palpated into the periphery. Arterial pressure is low to absent. The respiratory rate is reduced to 3-4 per minute with a faulty rhythm - the Cheyne-Stokes. Urinary incontinence is observed. Edematous brain, lungs, severe frostbite of the extremities and face is observed. This condition can go into a severe coma with cardiac fibrillation and cardiac arrest.

Necessary studies: complete blood count, blood sugar, urea, electrolytes, amylase, alcohol and drugs levels of blood, bleeding and clotting time, prothrombin index, ABB, diuresis, urine analysis of albumin, sediment. Functional study of cardiac activity, thyroid gland, radiography of the lungs and heart is needed. The ECG describes a pathological J-wave, between QRS and ST.

Differential diagnosis refers to other diseases, passed by disorders of consciousness, such as endogenous and exogenous intoxications, cerebrovascular disease, traumatic brain injuries, brain tumors, hyper- or hypoglycemic, uremic, hepatic coma and others.

Treatment includes heating the body with electric pillows or blanket, warm bed, bath with a water temperature of 37 ° C and according to a many of authors and up to 40 ° C, with constant control of the victim's consciousness. In moderate and severe hypothermia and body temperature below 32 ° C, constant monitoring of heart rate and rhythm is required to increase body temperature to 36 ° C (risk of cardiac fibrillation). Due to the spontaneous recovery of heart rhythm with warming, antiarrhythmic agents are usually not used. Infusions of gradual warmed to 40 ° C dextrose-salt solutions, low molecular dextran, humanalbumin are required. Avoid potassium-containing solutions (until stabilization of serum potassium level). It is necessary to manually and lung motor inhale with oxygen, prior to the warming up. Follow the diuresis every hour and constantly body temperature. Acidosis, hypoglycaemia, hyperkalemia are corrected. Antibiotics are used for infections. Data on brain and/or pulmonary edema uses dehydration therapy. Pretreatment and healing local frosts.

Generalized hypothermia is a severe condition requiring urgent treatment in an intensive and resuscitative ward.

The prognosis is good for patients with precede good health, without predisposing external and internal risk factors and timely medical attention.

There are a number of clinical forms of **local cold pathology**, which are generally divided into two main groups: externally visible cold injuries and cold lesions without externally visible disturbances (with the predominant involvement of deeper tissues).

The **first group** refers to **frosts (congelatio) and perniones**. The tissue damage begins with the formation of ice crystals in the extracellular fluid, the blood with platelet aggregation, and stopping capillary blood flow. The damage is most pronounced after warming.

Congelatio is a local cold defect due to freezing of tissues from superficial to deep structures. There are three, four, five-degree congelatio classifications depending on the depth of tissue disturbance. Wide application and more convenient to choose an adequate therapeutic approach is the four-stage classification:

I grade - congelatio erythematosus. Affects only the surface epithelium. It is common for short-term effects. It is characterized by mild or stronger redness with livid spots, slight swelling of the skin and pressure sensitivity. Observed 12 to 24 hours after warming. As a result of capillary dilation and aseptic inflammation, the previous cold, pale and dry skin becomes hot and red, superficial painful and stretched. For a short time, hyperaesthesias, pares-

thesias and lesser anesthesia persist. First degree frost zones recover without scarring (even without treatment), but often remain hypersensitive to cold and hypothermic. Sometimes they are blue-red, pale or abnormally pigmented.

Grade II - congelatio bullosa. Affects skin to stratum basale s. germinativum. After 24 to 48 hours of warming, local swelling (due to disturbed vascular permeability) and blisters occurred. Bubbles between corium and epidermis are initially filled with serous fluid, and later with opalescence-purulent of leukocytes and bloody, red-black of the erythrocytes. Bullets are formed only on the dorsal side of the limbs, face, body. After second-degree frost, a sudomotoric disorder with hyper- or hypo- to anhydrous zones is detected due to disordered innervation and circulation. Direct damage to the thermoreceptors leads to hypothermesthesia. Prophylactic measures against bulets infection are necessary. Anti-tetanus serum is required. Cytaraxes remain only after a secondary infection. Fingers usually fall off. Atrophic skin processes with increased vulnerability are observed. Frozen areas can remain cyanic or hyperpigmented. Paresthesias and spontaneous, local pains, as well as hypersensitivity to repeated cold effects, often remain.

Grade III - congelatio necroticans, gangrenosa seu excoreatica. All layers of epidermis and dermis are affected by passing through the previous two stages of local frostbite. An intensive edema with aseptic inflation of the entire area is observed. Necrotic processes can be restricted or cover deeper lying tissues. On the second and third day after warming, a fine, false line of demarcation is restricted, which could fall distantly during the warming process. Subsequently, the definitive deep demarcation groove is formed with protruding edges covered with granulations. Necrotic areas become dark red, brown or gray to blue-black (if no wet necrosis occurs). Mummification of dead tissues develops. There are black crusts that fall on their own, while tendons, ligaments, capsules and bones are not released operationally. The warming of frozen tissues is associated with blood reperfusion and simultaneous generation of free oxygen radicals. Healing is a long-lasting process: the skin is hypersensitive, dystrophically thin, often with slowly healing ulcers. The cicatrixes are crude and painful. The wounds are dressed with ointments enriched with vitamins and epithelizing substances and with cortisone to suppress granulation. Preventive measures against secondary infections and wet necrosis. Fascio-skin graft on wound areas after frost has a good effect.

IV grade - congelatio necroticans. It is characterized by necrosis of all tissues due to mechanical destruction of cellular and tissue structures. Warming does not lead to recirculation and recovery. A demarcation groove and a necrosis emerging, which requires urgent amputation and symptomatic and preventive antibacterial treatment. A very good prognostic value for determining the weight of frost and the demarcation line, as well as the treatment effect, is the biphasic scan of the bones of the limbs with technetium 99 m.

Often after frosting, there is a hypersensitivity to cold, tingling of the fingers, reduced sensation to touch, increased vasospastic reactivity.

Perniones (chilblains) are blue-red itchy spots with edema of the dermis and hypoderm located on the exposed parts of the body (face, ears, wrists, toes, legs) and accompanied by itching, burning, pain. It is possible to form sub-epidermal bulls, ulcerative hemorrhagic lesions, which are scarring with fibrotic or atrophic changes.

Cutis marmorate (marrow, skin networking - in women with hormonal disturbances and vegetative dystonia), ***livedo reticularis a frigore*** (spoted skin with a livid or red color on the extensor parts of the extremities and the side part of the body), ***erythromelalgia*** (redness and pain usually on exposed parts of the body), ***acrocyanosis*** (livid staining of distal parts limbs sometimes with hypersesthesia and edema) or ***erythrocyanosis (the same, but on the open parts of body)*** are cases, that are manifested or enhanced by cold effects but are not pro fessionaly determined. Differential diagnosis should also be considered, as well as contraindications to work in a cooler microclimate.

The second group of local cold pathology (without externally visible lesions) refers to **an immersion leg and hand, and cold-induced vegetative polyneuropathy**.

Immersed foot, submerged hand (immersion foot, trenchfoot, shelter leg, Flander's foot, immersion hand, main detranche, pied de tranche) after two or three hours at + 8°C to -15°C with wet clothes, and shoes, there is a cold defect that occurs in three stages: pre-hyperemic (lasting for several days) - slight edema, numbness, burning and local hypothermia are observed. The pulse is not felt; hyperemic stage (lasting from six to ten weeks) - redness, rising cold edema, hypothermia, paraesthesia, severe palliative pain is observed; post-hyperemic stage (lasting a few weeks to several months) - characterized by stiffness, paraesthesia, dysaesthesia, pain, muscular rigidity, pronounced cold edema, a net pattern on a pale or sloppy skin background.

The most common in occupational pathology is the neurovegetative and vascular microcirculative pathology of the extremities due to chronic cold

effects (with terminological diversity: cold angiotrophneurosis, cold angioneurosis, cold endarteritis, cold vegetative polyneuritis, cold neurovasculitis, angioneurodystonia et angioneurodystrophia a frigore). We have the term **vegetative polyneuropathy in cold weather**. Clinical events unfold gradually. In the case of a slight degree (upper limbs most commonly affected) subjective evidence of transient numbness, palpitations of the fingers and palms, paroxysmal change in skin coloration (reddening or pale) usually occurs in cold weather. From the objective clinical finding, a partial dissociation of sensation is established, with the influence of the extraceptive sensation for warm and cold and preserved sensation for touch, pain, deep and complex sensation with distantly increasing hypothermesthesia. Capillary polymorphism, at capillaryscopy, on a pale pink background. In cold provocative and mediatory tests and in the study of neurovascular reactivity, dystonic vegetative manifestations predominate with sympathicotony. Mean degree of cold-induced vegetative polyneuropathy is characterized by paresthesia-algic manifestations of tingling, burning, sprained pain in the depth of tissues, mostly at night and morning, ischemic or erythema-cyanothic skin, swollen fingers and initial dystrophic disorders of the skin and nails, rising hypoesthesia for temperature, touch and pain, distal rising hypothermia (to thermoamputation in the fingers), spastic capillaries with pronounced permeability disorders, pronounced hyperhidrosis. In severe grade disease, the tendency towards irreversibility of clinical symptoms with pronounced vagotonic vascular manifestations and pronounced dystrophic skin disorders and skin appendages is highlighted - stiffened, broken fingernails, smoothed dermatoglyphic outlines of the fingers (apex phalanx weakening), deformation of the interphalangeal joints, tendency to flexicone contractures. Changes in sensitivity indicate impairment of small and large diameter myelinized and non-myelinated nerves. Histomorphological changes are expressed in severe proliferative-destructive changes in vascular walls, arterio-venous anastomoses, glomic bodies, sweat glands, fibrotic cellular tissue elements.

In the differential diagnosis, vegetative polyneuropathy with other genes (over working, microtraumatism, vibration), cervical and lumbosacral radiculitis, plexitis, mono- and polyneuritis and neuropathies, endarterites, vasculitis, neurovascular compression syndromes, reflex sympathetic dystrophy, syringomyelia and others.

Treatment is preceded by taking the patient out of the cold environment.

The pathogenetic approach is determined by the prevailing clinical manifestations: nestheroidal anti-inflammatory agents such as Tenoxicam (Tilcotil), Ketoprofen (Profenid), Lornoxicam (Xefo, Safem), Meloxican (Movalis), Bumadizonum Calcium (Eumotol), Diclofenacum Natrium (Feloran, Voltaren) , Piroxicamum (Piroxicam), and others. They can be combined with Analgin, Alcozin, Tempalgin, Neuralgin and other combined analgesics. It is appropriate to include trophic improving vasoactive and vasodilating drugs - Naftidrofuril (Dusodril), Pentoxyphylline (Trental, Aagapurin), Pyridylcarbinolum (Radecol, Ronicol), Triphosadeninum (Atriphos, Phosphobion). Evidence of the role of thromboxanes and prostaglandins would change the therapeutic approach by using antiprostaglandin and other therapy.

In the case of an exaggerated sensory symptomatic and according to the general state of the organism, it is appropriate to include psycholeptics or psychoanaleptics: Opipramolum (Insidon), Diazepamum (Diazepam, Relanium), Chlordiazepoxidum (Elenium), Alprazolamum (Xanax).

Diluted neurological symptomatic include medications that improve nerve conduction such as Galanthaminum hydrobromide (Nivalin) or Bendazolum hydrochloride (Dibazolum). Vitamins belonging to group B, A, C, E are also suitable. It is also appropriate to combine natural balneal procedures with analgesic, improving tissue nutrition and a strenght giving (tonic) effect.

The occupational-medical expertise in cryopathology provides for temporary disability and temporary re-employment without a specific length of time. In heavy grades and advanced stages of illness, a disability group is defined.

Prevention. No work with collagenosis, Raynaud's phenomenon, vegetative dystonia, peripheral, vegetative and vascular insufficiency, vasculitis, pre-existing cold traumas, systemic diseases, cryoglobulinemia should be avoided in cold and microclimate conditions. Half-hour and one-hour breaks are arranged under comfortable microclimate, hot drinks and a balanced diet, suitable workwear and shoes, PPE. Periodic physio- and spa treatments. Periodic medical check-ups once a year for workers in cold-weather conditions involving internist, neurologist and capillaryscopy and cold test complete.

Zl. Stoineva

4.2 PROFESSIONAL THERMOPATHOLOGY

The development of metallurgy, machine building, chemical and light industry has created conditions for work on a microclimate overheating for many workers. The risky professions include those working in metallurgy - in rolling mills, blast furnaces and martin furnaces; machine building - foundries, smiths, metalworking workshops; textile industry - spinning and weaving workshops, dyeing and drying machines; In ceramic production; In chemical plants; thermal power plants; gas and boiler installations; bakers, bakeries, confectioners, chefs, miners, tractors, combine harvesters and others.

External factors that increase the risk of heat damage include increased humidity, low airflow speed, heavy physical work, rapid temperature rise, geographic specificity.

Internal factors that promote thermopathology include: sex, age, exhaustion, overwork, hunger, overeating, obesity, avitaminosis, smoking, alcoholism, systemic disorders, impaired adaptation and acclimatization, skin and endocrine disorders, cardiovascular diseases, use of medications that reduce sweating secretion, skin circulation or lead to dehydration (atropine, phenothiazines, tricyclic antidepressants, diuretics, laxatives, anticholinergics, antihistamines, vasoconstrictive means, MAO inhibitors, β-blockers).

Professional thermal injuries include **heat stroke, heat exhaustion, heat cramps, heat syncope, and local skin injuries**.

Heat stroke occurs rarely, mainly in emergency situations, in workers performing heavy physical work with inadequate work clothes and predisposing internal factors. It is a life-threatening and requiring urgent medical emergency, that occurs as a result of inefficient thermoregulation with hyperthermia exceeding 41.1 ° C. It is characterized by sickly altered consciousness, dizziness, weakness, nausea, vomiting, confusion, delirium, visual disturbances, clonic and tonic convulsions. The skin is hot, initially covered with sweat, and consequently dry. Blood pressure at the start may be slightly increased, but quickly dropped. The pulse is rapid, filiform. Breathing is rapid, superficial. Respiratory alkalosis and compensatory metabolic acidosis are established. Death occurs due to cerebral, cardiovascular, hepatic and renal disorders. Laboratory findings indicate leukocytosis due to dehydration, decreased serum potassium, calcium, phosphorus, hemoconcentration, thrombocytopenia, increased bleeding and clotting time, fibrinolysis and coagulopathy. Urine was concentrated by proteinuria, myoglobinuria.

Differential diagnosis is necessary with all endogenous and exogenous causes of a disease disorder of consciousness.

Treatment is aimed at rapidly reducing body temperature in a cool, airy room. The patient is swollen with cold water (15 ° C) or wrapped in wet sheets that change. Apply ice gastrolavage. Cooling continues until the body temperature gastrolavage to 39 ° C. Because of the risk of hypoxia and aspiration, intubation is recommended. Appropriate intravenous infusion of 25-50 mg Chlorpromazinum to suppress tremor, infusions of water-salt solutions to correct electrolyte imbalance and dehydration. Symptomatic treatment of renal, hepatic or cardiac pathology, the coagulopathy.

Heat exhaustion is observed in workers with severe physical labour during prolonged exposure in conditions of overheating microclimate with insufficient sodium chloride and water intake. It occurs as a result of dehydration and depletion of sodium due to loss of isotonic fluids (sweating). The patients complain of a strong thirst, weakness, nausea, fatigue, confusion, headaches. The body temperature is above 38 ° C, the pulse is increased. The skin is red and damp. Symptoms of heat syncope or heat cramps may occur. In some cases, hyperthermia leads to hyperventilation and secondary respiratory alkalosis. Heat exhaustion could be a heat stroke when increasing hyperpyrexia and reducing sweating secretion and sweating evaporation. The treatment consists in removing the patient from the conditions of overheating microclimate in a cool room and rehydrating and supplying with sodium chloride - orally or parenterally intravenously with physiological saline. The patient is temporarily incapacitated.

Heat cramps. In the literature, they also occur as heat seizures, spasmous disease of overheating.

They are due to disturbances in the water-mineral balance with severe hyponatremia due to the substitution of the profusion perspiration only by the intake of water. They are characterized by slow, highly painful, tonic contractions of the muscles involved in work process by 1 to 3 minute duration. The skin is cold and damp. The muscle groups covered are solid. Body temperature is normal or slightly elevated. *Differential-diagnostic* is needed to distinguish heat cramps from those with another genesis - hypothyroidism, myotonia, radiculopathies, hypokalemia, lateral amyotrophic sclerosis, neuropathies, hypomagnesaemia, myophosphorylase deficiency or phosphofructokinase deficiency and others. *Treatment* involves placing the patient in a room with a comfortable microclimate and physiological saline infusion or oral uptake of 1-2% salted water for 1 to 3 days.

Heat syncope. It is much more common than the heat stroke. It is characterized by sudden loss of consciousness due to reduced blood volume in cerebral blood vessels and cerebral hypotension. The skin is cold and damp, the pulse is weak. Systolic blood pressure is below 100 mm Hg. The *treatment* consists of resting on a favorable microclimate, cooling and hydration of the patient.

Chronic effects of overheating microclimate increase the risk of ***gastrointestinal diseases*** (ulcer disease, gastro-enterocolitis), ***cardiovascular diseases*** (arterial hypotension, later arterial hypertension, early atherosclerosis), ***avitaminosis, neurotic conditions, anemic syndrome***.

Solar shock is a form of overheating due to the direct action of the sun's rays on the unprotected head. It is characterized by headache, vertigo, fainting, weakness, tinnitus, nausea, vomiting. The face is red and damp. Unlike the heat stroke, hyperthermia is not detected. In the heavier stages there is a loss of consciousness, agitation, epileptic seizures, hallucinations. *Treatment* is symptomatic.

Skin diseases due to thermal effects. Canal occlusion of sweat glands in workers in conditions of elevated temperature and humidity leads to sweaty retention with vesicle formation, erythema, desquamation, macules and is called **miliaria**. They are **milaria crystallina** (very superficial), **miliaria rubra** (deeper in the derma) and **miliaria profunda** (the deepest clogging). Secondary staphylococcal rashes form **miliaria alba**.

In body folds and friction surfaces, an **intertrigo (dermatitis intertriginosa)** may be formed - an inflammatory process resulting from heat-sweating with erythematous and macerated skin.

In the case of electric welders, fireman, furnaces, etc., more often in the exposed parts of the body are formed single or grouped **teleangiectasiae**.

Localized or generalized heat urticaria **(urticaria a calore)**, summer itching **(pruritus aestivalis)**, erythem by fire **(erythema ab igne)**.

In the *treatment* of these diseases it is required to reduce or discontinue the thermal exposure and reduce the perspiration.

Burning (combustio) is an accident at work. The severity and prognosis of burns depend on the affected area, the depth of the affected tissues, the location on the human body, the age of the patient, the accompanying diseases and traumas. Heavy burns are more than 15% area for adults, over 3% of the face or haired part of the head and prognostically bad for total skin necrosis of over 20%. Depending on the depth of tissue burning, the following are distinguished: I degree - erythematous and painful edema; grade II - blooms are formed with clear serous fluid, which later becomes darker, resorted to the formation of crusts; III degree - skin necrosis; IV degree - necrosis of the skin and underlying tissues. Clinically severe burns undergo a phase of thermal shock, toxic infection, restoration phase, injury cachexia and the consequences of hypertrophic scarring, keloids and carcinogenic degeneration. *Treatment* of patients with thermal burns is undertaken by a team in an intensive care unit for emergency care. It is continuous and gradual (depending on the degree and severity of burning).

Occupational medical expertise. In the beginning, the patient is temporarily incapacitated, and often temporary or permanent change of work, is required to work without overheating microclimate (with persistent hypersensitivity to thermal effects). In severe conditions, a disability group is defined.

The prevention of thermal damage besides sanitary-technical measures requires a rational drinking and diet with 0.5% salted water, fruit juices rich in mineral salts, vitamins and trace elements, protein-carbohydrate diet, diet "A" of the professional protective nutrition. Regular work breaks are required in cool rooms. Do not work in heat-affected conditions patients with cardiovascular diseases, kidney and endocrine-exchange disorders, ulcerative disease, neuropsychiatric disorders. Periodic medical examinations once a year with the participation of an internist, ophthalmologist, dermatologist.

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L. Nakova

4.3 RADIO-WAVE DISABILITIES

The modern electronics industry is characterized by the widespread use of electronic devices for generating high frequency currents that excite in space electromagnetic fields (EMFs) with a certain physical characteristic.

EMF generators are widely used in modern life and work, in industry - radio electronics, instrumentation, agriculture, medicine, television and radio relay links, science.

The biological action of EMF increases with the intensity of the field, depending on its frequency and the time of exposure. The efficacy of the responses depends on individual characteristics.

In intensive EMF irradiance exceeding 10,000 W/cm^2, the absorbed energy is transformed into heat. It accelerates the metabolic processes and induces, on an interceptive basis, dystrophic changes of some brain structures and myocardium in mammals. The highest heat effect in humans is given by VHF fields, absorbed by less blood-flowing organs - eye lens, testicles, bladder. Under the conditions of modern labour and after the introduction of preventive measures (shielding, special protective means) the intensity of irradiation is below that of the thermal effect. In experimental and clinical medicine, the ***non-thermal specific action of radio frequency EMFs*** *is now leading. Functional changes in the central nervous system (CNS), predominantly in the autonomic nervous system (CNS), and in the cardiovascular system (SCD), have been shown to be essential. The biophysical nature of subtermal, chronic microwave irradiation suggests phenomena of molecular paramagnetic resonance, non-thermal denaturation of proteins, and alterations in the microstructure of the living cell. In mammalian experiments, the most sensitive microwaves are the cortex, hypothalamus and cardiovascular structures.*

The pathogenetic mechanism of radiofrequency response is thought to be indirectly mediated through the brain centers of cardiovascular regulation, after which reflex-induced hemodynamic disorders occur. Endocrine-humoral changes have also been observed - cortisol rhythm abnormalities and electrolyte balance; disturbances in neuromuscular transmission activity; disorders of erythropoiesis and leukocyte formula. Immunological studies have shown that VHF-EMF inhibits the formation of antibodies to exo-allergens in mammals and causes autoimmune reactions. Suppress the adaptive capacity of the body.

Occupational radiofrequencies are pathological conditions caused by the acute intense or chronic exposure of microwaves with sub-thermal intensity in the production environment.

The acute impact of radio frequency EMF on health is rarely seen - in the case of accidents to HF generators or rough violation of the technical safety rules. Intense irradiation provokes the thermal effect of EMF on the body. Workers complain of fainting, palpitations, relapsing headaches, increased body temperature, thirst, cramps of the skeletal muscles. After removal the person from the work environment and symptomatic treatment, complaints resolve for 24 hours to several days.

In chronic influence, a leading place in the so-called radio wave pathology occupy the functional changes in CNS activity and cardiovascular regulation. Clinical manifestations can be represented by the following syndromes:

- Cerebroasthenic;
- Angiodystonic (hypertensive or hypotensive type);
- Hypothalamic (diencephal) syndrome.

Cerebroasthenic syndrome occurs in the initial 3-5 years of EMF exposure with non-specific, neurosis-similar symptoms: headache, irritability, insomnia, emotional lability, decreased working capacity and mild memory impairment. Addition of cardiovascular changes: blood pressure fluctuations, rhythm disorders - extrasystolesia, sinus bradycardia, AV block, etc.

Vegeto-vascular dysfunction of the hypotensive type has a more favorable pass - systolic pressure is decreased by 30-40 mmHg at a preserved diastolic boundary. More persistent for treatment are people with hypertonic crises (RR to 180/120 mmHg). The development of hypo- or hypertension during the course of neurocirculation dystonia is also determined by the baseline of the body, and possibly by the characteristics of the electromagnetic factor and the specific working conditions (psycho-sensory intension).

At an advanced stage of chronic radio wave exposure, hypothalamic insufficiency develops. It occurs with the so-called diencephalic crises - anxiety, tremor of the limbs, profuse sweating, redness or paleness of the skin, subfebrility, changes in blood pressure, headaches, palpitations and transient pain, dysmenorrhoea in women, Paraclinic disorders of carbohydrate tolerance, electrolyte imbalance, changes in leukocyte formula and thromboelastogram, increased excretion of catecholamines are

detected. ECG shows depression of the ST segment in precordial leads, ventricular extrasystoles, less frequently AV block. Functional studies show increased vascular tone or pronounced angiodystonic changes - capillary spasm in the peripheral vessels of the limbs and the retina. In ultrasound diagnosis (Doppler sonography), increased vascular resistance of the digital arteries of upper limbs or vertebral vessels is reported.

The EEG in human beings subjected to low intensity radio waves shows at the beginning of the effect the activation of bioelectric activity, synchronization of oscillations, at the end of the work shift - predominance of the slow wave activity, as an expression of a progressive increase of the cortex retention process. The changes are of a phase character. Degenerative changes of the lens of the eye (cataract) are also often observed.

The described syndromology in its sequence (cerebrasthenic symptomatology, vegetative-vasal dysfunction and hypothalamic insufficiency) determines the stage pass of radio wave pathology. For the time being, it is not considered as an individual nosological unit (radio wave disease) and is not regulated by labour law (except for microwave cataracts).

The diagnostic process of radio wave effects on the human body is difficult due to the expressed polymorphism and the non-specificity of the responses. This implies an in-depth analysis of symptomatology in people who have been in contact with EMF for more than 10 years, as well as a dynamic tracking of their condition.

Differential diagnosis of the described disorders is made with systemic diseases, occurring with triadic cerebrasthetic manifestations, vegeto-vascular dystonia and hypothalamic dysfunction. First of all, hypertonic disease, ischemic heart disease and rhythm disorders of organic origin should be eliminated. Hypothalamic syndrome involves the exclusion of infectious, atherosclerotic or traumatic origin. It is necessary to specify other etiological factors for development of diencephalic pathology: endocrine disorders (diabetes, thyrotoxicosis), collagenosis, traumatic brain disease, early atherosclerosis, household intoxications, alcoholism, psychogenic moments, etc.

Treatment of radio wave injuries is symptomatic. Serum therapy, neuroleptics and antidepressants are administered in cerebrasthenic conditions and angioneurodystones. Migraine-like cephalalgies are well-affected by the use of combined-type preparations (Paracetamol or Efferalgan - 3 x 0.500 g daily, Acetofen - 3 x 1 tablet daily). Also included are vegetative harmonizers (Atropa Belladona alcaloides - Bellergamin - 3 x 1 tablet daily; Opipramoli chloridum - 2 x 0.050 g daily for a long time - 2 to 5 months). Pain allaying procedures, physiotherapy and balneotherapy are recommended in climate zones with negative aeroionization (Bankya, Etropole, Rhodope, etc.). Arterial hypertension is treated according to the classical principles of internal medicine. The diencephalic crises are treated with Carbamazepin 0.200 - 0.400 g daily for 2 months with vitamins B, Geritamin and vasoactive agents Nattidrofurilum (Dusodril) 3 x 1 tablet daily, Pentoxiphyllinum 0.200 - 0.400 g daily (Agapurin).

Prevention. The unfavorable impact of the EMF requires strict adherence to the engineering - technical rules to prevent intensities up from hygienic norms.

We recommend general wellness activities, a rational organization of work.

Do not work in environments with EMF persons with chronic diseases of the cardiovascular and central nervous system, blood diseases, vegetative dysfunction and mental illness, cataracts, endocrine gland diseases. Periodic medical examinations - 12-24 months, with neurologist, cardiologist and ophthalmologist.

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4.4 RADIATION ILLNESS

All living organisms are constantly exposed to ionizing radiation, which naturally exists in nature. According to UN data, the average annual effective dose due to natural sources of radiation is 2.4 mSv per person (ranging from 1 mSv to 10 mSv, depending on the specifics of a region).

With the widespread penetration of ionizing radiation in the different spheres of life - economics, industry, agriculture and science, including biology and medicine, the number of persons professionally associated with ionizing radiation is growing rapidly.

Various activities involving the use of ionizing radiation lead to additional exposure.

The basis of the biological action of ionizing radiation is the damage to the genome of the cell, namely DNA, which is mainly the initial event, whereby radiation causes long-term damage to organs and tissues of the body.

There are three main types of DNA damage, respectively chromosomes: nitrogen basis damage, single-chain disruption, and double-chain break. In most cases, these lesions are recovered spontaneously, and non-recovered result in one of two possible outcomes: cell death or cell survival but with impaired metabolism.

The function of most organs and tissues of the body does not affect even the loss of a significant number of cells, but if this loss passes a certain threshold there are harmful effects on the body. This type of effects that occurs in all subjects receiving acute irradiation with a dose ***above*** *a certain threshold dose for the effect is called* ***deterministic effects****.*

The main features of the deterministic effects are the following: the result is cell death; the effect always occurs if the threshold dose for the effect is passed; the severity of occurrence of the effect increases with increasing dose; the dose-effect curve is sigmoid; the dose power significantly influences the appearance of the effect; the threshold dose is relatively high and the latency period relatively short.

Such deterministic effects are: acute radiation disease, chronic radiation disease, local radiation damages, radiation cataracts, pulmonary fibrosis, and the like.

The early deterministic effects can be:

Skin irradiation: *erythema, phlyctena, edema, ulceration or tissue necrosis;*

Hemopoietic tissue irradiation: *Reduction of blood cells from lymphoid, myeloid, platelets and erythrocyte lines (initially with suppression and then with aplasia).*

Gastrointestinal tract irradiation: *the mucosa of the small intestine is the most vulnerable as the fastest dividing tissue in the intestinal tract. The clinical outcome is: nausea, vomiting, diarrhea, haemorrhage, perforation of the intestine, septicemia.*

Reproductive system irradiation: *reduction of fertility or oligospermia, sterility or endocrine disruption in women.*

Nervous system irradiation: *usually does not lead to clinical manifestations, but at higher doses, neuro-circulatory disorders, EEG changes, cerebral vascular damage, and even comatose status may occur.*

Whole body irradiation has combined effects from various organs and systems.

Unrehabilitated DNA damage *(which did not cause cell death) leads to cell modification. This modification is transmitted to the daughter cells and could lead to the development of neoplasms. If the modified cell is reproductive, then the next generation may increase the incidence of hereditary diseases. These effects of ionizing radiation (neoplasms and hereditary diseases) can not be predicted for one individual (they are predicted and evaluated at population level) and because of their probability character they are called* ***stochastic effects****.*

Stochastic effects can be caused by the damage of a single cell. However, as the absorbed dose increases, the number of damaged cells increases and the likelihood of the stochastic effect increases.

The main features of stochastic effects are: no threshold dose; as the dose increases, the likelihood of the effect increases; the severity of the effect does not depend on the increase in the dose; the dose power may not significantly affect the risk; the dose-effect curve is more or less linear (conservative approach for maximal radioprotection); the latency period is long (years); the individual risk is low even after high doses, but the population risk is significant.

4.4.1. ACUTE RADIATION ILLNESS

Acute radiation disease is a nosological form that develops after an external X-ray, gamma and/or neutron irradiation over a dose of 1 Gy obtained

for a short time (up to 10 days) or after incorporation of radionuclides (creating an adequate absorbed dose). This is the most significant deterministic effect of ionizing radiation.

The irradiation may be generally uniform or fractionated, relatively short-lived or prolonged external irradiation; combined - external and internal radiation; combined action of radiation and non-radiation factors.

Pathogenesis: Acute radiation occurs as a result of the development of complex biological processes caused by the interaction of the energy of ionizing radiation with the cells, tissues and the fluid environment of the organism. The primary biological activity of radiation is realized through physical, physico-chemical and chemical processes with the formation of active free radicals (H +, OH, HO_2), which have strong oxidation properties. In the process of radiation damage or immediately afterwards, regeneration processes are formed with formation of various peroxide compounds (H_2O_2, etc.)

Oxidative radicals and organic peroxides alter enzyme activity and thus influence autolysis processes in tissues. The activation of the ferments increases the permeability of the cell membranes. As a result of the destruction of radio-sensitive tissues and pathological metabolism, the manifestations of toxaemia are formed.

High is the radiosensitivity of rapidly regenerating tissues: haematopoietic tissue, intestinal epithelium and skin. The nervous system in the elderly is not a renewing cellular system but also has a high sensitivity.

Depending on the type of irradiation, two major variants of the disease can be most commonly developed: acute radiation with relatively uniform radiation and acute radiation with irregular irradiation.

1. Acute radiation illness at relatively even radiation

This is the typical variant of acute radiation illness. It is characterized by undulation or periodicity of run, which is due to the successive defeat of the critical systems of the body. This is best expressed in the bone-marrow form developing at radiation doses of 1 Gy - 10 Gy. In the other forms - intestinal (10-20 Gy absorbed dose), toxic (20-80 Gy) and cerebral (over 80 Gy) - due to the severe course and rapid onset of death, the typical periods and phases of the radiation illness no distinguished **(Table 1)**.

The most clinically important from a therapeutic point of view is the bone-marrow form of acute radiation disease, as the probability of survive it is greatest.

Clinical picture: There are three periods in the course of the bone marrow form of acute radiation: a period of disease formation (about 2 months), a recovery period (2-4 months to 3 years), and a disease outbreak period (and of the consequences).

The period of acute radiation illness formation is divided into 4 phases: phase of primary reactions, latent phase, disease development and primary (immediate) recovery. The total duration of this period is about 2 months (the duration of the period is shorter at a higher dose of irradiation).

Symptoms in **the primary reactions phase** can be divided into 4 groups:

- dyspeptic - nausea, vomiting, diarrhea;
- general clinical - sleep disturbances, weakness (tiredness), headache, changes in motor activity, temperature rise, tachycardia;
- haematological - lymphocytopenia, neutrophilic leukocytosis;
- local - changes in the skin and mucous membranes in the areas with the highest irradiation.

These prodromal responses can play an important role in assessing the severity of radiation damage and in predicting the clinical course. Particular significance is attributable to symptoms such as nausea, vomiting, diarrhea, erythema, increased body temperature. What matters is the time of their occurrence, frequency and severity. Several quanti-

Tab. 1. Clinical forms, degrees of severity and prognosis of acute radiation illness in whole-body irradiation

Dose (Gy)	Clinical form	Levels of severity	Prognosis	Outcome
1-2	Bone marrow	Light (I)	Absolutely favorable	Good 0% lethality
2-4	Bone marrow	Average(II)	Relatively favorable	Lethal to 0-50% in 6-8 weeks
4-6	Bone marrow	Severe (III)	Questionable	Lethal in 20 to 70% in 4-8 weeks
6-10	Bone marrow	Extremely severe (IV)	Unfavorable	Lethal in 50-100% in 1-2 weeks
10-20	Intestinal			Lethal 8-10 day
20-80	Toxemic			Lethal 4-7 day
>80	Cerebral			Lethal 1-3 day

tative indicators, such as absolute lymphocytopenia in the first (3) days, and leukocytosis over 12.10^9/l, also have an unfavorable prognostic significance. Bone marrow decreases the mitotic index, decreases the number of young forms and increases the leuco-erythroblastic factor. Circulating lymphocytes are highly radiosensitive and the magnitude of their absolute count deminution in peripheral blood is a widely used laboratory test for the determination of the severity and prognosis of acute radiation illness.

Widespread and reliable bio-dosimetry is also cytogenetic analysis - the frequency of chromosomal aberrations in cultured peripheral blood lymphocytes is reported. Screening for micronuclei in peripheral blood lymphocytes can also be used.

During the **latent phase**, prodromal reactions are reduced, the general condition improves. There are only some discreetly manifested clinical symptoms such as vegetative regulation lability, general asthenia, pulse and arterial pressure lability.

Most attention is paid to the dynamics of haematological parameters and cytopenia (it progresses) during the latent phase. In severe cases, anemia can also occur. At doses above 3 Gy, epilation also occurs.

The duration of the latent phase is from 3 to 30 days.

The clinical transition from the latent phase to **the disease development phase** occurs abruptly. Decreased self-esteem, appetite decreases, general weakness is increased, fever appears. Tachycardia, arterial hypotension, deafened heart tones, changes in the distal part of the cardiogram were found in the clinical study. The clinical picture is dominated by infectious-toxic complications in the nose, mouth, pharynx, respiratory tract, digestive tract. Severe ulcer-necrotic enteritis and enterocolitis can lead to peritonitis, bowel obstruction, sepsis.

Mandatory manifestations are skin and mucosal hemorrhages.

At the stage of the disease development, symptomatology is caused by the depression of bone marrow bleeding. There is abrupt leukopenia, thrombocytopenia, agranulocytosis.

Adverse prognostic features include head and body epilation, short or missing latent phase, severe bone marrow damage with primarily reticulous and plasma cells, peripheral blood monocytes deficiency, high BSR, high blood sugar, leukopenia under $0.5.10^9$/l, neutrophil count less than lymphocyte count, delayed appearance of young cellular forms in peripheral blood.

The duration of this phase is 2-3 weeks.

The onset of **the primary (partial) recovery phase** begins at the outbreak of the agranulocytosis patients. Temperature is decreasing, self-esteem increases, appetite recovers. Clear recovery of peripheral blood parameters is observed. The bone marrow has processes of active regeneration. Hair begins to grow, the condition of the mucous membranes and the skin improves. For a long time, however, manifestations of astheno-vegetative syndrome with reduced working capacity, diminished memory, headache, muscle pain, impaired neuro-humoral mechanisms, vegetative-endocrine and vascular dysfunctions remain. The duration of the phase is 1-2 months.

Treatment. Contemporary treatment of the victims of multiple radiation emergencies has shown that the 4 Gy of the semi-lethal dose can be increased and patients can survive even at radiation doses of 10 Gy. The treatment is directed to priming reactions, restoring the activity of the hematopoietic organs, fighting infectious complications and preventing and treating haemorrhages.

During the primary reactions phase, anti-emetic, blood-sweetening and detoxifying agents, preparations for metabolic balance maintenance are used. Cardiotonic and vasoactive agents are used for collapse and marked cardiovascular weakness.

During the latent phase, therapy consists of administering sedative and polyvitamin preparations, assessing the need for antibiotic therapy, and discussing the feasibility of bone marrow transplantation.

At the development of the disease, a guiding principle is to manage the complications arising from bone marrow depression - infections and haemorrhages. Treatment is targeted to combat infections by administering a combination of broad spectrum bactericidal antibiotics at maximum therapeutic doses, controlling hemorrhages by substitution therapy and haemostatics, detoxifying agents, polyvitamin preparations.

Currently, various hematopoietic growth factors are used to accelerate the repair of the bone marrow.

Bone marrow transplantation has a limited role and can only benefit those whose bone marrow is completely damaged. Bone marrow transplantation should be considered only for subjects who receive doses of the order of 8-12 Gy evenly distributed throughout the body without serious skin and other lesions.

Nutritional protein hydrolysates and hydrolysates of nucleic acids are administered to the patients.

In the primary recovery phase, the treatment of patients includes anabolic and vitamin preparations, central nervous system toning agents, protein-rich foods.

2. Acute radiation disease at irregular radiation

The clinical picture of acute radiation illness in uneven body irradiation is most often combined with signs of local radiation damage: radial dermatitis, pharyngitis, gastrointestinal tract damage. Radiated dermatitis occurs after irradiation of the skin and mucous membranes, depending on the dose and radiation in time skin changes may be early and late.

Clinical evolution of local radiation injury passes through three stages:

1. Inflammation: hyperremia and edema
2. Passive congestion: thrombosis and ischemia
3. Necrosis and fibrosis

Early radiodermatitis occurs after irradiation at a dose of at least 5 Gy. The development of acute radiodermatitis is shown in **Table 2.**

Most often acute local radiation injuries are observed today with defectoscopes (portable gamma-defectoscopes). The damage is mainly on the wrists and rarely on other parts of the body.

Late radiodermites are observed after systemic irradiation, mostly in the exposed parts of the body with X-rays or gamma rays. The first stage is dry skin atrophy. The second stage is characterized by ulceration. These two stages are considered precancerous. In the third stage, skin cancer occurs.

The most common radiographic lesions of other organs and systems are: haematological changes; eye changes - blepharitis, conjunctivitis, late cataracts; gonade changes.

Primary erythema is not treated as it goes spontaneously. The onset of secondary erythema (1-3 weeks) is an indication for **treatment**. Analgesics, non-steroidal anti-inflammatory drugs, topical antibiotics, vasodilators, anti-aggregates, epithelotonics, antiproteolytics are used. If necessary for surgical treatment it is done under an antibiotic umbrella.

3. Incorporation of radioactive substances

The fall of radioactive substances into the body can occur in several ways - respiratory, digestive, wounds and skin lesions and, exceptionally, percutaneous.

In the case of the incorporation of uniformly distributed radionuclides with a relatively short half-life, the clinical picture coincides with that of acute radiation, and in the other radionuclides it is dependent on isotopic accumulation in the relevant critical organ (bone marrow, lung, liver and others.).

If signs of long-lasting half-life of radioisotopes fall into the body, signs of chronic radiation can develop with certain organ manifestations. In less frequent cases, late effects may occur in the form of various malignant neoplasms.

To determine the type, concentration and activity of the radioactive substances, the radiophysical (determination of whole-body activity) and radiochemical (radiotoxicological) methods of investigation are applied.

Radiotoxicological analysis is carried out by examining biological excreta (urine, faeces, washes, vomiting, etc.) collected in the first hours after the accident.

In the treatment of radiation damage caused by poisoning with radioactive substances, early and complex measures are being implemented aimed at the accelerated removal of isotopes from the body. In case of poisoning with well-soluble and readily removable isotopes (etc. ^{137}Cs), it is advisable to drink plenty of fluids - forcing diuresis and boosting metabolic processes in the body.

In many cases complexe formative compounds (complexons, chelates) for their removal from the body - polyamines, polyaminocarbolic acid (EDTA), some organic acids (oxalic acid, citric acid), unithiol (in case of poisoning with cerium, zirconium, plutonium, etc.) are used.

Through the surface of undamaged skin, the radioisotopes of iodine, sodium, tritium, strontium, radium, plutonium can be absorbed. Fast and careful washing with lukewarm water and soap reduces by 90% the radioactive contamination. Special detergents, adsorbents and ion-exchange resins are also used. Do not apply alcohol and ether due to skin degreasing, and enhancing resorption.

Emetic, laxatives, cleansing enemas, absorption of many fluids, diuretics, and even (by circulating the isotope in the blood) hemodialysis are applied when gastrointestinal isotopes are present. Thyrotropic

Tab. 2. Acute radiation dermatitis

Degree of radiodermitis	Absorbed dose Gy	Duration of activity (days)	Lesions	Duration	Consequences
First - radio erythematous dermatitis	5-6	10-20	Erythema + pigmentation itching, desquamation	1 week	Temporary hair removal
Second - bullous radiodermit	15-25	8-10	Intense erythema, followed by phlyctenas and ulceration	1-2 months	Definitive pigmentation and hair removal
Third - ulcerative radiodermit	> 25	Some days	Intense erythema, edema, painful bulls, deep ulcerations	Months or years	Scarring, sclerosis

hormone is used to remove iodine 131 from the thyroid gland.

4. Acute relatively even irradiation at doses up to 1 Gy

In single exposure at doses up to 250 mSv there are no subjective complaints and no pathological abnormalities in the paraclinical parameters that can be established with the current methods of investigation.

At doses of 0.25-0.5 Gy, only some transient reactions by individual systems of the organism (nervous, hematopoietic system) are detected.

Dosages of the order of 0.5-1 Gy may cause so-called preclinical form of acute radiation illness. It is characterized by transient manifestations of neuro-visceral dysregulation (cardiovascular, endocrine system) and transient haematological changes (lymphopenia, leucopenia). In this preclinical form **an ambulatory treatment** is carried out with the use of symptomatic means, general strengthening means and compulsory labour outside of the environment of ionizing radiation. Compensation period (without additional dose load) applyed on the injured person, the length of which depends on the amount of dose received.

4.4.2. CHRONIC RADIATION DISEASE

Chronic radiation disease (CRD) is a single nosological form of radiation damage that develops in chronic irradiation (externally, internally) of the organism at doses exceeding the permissible levels. Today in our country there are no cases of chronic radiation disease due to the strict radiation-hygienic control over the working conditions and the dynamic medical surveillance of all persons working in the environment of ionizing radiation. CRD is characterized by damage to a number of organs and systems and depends on the total radiation dose, its spatial distribution, the intensity of the radiation and the physiological features of the organism.

Chronic radiation illness develops at a radiation intensity of 1-5 mGy/day at a total dose of 0.7-1 Gy. It can occur with 2 types of radiation: a long total relatively uniform irradiation and a continuous total unequable irradiation (irradiation of body segments). It should be in mind that the biological action of total irradiation is always greater than that of the local exposure at the same exposure dose.

For chronic radiation disease, the long-term wavy clinical course of development is characteristic, often with simultaneous rehabilitation reactions.

Active proliferating tissues (with intense cell division processes) have long retained their morphological recovery capabilities, while stable in this regard - the nervous, endocrine, cardiovascular system, respond to chronic radiation with complex functional responses in delayed dystrophic changes.

Chronic radiation illness has several stages of development.

During **the first stage** there are functional violations in the activity of individual organs and initial vascular changes. The main syndrome is neurocirculation dystonia. Permanent changes in peripheral blood (leukopenia with lymphocytosis) are observed.

The second stage is characterized by gradual intensification of the above phenomena, trophic skin changes, memory weakness and eventually signs of organic damage to the central nervous system.

At **the third stage** of the disease there are severe complications in the activity of all organs and systems. The restoration capacity of individual tissues is completely lost. There is a severe adynamia accompanied by haemorrhages from the mucous membranes and the internal organs and pronounced nervous symptoms.

In the clinical aspect, the 4 grades of CRD are distinguished:

First (mild) grade: characterized by disorders of neural visceral regulation (easy tiredness, general weakness, loss of appetite, pulse lability and blood pressure, sweating, functional gastrointestinal disturbances, changes in libido, manifestations of dysmenorrhea in women, etc.). All these violations are reversible. Bone marrow is affected by normal bleeding processes. In peripheral blood there is unstable leukopenia, sometimes combined with thrombocytopenia.

Second (moderate) degree: Deepening of regulatory disorders to a state of functional failure of the nervous, cardiovascular, digestive and other systems with added morphological lesions of the most radiosensitive peripheral blood elements - leukocytes and platelets. Some disorders of metabolic processes in the body may also occur.

Third (severe) stage: Clinical symptoms are quite diverse: on the nervous system - signs of distracted encephalomyelitis; in the cardiovascular system - myocardiodistrophic changes, endothelial damage of medium and small caliber vessels, presence of circulatory disorders; development of atrophic processes in the gastrointestinal tract. Changes in the bone marrow and peripheral blood are getting worse. An infectious-septic condition often occurs as a consequence of disorders in the immune system.

Fourth (extremely severe) grade: characterized by infectious complications, pancytopenia and others. Changes in the nervous and cardiovascular system are deepened. The parallel development of

reparative processes can lead to new forms of regulation of a number of systems in the body.

The prognosis in stages I and II is still good, in third stage patients it is doubtful or bad and in grade 4 -bad.

For the diagnosis and treatment of CRD, it is also necessary to:

- review after a detailed detachment of the history;
- detailed laboratory tests (including bone marrow puncture if necessary);
- cytogenetic examination of peripheral blood lymphocytes;
- consultation with an ophthalmologist (risk of developing cataracts);
- conducting radiometric examination of the thyroid gland, biological excreta, determination of whole body activity of the organism (anthropogamametry), etc.

The basic principles of **treatment** coincide with those of acute radiation illness. The main efforts are directed towards the general strengthening of the nervous system, the treatment of the manifestations of neuro-visceral dysregulation and the bone marrow insufficiency. Careful use of hemogenic stimulants, immunostimulants, antibiotics and anti-haemorrhagic agents is necessary. To enhance the body's vitality and reactivity, preparations that stimulate the function of the central nervous system and sanatorium-resort treatment are used.

Patients with chronic radiation illness are constantly labour readjustment and carefully refining the need for X-ray and radioisotope research in the future.

Medical prophylaxis (health monitoring) of workers exposed to ionizing radiation

The medical units that carry out health monitoring of the professionally irradiated persons in Bulgaria are:

- National Center for Radiobiology and Radiation Protection - central diagnostic expert and medical-advisory unit on the issues of radiation pathology;
- RIPCPH (RIOCOZ, HEI) - Radiation hygiene departments, in Plovdiv, Varna, Burgas, Rousse and Vratsa;
- Occupational Medicine Service at Kozloduy NPS;
- Departments (8) to combat silicosis (for former uranium miners);
- Military Medical Academy (for employees of the Ministry of Defense).

Health monitoring is carried out on more than 20 000 persons - working in Kozloduy NPS - 5000; workers in other industries (medicine, industry, science) - 11,000 and over 4,000 former uranium miners.

The types of health monitoring are:

- usual: preliminary medical examinations, periodic medical examinations and medical examinations of former workers;
- operational: before and after refueling of nuclear reactors, before performing risky operations and manipulations, in case of emergency exposure of the body;
- special: in emergency radiation of the body.

The volume of the preliminary medical examination includes:

- Clinical examination by: therapist (radiobiologist), neurologist, ophthalmologist, surgeon, dermatologist, gynecologist;
- Peripheral blood study - hemoglobin, erythrocytes, reticulocytes, hematocrit, differential count leukocytes, platelets, BSR;
- Urine analysis - relative weight, protein, sugar, ketobodies, bilirubin, urobilinogen, blood, sediment;
- Electrocardiography, sphygmometry. For operational and repair personnel at the NPS - and review by an otorhinolaryngologist; For NPS operators - and psychophysiological and psychological research.

The volume of periodic medical examination is as a preliminary medical examination, with additional research - biochemical, immunological, cytochemical, cytogenetic, electrophysiological, biophysical, radiochemical, etc. at indications.

The choice of diagnostic methods for the incorporation of radionuclides is determined by the nature and amount of the incorporated isotope, by its toxicological characteristics and by the dose load formation.

Where there is a doubt about the occupational disease from the radiation exposure - leukosis, cancer, cataracts, etc., the worker is referred to LEMC (TELC).

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V. Kostova

4.5 VIBRATIONAL DISEASE

Vibration disabilities continue to be of interest to professional pathology despite their long-standing research in various fields. In 1883, the use of pneumatic vibratory instruments in French mines was introduced, but only in 1911 G. Loriga described for the first time vasomotor disorders in the fingers after the use of pneumatic guns. This message was followed by the classical studies of vascular syndrome by A. Hamilton (1918), T. Rothstein (1918) and J. Leake (1918).

In our country, vibrational lesions are described as a nosological unit, which includes the following definition: a specific occupational disease that develops progressively, progresses phase, and is characterized by a polymorphic clinical picture with leading nervous system manifestations (Methodological instruction - MH) - 0-56/79).

The term vibrational disease (identified as a single nosological unit by E.C. Andreeva-Galanina - 1955) is used in Japan, the former USSR and Eastern Europe and the Scandinavian countries. In the Anglo-Saxon literature the synonyms of the disease are vibration syndrome, traumatic vasospastic disease, Raynaud's professional phenomenon or Raynaud's professional disease, vibrational "dead" fingers, hand-arm vibration syndrome and others.

In the structure of occupational morbidity in Bulgaria vibration disease (VD) is third and shows a tendency to decrease its relative share in recent years - from 21.2% in 1996 to 9.6% in 1998 (related to a number of factors, diminished disclosure of occupational diseases, restructuring of industry, health care reform, etc.). Its gender and age distribution shows a higher incidence of male sex and the highest incidence in the 46-55 age group. The greatest share of VD is over the duration of the specialized service over 10 years.

Etiology of vibrational disease

The disease is caused by complex unfavorable factors of production - local or general vibration, excessive noise, unfavorable microclimate, dynamic load, static and psycho-emotional overtension, and as leading factor in etiology - vibrations.

According to most authors, a major role in the effect on the human organism and the clinical symptom is the role of vibration frequencies. It is assumed that vibrations with frequencies between 6 and 16 Hz cause predominantly motor disorders, autonomic disorders and spinal lesions; frequencies 16-30/40 Hz lead to bone-joint injuries in the hands and shoulders; effects on peripheral blood vessels and neuro-muscular structures occur at frequencies between 30/40 and 1000 Hz. According to other researchers, the most pathogenic impact on the development of vibropathology has the frequencies of 16-250 Hz.

Major pathogenetic mechanisms

There is no single theory about the mechanisms under which the VD develops. It is assumed that its pathogenesis is multifactorial, with different pathogenetic mechanisms involved in the various stages of the disease. Several hypotheses find a major place in explaining vibration pathology.

The greatest number of supporters has the theory of **direct mechanical damage to peripheral structures** *in the extremities of the vibrations, resulting in the following changes:*

- in the arterial wall: lamina interna disruption, endothelial desquamation with subsequent release, adherence and platelet aggregation at the site of the damage; proliferation of smooth muscle cells; thickening of intimate small blood vessels; fibro-cellular thickening of arteriol and arterial walls; development of focal cell proliferation; regenerative formation of collagen and elastin; increased collagenisation of the dermis, vascular walls and perivascular spaces.

- peripheral neural lesions - damage to vasomotor nerve endings, axonal degeneration, demyelinization, and extensive collagenisation of perineurium and endoneurium.

- direct damage to the mechanoceptors in the fingers - mostly fast adapting units II, associated with Pacini's bodies (and perhaps with Golgi-Mazoni's bodies): sensitized for frequencies above 40-50 Hz and SA I-type (slowly adapting units I), including Merkel's disks and subsequent dysfunction and hypofunction of receptor system.

- influencing "local" or peripheral factors involved in arterial tone control - so-called vascular tone modulators associated with the endothelium: thromboxane A2 (potential vasoconstrictor); prostacyclin (potential vasodilator); endothelin-1, von Willebrand-Factor antigen, ACE and other endothelin-dependent substances, and causing vascular spasm and impaired circulation.

Vasoconstriction and increased vascular tone are explained in addition to direct, local action and with a general effect of the induced **dysfunction of the**

autonomic nervous system*: suppression of its parasympathetic part and especially affection of sympathetic nervous system - sympathetic hyperactivity with activation of central sympathetic reflex mechanisms (limbic system and hypothalamus).*

It is also assumed that the complex of adverse factors (including vibrations) unlocks **the response of chronic or intermittent stress** *with the activation of the hypothalamic-pituitary-adrenal cortex and sympathic-adrenal system and their subsequent dysfunction. As a result, there are neuro-humoral and enzymatic disorders and disorders of tissue metabolism and homeostasis of the whole organism.*

The Classification of Vibrational Disease (MH Methodological guideline - 0-56/79) defines:

- VD due to local vibrational effects
- VD, due to general vibrational impact.

Depending on the degree of disability, three stages of the disease are indicated:

- Stage I (initial);
- Stage II (moderate);
- Stage III (expanded, expressed).

Clinical and labour-expert practice also uses intermediate stages:

- VD I-II stage (transient);
- VD II-III stage (advanced, with chronic leakage).

4.5.1. VIBRATORY DISEASE FROM LOCAL VIBRATIONS

Threatened occupations are: miners; woodworkers; band sowers; polishers; grinders; rough grinders; riveters; sharpenters; mosaicists and others.

The tingling of the fingers is one of the early signs of the disease. Appears at night, after work started, usually disappears. These complaints are accompanied by morning stiffness of the fingers and diffuse swallow pain in the upper limbs. Characteristic are spontaneous fits of fingertips whitening. Typically, patients initially report whitening of the toes of one or two fingers, and only on cooling. Later, there is a whitening that covers the end, middle or all phalanges of the II-V finger of the hands and has a different duration. Restoration of the bloodstream after such an attack is usually accompanied by "throbbing" soreness or paresthesia, sweating and blueing of damaged fingers. Often complaints of functional weakness in the hands and ringing in the ears and faill hearing.

The objective study shows *peripheral autonomic disorders* - sympathicotonia or autonomic dystonia. There is distal hypothermia in the upper limbs (often 22 ° C - 24 ° C to 17 ° C - 18 ° C on the fingers). Some patients experience spontaneous hyperhidrosis of the palms and fingers. Changing the color of the palms of the skin - cyan or pale spotting. According to the process, there is a lace lines, imbibition or puffiness of the palms and fingers. There are also positive symptoms of white spot (Laignel - Lavastine), of Pall, of Bogolepov and others. In clinical symptomatology, angiospastic or angiodystonic manifestations in the hands are of major importance. Observing the so-called dead fingers - *bleeding attacks* (most often on the II, III or IV fingers) occurring at local and general cooling or spontaneous. The duration of bleeding attacks ranges from a few minutes to hours. Peripheral autonomic and vascular disorders develop gradually and show a tendency towards progression. The parameters of local vibrations (especially their frequency), other unfavorable factors of production (eg cold effects) as well as the general health status of the individual are affecting. Concomitant diseases - cardiovascular, endocrine and metabolic disorders, generalized musculoskeletal disorders or collagenosis, neurosis and mental disorders, as well as harmful habits - smoking, systemic alcohol use, etc., facilitate the development of peripheral autonomic and vascular pathology.

Sensorial disorders are a common objective finding and are in the direction of distantly increasing hypoesthesia for pain and touch and reduced discriminative and vibratory sensation in the hands. Some authors describe hypopalestezie as a pathognomonic mark of vibrational disease, but it should not be forgotten that the elevated threshold of vibration sensation is present in diseases damaging the backs of the spinal cord, spinal roots, peripheral nerves, and possibly the lateral columna. Hypopalesthesy is common, for example, in peripheral neuropathies of different etiology - diabetic, alcoholic, in various endocrine-metabolic diseases - acromegaly, hypothyroidism, etc., in carpal tunnel syndrome, radiculopathies, etc.

As the pathological process progress, develops *trophic nail disorders* (striae, koilonychia, nails type "clock glass"), the skin of the palms and fingers (smoothing the skin of the fingers, skin thickening to the sclerodactylium, hyperkeratosis, xanthochromia, rhagades on the palms) or bones (mostly carpal bones and phalanges - focal osteosclerosis, osteoporosis, bone cysts). In some cases there is thickening and deformation of the end phalanges of the fingers with formation of so-called clubbed fingers. In the advanced stages of the disease, hypothrophies of thenar, hypothenar, mm.interossei are also found.

Hearing disorders are the result of the combined effects of local vibrations and over-noise. It occurs in tinnitus and hypacusia, and the reduction in hearing acuity is usually bilateral, symmetrically initially in the high frequency areas - 4000 to 6000 Hz.

Additional clinical signs describe:

- Primary lesion of the peripheral nerves of the upper limbs: e.g. neuropathy of n.medianus in the carpal canal; of n.ulnaris in the cubital duct or cervical radicular syndromes with combined genesis;
- Bone-joint disorders - e.g. deforming arthrosis of the elbow joints; risarthrosis; cervical osteochondrosis and spondylosis;
- Musculo-tendon lesions: humerus-scapular periarthritis, radial and ulnar epicondylitis and tendomyositis

For the development of musculoskeletal damage, static overload, significant dynamic load, overcooling, and increased humidity are of particular importance.

Clinical-functional studies. The development of a diagnosis - VD due to local vibrational effects requires a number of studies to evaluate the functional status of the peripheral blood circulation, the nervous system, the autonomic background and the autonomic reactivity (including the neurovascular endpoint), as well as the differential diagnosis exclusion of other diseases with similar symptoms.

Wide application finds methods of peripheral blood exploration - in routine practice, capillaryscopy and capillarygraphy arteries are used. The changes in the basic color and background of the field of view, the number, shape and position of the capillaries are observed. Depending on the stage of the disease, capillary polymorphism, spastic capillaries, spastic-atonic complexes, or even paretic capillaries with pronounced atony, AV-aneurysms, changes in blood flow and permeability disorders. A modern non-invasive method for quantification of vasomotor reflex activity and microcirculation disturbances is laser Doppler flowmetry. Skin blood flow and microvascular reactivity of the hands were examined by monitoring the perfusion, the concentration of the formed blood elements and their speed in the investigated volume of tissue in a resting state and at thermal heat (44 ° C) with subsequent relative cooling to 32 ° C; a post vein-arteriolar test and a reactive hyperemia test were also used.

Other non-invasive methods for recording peripheral angiopathology (especially for larger blood vessels) are oscillometry and oscillography, rheography, Doppler sonography and plethysmography. Through these, angiodystonic or peripheral vasospasm is defined in the upper limbs, the asymmetry in the left / right hand indicators. A commonly used indicator of angiospastic assessment is the measurement of the fingers systolic pressure in the initial position and after a cold provocation - a sympathetic vasoconstrictor reaction is observed.

Measurement of the skin temperature gives information about circulatory deviations and neuro-vascular reactivity. Thermistor thermometry, liquid crystalline cholesterol thermometry, infrared thermography are applied. Hypothermia (to thermoamputation) in the distal hand compartments and sometimes thermoassimetry (above 1.5 ° C) for the left/right hand is established.

To identify sensory disturbances in the upper limbs, the following are used:

- algesiometry - to determine the surface sensation for pain, and thermoesthesiometry - to determine the sensation of warm and cold. There is an increased threshold for pain sensation and thermohypesthesia in the distal compartments of the upper limbs - fingers, wrists.
- esthesiometry - to determine the threshold of deep sensitivity and two-point discriminatory sensation - reduced sensory function is established.
- palestesiometry - to measure the threshold of vibrational sensation at different frequencies. There is a reduced vibrational sensation.

A number of provocation tests are used to assess autonomic reactivity. Particularly informative is the cold provocation test carried out in various modifications. Prolonged recovery of baseline skin temperature, as well as frequent enhancement of clinical signs following cold provocation, are observed.

Other methods used are:

- X-rays of the cervical spine, upper thoracic aperture, the bone-clavicle space, the hooped joints with the fingers, the elbows or the shoulder joints. There are dystrophic changes in the spine (cervical osteochondrosis, spondylosis), aseptic necrosis of os lunatum or os scaphoideum, deforming arthrosis of the elbow or shoulder joint;

- Alternating Dynamometry - reduced muscle strength in the distal compartments of upper limbs;
- Electrophysiological methods, cardiovascular tests, various biochemical parameters, tonal audiometry and auditory evoked potentials. Electrophysiological methods show the involvement of peripheral nerve structures in the pathological process. Determination of the peripheral nerve conduction velocity of the upper limbs, most commonly n.medianus and n.ulnaris, shows slowing rate and prolonged distal latency time.

The main signs of the disease in stages are:

Stage I - occasional night numbness and pale autonomic and vascular disorders in the hands. In the objective study, a slight degree of hypopalesthesy, dystonic capillaries, a tendency to prolong the time

of recovery of the skin temperature in the cold provocation test.

Stage II - pronounced night paresthesias and morning stiffness of the fingers that pass after movement, insensibility, awkwardness and gripping weakness in the hands, whitening of the end phalanges of II-V finger provoked by cold impact; swollen fingers and palms with cyanotic and cold skin, pronounced hyperhidrosis, hypersthesy for pain of distal-type in upper limbs, moderate degree of hypopalesthesy; frequent spontaneous seizures of white in distant and middle phalanges of II-V finger fingers; (22°C - 24°C to 17°C - 18°C), distally elevated vascular resistance, pronounced capillary spasm and pericapillary edema, significantly prolonged skin repair time in the cold provocation test; moderate hearing abnormalities - ear noise and hearing loss for high frequencies (4000-6000 Hz).

Stage III - permanent and intense paresthesias and diffuse pains, covering almost all hands to the shoulders that do not pass and after movement, pronounced gripping weakness and clumsiness; increase and deepen the angiospastic crises by covering all phalanges of II-V finger and longer duration of seizures (over 10-15 min). The objective finding shows autonomic and vascular disorders in the hands (but with significantly greater from stage II intensity); trophic disorders of the skin of the palms and fingers, nails, clubbed fingers; hypothrophies of thenar, hypothenar; flexion contractions of fingers; various bone-joint dystrophic and muscle-tendon changes; significant hearing loss with reduced hearing acuity in the area and in low frequencies; tendency to generalization of disturbances - in lower extremities, vestibulopathy, endocrine-metabolic dislocation, etc.

4.5.2. VIBRATIONAL DISEASE OF GENERAL VIBRATIONS

The endangered occupations are: tractors, power shovels, bulldozers, heavy truck drivers, cran operators, vibroconcretors, miners who work with telescopes on vibrating platforms, etc.

Symptoms of VD from general vibrations are represented by several syndromes.

Polyneurial syndrome mainly with sensory and autonomic limb symptoms (so-called vegeto-sensory polyneuropathy). It occurs with finger numbness mostly at night, diffuse pain at rest, especially in the distal compartments, frequent sweating, bleeding attacks and stiffness of the fingers. Objectively, there are changes in general sensitivity in polyneuritis type. Above all, the sensation of pain, vibrational sensation, temperature and discriminatory sensation suffer. There are also autonomic disorders in the extremities (paleness, cyanosis, changes in skin temperature, swelling, hyperhydrosis). In advanced forms of VD there are also various trophic disorders. The lower limbs are most often involved, but because the exposure is of mixed vibrations - local and general, the abnormalities are also found later in the upper limbs.

Mononueuritis syndrome. It is characterized by involvement of one peripheral nerve, most commonly of the upper extremities - nn.ulnaris, medianus et radialis. The damage is a result of constant nerve compression in the osteo-fibrosis canal, i.e. tunnel neuropathy develops. In addition to vibrations, etiopathogenetic significance is also the static overtension and forced posture, determining the compression of the nerve. Clinically characterized by numbness, tingling and burning, limiting in the area of nerve propagation, pain and weakness in the hand. Objective signs are: impaction and thickening of the peripheral nerve at palpation over the compression site; sensory disturbances by zone type and positive specific symptoms at the squeeze site (eg, the symptom of Tinel, Phalen, etc. is positive); weakened tendon and superficial reflexes and motor disorders (hypotension, hypotrophy, decreased muscle strength). Characteristic signs are autonomic and vascular abnormalities - hyperhidrosis, distal hypothermia, change in the color of the skin of the palms and fingers.

Radicular syndrome. Secondary (vertebrogenic) lumbosacral and cervical-radicular lesions are most frequently found. They are diagnosed based on the characteristic clinical signs of spinal root damage supported by electrophysiological and X-ray studies.

Bone-joint dystrophy syndrome. In the case of general vibrational impact, the spine structures in the lumbar region are affected. Lumbar osteochondrosis and spondylosis develop with secondary symptomatic radicular manifestations. Certain etiopathogenetic significance also has the accompanying impulses and static overtension in the lumbar region (winding, twisting and prolonged sitting) as well as the adverse microclimate. Clinical symptoms are characterized by gradual development, restraint of movements and blunt pain in the waist area, often irritating down the leg and numbness of his toes. Objectively, there are positive stretch phenomena, soreness in the Valleix paravertebral points according to the level of affected spinal cord structures and root-type sensory defects in the dermatomas. Very rarely, peripheral paresis (hypotension or hypotrophy of damaged muscles according to the affected spinal cord roots), as well as weakened Achilles or knee reflexes.

Cervical osteochondrosis and symptomatic radiculopathies, deforming arthrosis of the elbow joint, omarthrosis or aseptic bone necrosis of hoop joints with corresponding clinical symptoms are also frequently found.

Musculo-tendon syndrome. The complex of adverse factors, especially the overtension and adverse microclimate in combination with vibrations, often result in the occurrence of stenotic tendovaginitis, insertiopathy (most commonly radial epicondylitis), shoulder periarthritis, tendomyosis of the hand extenders, etc. Their clinical symptoms are not different from those of professional musculotendinous syndromes at all.

Cerebral dysregulation syndrome. It is characterized by a non-permanent (transient) general brain symptomatic with functional character, lacking focal neurological lesions. There is a diffuse headache (no nausea and vomiting), unsteady stagger, dizziness, instability, sleep disturbance, decreased concentration of attention and weakening of memory, morning (somato-psychic) asthenia. In some cases, there are various autonomic (neuro-circulatory) disorders: pulse lability, fluctuations and instability of the arterial pressure (most often occurrence of elevated values), orthostatic hypotension, palpitation (sometimes single "functional" extrasystoles), vasomotoric "play" (facial flushing and fainting, limb coldness), hyperhydrosis, etc. There are no deviations in biochemical and haemorheological parameters, from neurosonographic and impedance techniques (ie no evidence of organic brain circulation deficiency). Occasionally, non-invasive methods for brain haemodynamics - reoencephalography (including speed REG), Doppler sonography of cerebrovascular vessels, etc., occasionally result in increased vascular resistance, transient changes in blood flow, and asymmetries in right/left, directed, for circulatory dystonia. There may be slight variations in the study of orthostatic self-regulation of the cerebral blood flow. EEG has no significant diagnostic significance. In some cases, however, alterations in the bioelectric activity of the cerebral cortex and signs of dysfunction in deep brain structures may occur. In the presence of generalized autonomic dystonia, there are abnormalities in the autonomic background and reactivity, usually in the direction of sympathicotonia and increased sympathetic reactivity, as well as a deficiency in the autonomic regulation of functions.

Central otoneurological syndrome (vestibulopathy). Central vestibular disorders are headaches, dizziness, stunning, indefinite staggering, and transient vestibular-autonomous crises. Horizontal nystagmus, instability in the Romberg test and agitated vestibular manifestations may also be found. Autonomic disorders - nausea, vomiting, paleness, sweating, pulse and arterial pressure changes, coupled with vestibular disorders - usually increased vestibular reactivity in the caloric and rotation tests. Often hearing loss is reported, and tonal audiometry shows a reduction in auditory acuity bilaterally and symmetrically for frequencies 4000-6000 Hz. In these cases, it is a hearing-vestibular syndrome.

Hypothalamic syndrome. Such a syndrome, caused only by a general vibrational impact, has rarely occurred in recent years. Separate hypothalamic signs and symptoms can be identified with combined effects of intense general vibrations, significant over-noise and toxic noxae. Neuro-circulatory abnormalities (hypertensive reactions, palpitations, changes in heart rate) and thermoregulatory and sudomotor disorders of sympathetic nature are most commonly observed. Often they have a relapsing nature: sympathetic-adrenal crises or, more rarely, of mixed type - sympathetic/parasympathetic paroxysms. In other cases, isolated endocrine disorders: sudden increase in body weight, appetite disorders (bulimia, rarely anorexia), increased thirst, hyperglycaemic reactions, sexual disorders, ovarian dysfunction and others. Very rare signs of trophic (vitiligo, alopecia, scleroderma, etc.) or neuropsychic (psychastheny, depressive reactions, emotional crises) have been reported. Hypothalamic syndrome can only be diagnosed when the listed symptoms occur at the same time - i.e. the combined symptom complex shows the primary damage to the hypothalamus.

Clinical-functional studies. The main methods include doppler sonography of brain vessels, reoencephalography, EEG, otoneurological examination (including evoked auditory potentials), lumbar/cervical spine radiographs, computed tomography (if necessary), as the methods for examination local vibrations.

The main clinical signs and constellations *according to the stage of VD are*:

Stage I - discrete, barely targeted subjective and objective data of polyneuropathy with autonomic and sensory limb symptoms, intermittent vestibular disorders or separate general autonomic manifestations.

Stage II - pronounced and persistent symptoms including the features of the underlying syndromes (listed above), with specific clinical features dominated by the specific profession. For example, the clinical picture of VD in crane operators is the central dysregulation syndrome, usually in combination with vestibular disorders, most often lacking signs of polyneuropathy in the limbs. For truck drivers, bulldozers and tractors are lumbar osteochondrosis, spondylosis

The main criteria for diagnosing a vibratory disease are:

VD from local vibrations	VD general vibrations
Proof of sufficient daily average (min 4 h) and total (min 5 years) exposure to local vibrations	Proof of sufficient daily average (min 6 h) and total (min 10 years) exposure of the general vibrations
Constellation of peripheral autonomic, sensory, vascular and trophic disorders of the upper limbs and hearing disorders	Proof combination of minimum two syndromes typical of the clinic VD vibrations
Exclusion of other diseases and conditions occurring with symptoms similar to VD - ie accurate differential diagnosis	Accurate differential diagnosis - exclusion of diseases occurring with similar VD syndromes
Course of disease (especially after cessation of exposure to vibration and adequate therapeutic behavior - elimination test)	Dynamics of disease (especially after termination contact with the unfavorable factors of work and correct therapeutic behavior - elimination test)

and symptomatic lumbosacral radiculopathies, followed by similar cervical lesions and other bone-joint dystrophic abnormalities in the limbs. In all these professions, other risk factors are also important.

Stage III - expressed symptoms of autonomic distal polyneuropathy in the lower and upper limbs (with the addition of sensory and trophic disorders); persistent and severe vestibular crises and significant hypacusia - binaural reduction in hearing acuity is moderate to severe, also in areas above and below 4000-6000 Hz; pronounced bone-joint dystrophic lesions of the spine - severe lumbar or cervical osteochondrosis, spondylosis, discopathies with secondary radiculopathy with motor disorders, tense course and frequent recurrences; hypotalamic manifestations - neuro-circulatory disorders, hyperglycaemic crises, sexual disorders, psychosomatic reactions with depressive elements, etc.

The differential diagnosis of VD is most commonly caused by the following diseases:

- Raynaud's disease;
- Secondary Raynaud's syndrome (collagenosis, endocrine and metabolic diseases);
- Compression syndromes in the upper thoracic triangle (thoracic outlet syndrome, hyperabductive or bone-clavicular syndrome, etc.);
- Syringomyelia;
- Lateral amyotrophic sclerosis (LAS);
- Deforming spondylosis with osteochondrosis and symptomatic radiculopathies with non-professional genesis;
- Neuroses (create diagnostic difficulties in some cases due to the full autonomic symptoms and neuro-mental changes);
- Polyneuropathies of different genesis (insuffiencly and dysmetabolic, toxic, collagenous, etc.).

Treatment. Etiological treatment - Removing the patient from the contact with vibrations by temporary or permanent rehabilitation of work without vibrational impact according to the stage of VD.

Medication treatment includes:

Peripheral vasodilators - myotropic drugs with vasodilator effect, other peripheral vasodilators, capillary tonics drugs and others. Most commonly Pentoxifyllinum (Agaprin, Trental, Pentofyllin) is administered at a dose of 300 to 600 mg/day; Naftidroflurilum (Dusodril, Drosunal) at 100-150 mg/day; Cinnarizinum (Cinnarizin, Stugeron) - 75-100 mg/day and others. Non-steroidal anti-inflammatory drugs (NSAIDs) - most commonly used: Indometacinum (indometacin) - 75-100 mg/day; Piroxicamum (Piroxicam, Feldene, Remoxicam) at 20 mg/day; Diclofenac sodium (Feloran, Voltaren, Infla Ban) at 75-100 mg/24 hours.

Combination preparations with pronounced analgesic effect - are usually used in the initial stages or at the start of treatment to obtain a curative effect of NSAIDs. Most commonly, Alcozin, Tempalgin, Neuralgin, Sedalgin Neo and Alnagon are administered at the usual dosage of 2-3 tablets/day.

Sedative agents and soft antidepressants with an effect on the autonomic nervous system (cause vasodilation, improve peripheral blood circulation). Applications are found mostly: Bellergamin 2,3x1 dr. daily; Tofisopamum (Grandaxin) - 50-100 mg/day; Deanxit 1 tabl. morning and afternoon, etc.

Vitamins - include B group of parenteral (muscle) or various combination drugs: Neurobex; Vit.B complex; Milgamma N 3x2 to 3x1 caps/day; Benfogamma 3x1 dr/day. Also apply Vit. E or Geritamin and others.

Physiotherapy. Apply thermal procedures, short-wave diathermy, ultrasound, ionophoresis, common juniper baths. Reflective massage is also useful - hand or with instruments, and healing gymnastics. Balneological sanatorium treatment is also recommended.

Regime and diet. Includes whole nutrition - diet A of protective professional nutrition; adding pectin; avoiding smoking.

Labour expertise. In VD I-II stage 20-30% loss of working capacity is determined, which imposes temporary labour-free work without vibrations, stat-

ic overtension and unfavorable microclimate. VD II stage results in a 50% loss of working capacity - i.e. temporary incapacity for work. Usually, patients with VD II-III and III stage receive a permanent decrease in working capacity - more than 50% for a certain period of time (usually 1 year).

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A. Savov

4.6 PROFESSIONAL DEAFNESS

Occupational hearing loss can be caused by trauma, toxic substances and noise impact.

LOSS OF HEARING by trauma

Ear trauma can be caused by a wide range of injuries. The most common cause of traumatic hearing loss is a blunt stroke on the head that creates a squeezing wave in the skull. Damage to the cochlea after a dull head trauma histologically and audiologically is similar to that of high frequency acoustic trauma.

Vehicle catastrophes are accompanied by blunt injuries and cause about 50% of temporal bone injuries. Relatively rare are penetrating temporal bone injuries - about 10% of cases. Other professional reasons for these traumas include falls, explosions, burns with corrosive chemicals, open fire or welding slag that can penetrate the ear.

Traumas that cause conductive hearing loss may be due to hematoma of the eardrum (provided it is free of fracture of the temporal bone). If this is the only injury, hearing usually recovers in a few weeks. Burns usually heal slowly and often get a chronic infection. A strong explosion with a level above 180 dB can cause a rupture of the membrane tympani. Ruptura are usually healing spontaneously if no secondary infection develops.

Conductive hearing loss lasting more than 3 months after the injury is usually due to residual perforation of the membrane or destruction of the ossicular chain. These lesions are suitable for surgical intervention.

Injuries causing neurosensory hearing loss are trauma to the inner ear. In a labyrinth concussion, transient vertigo, permanent hearing loss and tinnitus usually occur. Therapy is waiting, with vestibular suppressants, giving symptomatic relieve to vertigo. The trauma can also cause a rupture of the round or oval membranes, leading to fluid leakage from the inner ear in the middle ear. Most perilimphatic fistulas heal spontaneously. However, a persistent perilimphatic current is difficult to diagnose and requires surgical treatment (recurrent episodes with vertigo and hearing loss, often associated with increased physical activity).

Temporal bone fractures usually occur along the lines connecting the weak points at the base of the skull. Longitudinal fractures occur more frequently (80% of cases) and are most often the result of a side impact. In them, the structures of the middle ear, rather than the inner ear, are disturbed. Transversal fractures are less common (20% of cases) as result from a strong occipital stroke. They are usually accompanied by severe intracranial injuries. Typical of them is that they pass through the inner ear and lead to complete hearing loss of the neurosensory type and destruction of the labyrinth. They are accompanied by strong vertigo, lasting weeks, even months.

Temporal bone fractures are clinically recognized by the presence of blood and cerebrospinal fluid in the ear canal, or by the presence of blood in the middle ear behind the uninjured m. Tympani. An ear that is freshly traumatized should not be moistened.

OTTOXIC LOSS OF HEARING

Most ototoxins damage femoral cells directly or by demolition of other cochlear homeostatic mechanisms. In most cases, ototoxic hearing loss occurs with the use of drugs such as aminoglycoside antibiotics (eg. gentamicin), diuretics (furocemide), antineoplastic agents (cisplatin) and salicylates (aspirin). In the noisy work environment, workers treated with ototoxic drugs are at increased risk for greater hearing loss.

Heavy metals - arsenic, cobalt, lithium, lead, mercury and thorium, have proven ototoxic potential. Other chemicals with ototoxic action are cyanides, benzene, aniline dyes, iodine, fluorophenone, dimethyl sulfoxide, dinitrophenol, propylene glycol, methylmercury, potassium bromate, carbon disulfide, carbon monoxide, carbon tetrachloride, styrene and toluene.

HEARING LOSS IN NOISE EFFECTS

Professional hearing loss due to noise effects is a slow-running process over a long period of time (several years) as a result of constant or intermittent strong noise effects. Occupational acoustic trauma is a sudden change in hearing as a result of one-time, loud, impulsive, blast-like noise.

Under the conditions of contemporary production, professional hearing disorders have become the leading occupational diseases in Bulgaria. They are the second most frequent cause of overtension diseases of the locomotory system and the peripheral nervous system. Their relative share in the structure of occupational diseases varies from 9 to 15%.

The greatest percentage of hearing impairment occurs in the age groups 50-59 (64%) and 40-49 years (25%). They are more common in men than women.

There is an increase with an increase in length of service.

The highest is the relative share of hearing impairments in the ferrous ore mining and steel industry, transport, textiles and knitwear, machine building and metalworking, chemical industry.

Accordingly, professions such as: metallurgists, miners, blacksmiths, boilers, firemans, riveters, turners, millingers, motorcycle testers, avionics technicians, motorcyclists, weavers, agricultural mechanics, compressors and many others are threatened by the impact of noise.

Pathogenesis. *Hearing loss due to noise impacts is the result of trauma to the sensor epithelium of the cochlea, damaging the stereocilias of the ciliary cells.*

In the acute acoustic trauma of the labyrinth, the acoustic action of the powerful sound is coupled with that of the shock wave. Internal ear elements are shuffled, haemorrhages, trophic disorders, and later degenerative dystrophy of the Corti organ, ganglion cells and nerve fibers appear.

In chronic acute trauma, the underlying pathogenetic mechanism is limited to changes in the inner ear; endolimphatic hydropae in the context of neurovegetative vascular dystonia, metabolic and structural changes in the Corti organ, utriculus, sacculus and semi-circular canals. Various degenerative and atrophic processes in these formations were later observed.

Sensory and mental abnormalities resulted in a significant increase in plasma cortisol levels, an increase in minute volume and heart rate, and an increase in blood pressure. Noise helps increase the level of adrenaline and noradrenaline in peripheral blood. Parallel to this, there is a spasm of the cochlea's blood vessels, especially the spiral vessels of the basilar membrane and the stria vasculiaris.

The effects of noise alter the serum-biochemical equilibrium, the function of the glands with internal secretion and the immune-biological reactivity.

Under the influence of an over-threshold energy sound stimulus, and depending on the duration of the sound pressure, the level of glycogen in the sensory cell initially increases and then decreases. Similar biases are also found in the activity of enzymes associated with biological oxidation.

The risk of hearing disturbance is related to the duration and intensity of exposure, but also to the genetic susceptibility to noise trauma. Prolonged exposure (over 8 hours a day) of sounds with an intensity above 85 dB/A causes hearing damage. Permanent exposure to noxious noise levels has the greatest effect in cochlear zones that perceive high frequency tones. Hearing loss is prone and strong for frequencies around 4000 Hz, with downward spanning to speech frequencies (500-2500 Hz) occurring after longer intense exposure.

Clinic. Patients complain of gradual hearing loss. The most common complaint is a difficulty in perceiving speech, especially in underlying background noise. Because patients with professional hearing loss have mostly high frequency abnormalities, they hear vowels better than consonants. This leads to distortion of speech sounds. Background noise, which is usually low-frequency, masks better the preserved part of the hearing spectrum and makes it even more difficult to understand the speech. Loss of hearing is often accompanied by tinnitus - most often patients describe it as a high-frequency tone (ringing), but sometimes it is lower, such as buzzing, blowing, hissing, or even without sound (clicking or snaping). This feeling can be intermittent or permanent and usually deepens. Due to the tinnitus, patients complain about the inability to sleep or concentrate in a quiet room.

In the tuning fork study, the patient has an air conductivity better than bone conduction - neurosensory hearing loss. When using serial tuning forks from 512 to 4096 Hz, there is often a noticeable reduction in hearing at higher frequencies. Audiometry usually shows two-sided, mainly high-frequency neurosensory hearing loss with a maximum drop in clear tone thresholds of about 4000 Hz. Since the most important thresholds for understanding human speech are between 500 and 3000 Hz, there is no significant reduction in understanding and perception of speech. The degree of speech discrimination is normal in the initial stages of hearing loss but may be aggravated by progressive hearing loss.

Permanent hearing changes occur most often between the 4th and 10th years of work experience. They are symmetrical or nearly symmetrical, but in some cases small differences between the two ears are possible if the working posture exposes one ear to a stronger exposure.

The harmful influence of noise on the central and autonomic nervous system and other organs and systems is manifested at a noise intensity significantly lower than that causing persistent auditory changes. Functional CNS disturbances precede the occurrence of persistent auditory changes and are particularly common in younger (newcomers) working in noisy environments. At the beginning of work experience, complaints of tinnitus, headache, restless sleep, nervousness, unsteady vertigo are common. They are light, reversible, with the character of hearing fatigue. More pronounced at the end of the shift and the working week. Subjective noise in the ears has the most frequent high frequency characteristic. In the beginning he is interrupted, and then becomes permanent. Its intensity ranges from 5 to 10 dB with a frequency of about 4000 Hz. In the lat-

er stages the noise becomes strong (in the order of 25-30 dB), disturbs the patient's sleep and generates neurasthenic complaints.

In the course of the noise deafness a certain phase is observed. At the outset, the common complaints are: fatigue, headache, depression and tinnitus (recruitment phase). It lasts most often from one week to several weeks or months. This is the phase of the best tolerance. Its duration depends on the noise intensity. This phase can last for years and then follow the crash phase (critical period). Later on, there is a delay in the course of hearing impairment (fed up phase).

There are three forms of occupational hearing impairment:

- Isolated defeat of the inner ear that progresses with work experience;
- Combined damage to the inner and middle ear and
- Chronic catarrh of the middle ear with symptoms of inner ear irritation and vertigo attacks of the type of Menier's disease.

Three levels of professional auditory neuritis have been adopted:

1st degree - the reduction for 4000 Hz reaches 35dB and for low frequencies - up to 10dB;

2nd degree - respectively up to 45 dB and up to 15 dB and

3rd degree - hearing loss for 4000 Hz is up to 50 dB and for low frequencies up to 20 dB.

The otoscopic picture is usually normal. Vascular hyperemia of the tympanic membrane is often observed. Long-time workers are found to be darks and swallowing the eardrum, and the incidence of chronic otitis and dry perforations of the eardrum is greater than that of those who do not work at the noise.

The non-specific (extra-aural) effect of noise on the body is characterized by a variety of changes in other organs and systems. This applies especially to the central and autonomic nervous system, the digestive system, the internal secretory glands, the cardiovascular system.

Noise increases pulse rate and breathing, changes in blood pressure, increased intracranial pressure, changes in gastric peristalsis and gastric secretion, etc.

Objective studies indicate: most commonly tremor of the hands and eyelids, decreased corneal, tendon and abdominal reflexes, nystagmoid, hypoesthesia, decreased vibrational sensation, reduced expression and duration of demographic reactions and inversions, opthalmocardiac and orthoclinostatic reflexes.

The effects of noise are associated with mood transitions and some retention of intellectual processes. Functional neurodynamic studies indicate a significant prolongation of the latency time in the visual-motor response. In the EEG study, a characteristic low-energy bioelectric activity with dominant slow waves and high inertness reaches full noreactivity in light irritation.

All this leads to a new nosological unit - a **noise disease** (Andreeva-Galanina E. C., 1972), whose autonomy is still debatable. Clinical manifestations are reduced to several syndromes:

- vegetative-vascular;
- astheno-vegetative;
- hypothalamic and
- discirculatory encephalopathy syndrome.

Depending on the exposure and the nature of the noise, the individual syndromes may be stronger or less pronounced, and in some cases some of them may be missing at all.

The main characteristics of occupational hearing loss due to noise effects are summarized as follows:

- the sensory cells of the inner ear are always affected.
- almost always the damage is bilateral. The audiometric indicators are similar bilaterally.
- almost never damage does not cause profound hearing loss. Normally, the low-frequency range is about 40 dB and the high frequency is about 75 dB.
- once the noise has interrupted, further progress in hearing loss is negligible.
- previous damage due to noise effects does not cause greater sensitivity to future noise effects. If the threshold of hearing perception increases, the rate of further decrease in hearing sensitivity decreases.
- the earliest damage to the inner ear reflects hearing loss at 3000, 4000 and 6000 Hz (usually highest at 4000 Hz). Hearing impairment beyond 3000-6000 Hz requires longer exposure times.
- in case of constant exposure to noise, hearing loss at 3000, 4000 and 6000 Hz usually reaches maximum after about 10-15 years.
- in workers from "noisy" productions and occupations, vestibulometric investigation shows spontaneous vestibular symptomatology (statokinetic abnormalities and nystagmus). Damages of the peripheral vestibular apparatus predominate, due to vessel vasospasm in the labyrinth vascular system and changes in the receptors of hearing and vestibular analyzers.

Diagnosis. Of great importance is a detailed history - a clarification of the professional factor (including past professions); previous illnesses, pretreatment with ototoxic and vestibulartoxic medications,

etc.; ototoxic, acumetric (tuning fork) and audiologic data as well as vestibulometry.

The magnitude and nature of occupational hearing impairments and labour expertise are solved by means of different methods of hearing research.

The usual methods are easy to apply, but with little informality (hearing with tuning forks, of whispering and conversational speech, etc.).

The special methods of hearing investigation are more reliable. Audiometry is the main methodology for determining the degree, nature and level of hearing impairment.

The methods of tonal (threshold and supra-threshold) audiometry - subjective audiometry, objective and speech audiometry are used.

At tonal audiometry differently modeled clear tones such as acoustic signals (125 to 8000 Hz) are used and the minimum threshold of hearing about air and bone conduction for each ear.

Tonal over-threshold audiometry has a higher topo-diagnostic significance.The character of the perceptual acoustic signals and especially the deformations in the sound intensity, known as the "recruitment phenomenon" (accelerated increase of loudness), are determined. The most common use is made of the overthreshold samples (tests) of: Fowler - measurement of loudness alignment; Langengbeg - measuring the effect of deafening; Karhard and Kitz - measuring adaptation and fatigue. Overthreshold tests are important for early diagnosis because they are very positive early on.

Speech audiometry is an important method of hearing research. Use a specially selected, phonetically balanced speech material from words, numbers, sentences. The method is an over-threshold test of importance for the labour expertize, allowing the combined assessment of the function of the cochlear departments of the hearing analyzer and of the cortex sections of speech and hearing.

Objective audiometry is based on the registration of various motor and vegetative reactions or bioelectric potentials arising in response to sound irritation.Impedance methods provide information about the prereceptor, receptor, and root - trunck part of the auditory analyzer.

Hearing-induced potentials (ERA) that occur at different hearing levels are recorded in clinical practice by advanced computer electrophysiological and electroacoustic equipment.

To assess the overall noise impact, neurological examination, EEG, rheogram, neuro-vegetic, and some other specialized studies - gastric functions, cardiovascular indicators are needed.

Differential diagnosis. The following disorders should be ruled out: old deafness (presbycusis); hereditary neurosensory hearing loss; metabolic disorders - diabetes, impaired thyroid function, kidney disease, autoimmune disease; cochlear otosclerosis (family burden); sudden neurosensory hearing loss; Vascular disorders (sudden loss of hearing loss due to blood vessel blockage or migraine); diseases of the central nervous system - small brain tumors and pons Varolii; demyelinating diseases (e.g., multiple sclerosis); Meniere's disease and its variants;

Prognosis. The development of occupational hearing impairments depends on the intensity and frequency of noise, the nature of the toxic noxae, the duration of the daily impact, the working conditions and the individual characteristics of the organism. Initially, the disabilities are functional and reversible, but later they become lasting and almost irreversible.

Treatment. Treatment is causal and symptomatic. Efforts are directed at regulating the excitating processes in the cerebral cortex. Sedative and psycholeptic agents (Diazepam, Dimenhydrinat, Elenium, Lexotan, Deanxit, Nootropil, etc.) are used. To inhibit the increased tone of the parasympathetic vegetative nervous system, 0.1% Atropine, cortex sedative (Scopolamine 0.5%) is administered. Good effect is Galantamin (Nivalin)/0.25%, as well as strengthening agents (iron, arsenic, calcium, etc.), stimulating nervous activity and metabolism, vitamins (A, B, C, E), Atrifos, Cocarboxilase. They are combined with intravenous administration of glucose (40%, 40 cm^3) and vitamin C (5 cm^3), Mannitol, etc., achieving a dehydrating effect and boosting oxidative processes. At the same time, physiotherapy procedures (Kalii jodati electrophoresis, Ephedrine, massage collar, etc.), appropriate hygienic regime, gymnastics, tourism, etc. are recommended.

The treatment of permanent damage is difficult. There are no specific means of therapeutic impact, but only means that act protectively and symptomatically. Widely used vasodilators (Sadamin, Cavinton, Stugeron, Trental, Radecol, Dibazol, Divascan, Euphyllin, etc.). Good results are obtained from the administration of Ac. Glutaminici, Nivalin, Strichnin, Papaverin, Group B vitamins (B_1, B_6, B_{12}, B_{15}) and others.

Labour expertise. An important condition for the accuracy of the expert's conclusion in professional hearing neuritis is the precise determination of hearing loss. Binaural hearing loss in percent is determined by a special formula (Fletcher). Up to 10% binar hearing loss for the 500, 1000, 2000, and 4000 Hz frequencies is defined as the initial stage of occupational auditory neuritis; from 10 to 20% loss - as a mild degree; from 20 to 30% - moderate grade; over

30% - severe.

D. Dimov (1986) suggests that auditory loss is defined as the mean value (in dB) of the hearing threshold values of the four main frequencies - 500, 1000, 2000 and 4000 Hz (the field of social hearing). There are 4 degrees of hearing loss:

1st grade (up to 30 dB), the hearing is socially adequate. Workers are able to work for noisy production, but subject to dynamic monitoring. Audiometry is done once a year. Individual prophylaxis is required. The professional nature of the disease is not accepted.

Grade II is a severe hearing (hearing loss is 30-60 dB for a better listening ear):

a) At 30-45 dB hearing loss requires labour rehabilitation without deterioration of the qualification, dynamic monitoring and treatment by an otorhinolaryngologist. The duration of the rehabilitation is individual, depending on the results of the treatment;

b) At hearing loss 45-60 dB has difficult social contact, limited working capacity for the profession in excess noise or the 3rd group of disability is defined.

c) Practical deafness (loss of 60-90 dB). These workers are receiving hearing aid or given a third group of disability for life. At over 90 dB hearing loss can be worked in noisy production because such workers practically have no hearing and do not need prophylaxis.

V. E. Ostapkovich and N.I. Ponomarjova (1979) also offer similar assessment criteria for hearing function **(Table 1)**.

In case of severe hearing, practical deafness and deafness, you should always send a quick notice. F. 58 to NII and the case to be discussed in the regional LAMC after a poll.

Prevention. A major preventive task is the removal or reduction of noise through sanitary-technical prophylaxis - replacement of noisy machines with less noise; technical maintenance of machines; noise protection screens and isolation with sound-absorbing materials, etc.

Personal preventive measures - internal and external antiphones, helmophones are also important.

Outdoor recreation helps restore hearing sensitivity. Relaxation rooms are organized and people with progressive hearing loss are brought to silent work. Proper medical-professional selection - chronic catarrhalis, adhesions and purulent diseases of the ears are contraindicated for working under noise conditions. Contra-indications for work are also neuroses, hypertonic disease and other cortico-visceral diseases.

Periodic medical examinations are once a year, with the participation of otorhinolaryngologist and neurologist, and conducting an audiometric study. It is recommended that diet A of the professional preventive diet.

Medicinal ototoxins should be prescribed in minimal doses sufficient for therapeutic efficacy. The simultaneous administration of various ototoxic agents should be avoided when this is possible in order to avoid a synergistic effect.

Tab. 1. Criteria for assessing the condition of the hearing function (V.V. Ostapkovic and N.I. Ponomarjova)

Degree of hearing loss / **Rating hearing**	**Tone threshold audiometry**			**Speech audiometry**	
	Hearing loss in sound frequencies 500, 1,000 and 2,000 Hz/ arithmetical mean in dB	**Hearing loss at 4000 Hz and a range of possible deviations in dB**	**50% intelligibility threshold in dB**	**100% intelligibility threshold in dB**	**Adopting whisper in m**
1	2	3	4	5	6
I. Practically normal hearing	1-5	50 ± 20	30-35	40-45	6 ± 1
II. Initial signs of cochlear neuritis	6-10	50 ± 20	30-40	45-50	5 ± 1
III. Cochlear neuritis with mild hearing loss	11-12	60 ± 20	35-45	50-60	4 ± 1
IV. Cochlear neuritis with moderate hearing loss	21-30	65 ± 20	45-55	60-75	2 ± 1
V. Cochlear neuritis with a significant degree of hearing loss	31-45	70 ± 20	55-70	75-90	1 ± 0.5

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A. Savov

4.7 OCCUPATIONAL DISABILITY OF THE VESTIBULAR ANALYZER

Occupational disability of the vestibular analyzer occurs when there is a harsh (sudden) or long overnorm impact of: production noise; vibrations; toxic substances; speed; acceleration; atmospheric pressure (oto-borotraumatism); ionizing, electro-magnetic, opto-kinetic effects, weightlessness and others.

Threatened professions. *The main risky productions and professions with more frequent diseases of the vestibular apparatus are: the textile industry, construction, mining, machine building, logging and the woodworking industry (work with compressors, vibrators, electric motors, etc.), agriculture, transport (aviation workers, truck drivers), caisson workers, aqua-lungers, athletes, toxic industries, work in space.*

The vestibular analyzer is first formed in embryogenesis and, in its complexity, close links to the central nervous system and functional properties, differs sharply from all the cranial brain nerves: it is one of the main systems that stabilize the position of the center of gravity in the body and make a permanent Control of the locomotor and oculomotor apparatus, which provides the complex equilibrium function.

The peripheral part of the vestibular analyzer is located in the pyramidal part of the temporal bone. Its receptors are represented by the three semi-circular channels located in three mutually perpendicular planes and the otolithic apparatus made of the sacule and the utricle. The entire system is performed by the endolymph, and in its motion in the result of angular (recurrent) or rectilinear accelerations, potentials are generated in the neurosensory cells of the cupulo-macular apparatus.

Pathogenesis. *In the event of adverse professional factors, the vestibular apparatus produces numerous variations in all body functions (somatic and vegetative) due to extensive inter-central nervous connections with the central nervous system.*

In the vestibular department of the labyrinth, more significant changes are recorded in the area of the antechamber, especially in the sacule, and in the semi-circular canals - in the sagittal and horizontal channels.

Ultrastructural changes in the receptor cells of the vestibular analyzer were found - cytoplasmic vacuolization, mild changes in preganglionic myelin nerve fibers, nuclear lesions.

The vestibular analyzer is intimately linked to the autonomic nervous system (parasympathetic part), and through the Dither spinal path, the posterior longitudinal bundle, the reticular formation and other conduction systems with the other cranial-brain nerves, the anterior horns of the spinal cord, etc. At significant overthreshold irritants suppression of the muscles (limbs, bulk), digestive system tone, changes in serum-biochemical equilibrium, enzyme activity, internal secretion glands, cardiovascular activity, etc., occur.

Vibration *is an adequate irritant to the vestibular analyzer. In production conditions, they are usually accompanied by intense noise with a different spectrum, of linear or angular accelerations (at general vibrations).*

Certain responses of ganglion cells and neuroepithelial of of vestibular apparatus, of degenerative - atrophic character have been identified - hyperemia, edema, pycnose, ganglion cell decentralisation of the otolithic apparatus.

General vibrations transmit over the endolimph high-level, supra-threshold accelerations that, as a result of prolonged exposure, cause degenerative-atrophic changes. The pathogenetic basis is the disruption of the vegetative-vascular reflexes at different integration levels in the central nervous system. General neurovegetative dystonia causes such transient phenomena as hypotension - hypertension, bradycardia - tachycardia. At the same time, it also causes microcirculation disorders in a number of parenchymal organs, as well as frequent transient disturbances of the labyrinthial circulation.

With sudden fluctuations in ***atmospheric pressure****, various changes occur in the inner ear. They are encountered in the case of caisson workers, underwater sports, researchers at great sea depths, on the seashelf, working in aviation, in space. Detonation trauma is associated with explosive work.*

At caisson disease suffers the sacule. Compression and decompression are adequate irritants of the sacule receptor apparatus. Under these conditions, hydrops occur in the labyrinth, and decompression can produce a gas embolism from the releasing nitrogen.

Clinic. The vestibular apparatus suffers low from a **noise** compared to the hearing analyzer. With loud noise and unadapted more workers, there are phenomena of dizziness, disturbing balance, difficulty in orientation, especially towards the end of the

working day. These complaints disappear after a rest or after an adaptation of the vestibular apparatus. However, with sustained and repetitive noise effects, permanent damage may occur.

Clinical studies show the individual specificity of the respective to nystagmatic responses. With long-term impact, spontaneous nystagmus was observed in 20% of cases. The excitability of the vestibular analyzer usually decreases, which is manifested by depression of nystagmus reaction (experimental post-nystagmus and especially that of caloric stimulation).

More typical clinical manifestations are expressed in reduced duration, frequency and speed of the slow phase of caloric and post rotation nystagmus; dropping the quick component; asymmetry; occurrence of dissociated reactions; changes in vestibular-sensory and vestibulo-vegetative responses; disharmony of reflexes.

In **vibrational illness**, vertigo complains are in three symptom complexes:

- with a sense of disturbed balance at a different head position, without objective vestibular symptoms;
- with instability of the body support;
- with a typical labyrinthic dizziness.

Objective clinical signs are:

- spontaneous nystagmus, positional nystagmus, hyporeflexia (chronic vibrational trauma);
- dissociation between caloric and rotation test (a sign of high diencephalic damage to vestibular centers and paths);
- a phenomenon of floating eyeball movements (after vestibular irritation, in the middle and end of experimental nystagmus);
- disturbed nystagmus rhythm (central sign of vestibular disorder);
- falling of the rapid component of the nystagmus (a central sign of damage to the vestibular pathways and centers) - an early sign of central nervous system damage.

When **combined with noise and vibrations**, central vestibular disorder syndrome develops. It runs parallel to the cerebral form of a vibratory disease, and in some cases precedes it. Often, manifestations of the otolithic system and combination of symptoms of positional disorders are observed.

There are two clinical forms of **barotrauma**: acute and chronic. The acute barotrauma is manifested by a Meniere's-like syndrome (ear noise, reduced hearing, dizziness, nystagmus, harmonic-like statokinetic deviations, nausea, vomiting, sweating, pallor, collapse). In the chronic form, the hyperreflective responses to the caloric provocation of the labyrinth, predominate.

Detonation trauma occurs in rapid, sudden compression (blast). Typical changes occur in semi-circular canals and utricle (bleeding, atrophy and degeneration of the neurosensory epithelium). There are three forms of vestibular disorders: peripheral, central and mixed.

The clinical characteristics are:

- lack of caloric and rotation stimulation reactions;
- dissociation between a rotation and a caloric test (a sign of retrolabyrinthic injury);
- positional syndrome (commotio syndrome)
- vegetative reactions;
- vestibulo-vasomotor reactions - diencephalo-vegetative symptoms, decompensation of the vestibular-vasomotor mechanisms of the cerebrum.

The vestibular analyzer is harmed in **industrial intoxications** with lead, arsenic, mercury, silver, phosphorus, manganese, carbon monoxide, carbon dioxide, petrol vapour, benzene, aniline, paint, acetone, alcohol, nicotine. There are two mechanisms of impact. In acute poisoning, toxic substances act directly on the structures of the inner ear - cell degeneration or at blood vessels - hyperaemia, haemorrhage, atherosclerotic changes, etc., which aggravates the trophic of the specialized epithelium.

Disorders are bilateral and at different levels (receptor, ganglia, lateral-vestibular nucleus, cerebellar vestibular structures, vestibular connections, reticular midbrain formation, etc.).

The clinical picture includes: dizziness, spontaneous nystagmus, bilateral hyporeflexion of the labyrinth, or a complete loss of vestibular function (lead), looking paresis (mercury), stem and nuclear damages (carbon monoxide), damage to the otolithic apparatus (aniline).

In acute poisoning, the vestibular crisis appears in the background of general intoxication, and in chronic it is slow, not very pronounced, with transient vertigo, instability, nystagmus.

Methods of research. After taking into account the history of vestibular vertigo, disturbed balance, vestibulo-vegetative and vestibular-somatic reactions, a full otoneurological study is performed which includes:

- ORL review;
- audiometric investigation - tone threshold, over-threshold, speech audiometry, objective audiometry, impedancemetry;
- study of the most specific symptom - nystagmus (spontaneous and provoked): with Bartels and Francels glasses, and study of experimental nystagmus after vestibular irritations: opto-

kinetic (opto-kinetic stimulator), thermal (cold and warm caloric test), Fitzgerald-Holpick test rotating, (nystagmography), accelerating (with accurate dose of positiveand negative angular accelerations and constant velocities) tests;

- statokinetics (posturography) - Barany's index test, Romberg's test, Babinski-Vale's blind walk, Unterberger's test;
- precise stabilizing methods.
- cranio-corpography (according to Klausen);
- static and dynamic stability (static ataxia is investigated with a special platform and sensors for 1 min - 30 s with open and 30 s with eyes closed, dynamic disturbances are investigated with Unterbergerograma - in angular degrees);
- study of perceptions of space, time and movement (A. Kehayov);
- study of olfactory and taste function.

Labour expertise. At the compensation stage there is no spontaneous and latent vestibular symptomatology. Workers are able to work, but attention is needed in certain professions (crane drivers, excavator drivers, truck drivers, etc.).

In the subcompensation stage latent nystagmus, positional nystagmus, statokinetic and vegetative disorders are established. Workers are temporarily incapacitated, to receive compensation.

A spontaneous nystagmus is found at the decompensation stage. Workers are temporarily incapacitated for any type of work. Appropriate therapy and surveillance at the hospital are carried out.

With simultaneous impairment of hearing and vestibular function, the expertise is made complex.

If these injuries develop in young workers and for a relatively short period of time, concomitant occupational diseases of the nervous system, cochlear neuritis with a significant degree of hearing loss and frequent vestibular crises, further work on noise effects is contraindicated. There is a need for retraining and rational employment (with or without a disability group for occupational disease).

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5 OCCUPATIONAL DUST DISEASES OF THE RESPIRATORY SYSTEM

E. Petrova

5.1 PNEUMOCONIOSIS. SILICOSIS

5.1.1. PNEUMOCONIOSIS. GENERAL PART.

The development of biology, molecular medicine, genetics, electronics and the introduction of new imaging techniques have boosted the pathogenetic mechanisms of pneumoconiosis. There has been a growing number of studies aimed at elucidating epidemiology, etiopathogenesis, immunology, diagnosis and genetic predisposition for pneumoconiosis. New diagnostic methods and drugs are continually being tested. More important in the clinical aspect are the pneumoconiosis, manifested by the growth of fibrous tissue in the lungs. Along with the fibrousogenic effect of the powders, their other effects - irritant, carcinogenic and immunoallergic - are being studied more and more widely.

Pneumoconiosis *is the accumulation of dust in the lungs and the response of the lung tissue to it. Pneumoconiosis is caused by different dusts.*

Pneumoconiosis is the most significant occupational pulmonary disease in the world and in Bulgaria. They lead to serious health, social and economic consequences for patients, employers and the state. The total number of collagenous pneumoconiosis patients in Bulgaria in 1995 is about 5776, and about 2000 at the year 2000. The risk of pneumoconiosis (controls) registered in the silicosis divisions in the country ranges from 11,200 to 23,000. Recently, there has been a significant share of hidden morbidity and morbidity from pneumoconioses in connection with regulatory, economic and organizational reasons or insufficient knowledge of this subject by general practitioners and specialists in the areas of internal medicine. A large proportion of pneumoconiosis arise or progressive after termination of employment with powder. The deposited aggressive dust in the lungs of former workers has long preserved its fibrousogenic properties and is the basis for the so-called "late forms" of pneumoconiosis (silicosis, asbestosis, mixed pneumoconioses of mineral and metallic powders, anthracosis). Pneumoconiosis from working in small and medium-sized private enterprises with old or modern technologies, with new dust factors in the working environment is expected in the future. Become more frequent reports for unknown now variants of pneumoconiosis (pneumoconiosis in dental technicians, pulmonary dust damages from asbestos substitutes (glass wool, ceramic fibers), pneumoconioses from synthetic abrasive materials (corundum, carborundum), pneumoconiosis and other injuries (broncho-obstructive conditions, lung carcinoma), of metallic powders, pneumoconiosis from solid metals, tonic pneumoconiosis, and others.

Pneumoconiosis classifications:

From a pathoanatomic point of view, pneumoconioses are:

- *Non-collagenous* - accumulation of dust without the expansion of collagen (fibrous) tissue in the lungs (baritosis, stannosis, pure siderosis);
- *Collagenous*-growth of collagen tissue (silicosis, silicatoses, coal pneumoconiosis, mixed pneumoconiosis - from mineral and metallic powders, etc.);
- *Intermediate with respect to collagen formation in the lungs are pneumoconioses* of respirable aluminum powder, different variants of coal dust and others.

According to the etiological principle, the pneumoconioses are subdivided into **(Table 1)**:

- *silicosis* (free crystalline silica - quartz, micro- and cryptocrystalline modifications of silica, high temperature modifications of crystalline or amorphous silicon dioxide (crystobalite and tridymite),
- *coal pneumoconiosis* - anthracosis (from highly carbonated coal with different free crystalline silicon dioxide content),
- *silicatoses* (from different silicon dioxide linked

variants, asbestosis - from asbestos fibers, kaolinosis - from kaoline, talcosis - from talc)

- *pneumoconioses of mixed powders* (mixed mineral powders - talcasbestosis, pneumoconiosis in stove builders, pneumoconiosis in stove repairers, etc.)
- *pneumoconioses of metallic powders* (*siderosis* - pure, iron powder (rare), *aluminosis*- breathable aluminum powder, *pneumoconiosis in electric welders* - iron powder, low silicon and irritant gases in welds, *corundum pneumoconiosis* - from dust composed of about 95% Al_2O_3, connecting clay minerals, minimal amounts of free silica; *pneumoconioses of solid metals* - tungsten, cobalt, vanadium, nickel, titanium, tantalum, chromium and others and *berylliosis* - a type of hypersensitive pneumonitis);
- *other pneumoconiosis* (apatite, barite, volcanic minerals, glass, ceramic fibers, graphite pneumoconiosis, tonic pneumoconiosis, pneumoconiosis in dental technicians). They are rarely occured or insufficiently studied.

Rangening Classification of Pneumoconioses - *ILO Internatinal Classification of Radiographs of Pneumoconioses, Geneva-1980 (ILO'80).* This classification provides a qualitative and quantitative description of the X-ray signs of pneumoconiosis recorded in anterior-frontal conventional pulmonary X-ray, technically performed according to the requirements of the ILO'80 guideline. The qualitative description of X-ray findings were made with certain alphabetic characters. The quantitative evaluation of the small irregular (stripped) and small oval (spotted) shadows per unit area in the lungs is performed using a 12-point density scale (expanded, precision version) or a 3-degree density scale (brief version, expressed by categories 1-3).

Clinical roentgenomorphological classification of pneumoconiosis. It is co-authored by the Johannesburg classification of pneumoconiosis and was adopted at the First Congress of Pneumoconiosis in 1930. It was updated in 1956 and was adopted in Bulgaria. According to its criteria, nodosal pneumoconioses are subdivided into 3 stages, roughly taking into account the way the disease progresses (rapidly, slowly), the mode of manifestation (acute, chronic, early, late form) and the characteristic of the respiratory function (compensated, decompensated). Two preclinical conditions (Controla "Z" and Controla "L"), taken from the first version of the 1956 ILO Classification of Pneumoconioses, are also reported in our country.

5.1.2. SILICOSIS

Studies on ancient Egyptian mummies give rise to the assumption that silicosis has existed since antiquity. The first descriptions of the disease are from Agricola (1556), Paracelsus, Ramacini, Straton, Visconti, Lomonosov, Collins and others. The term "silicosis" was created by Visconti and is borrowed from the Latin translation of the word silicon - "silex". An important moment in the development of the medi-

Tab. 1. Etiological classification of pneumoconiosis

Collagen pneumoconiosis				**Non-collagenous pneumoconiosis**	
Mineral pneumoconiosis	***Metal pneumoconiosis***	***Mixed pneumoconiosis***	***Other pneumoconiosis***		
Silicosis - of free crystalline silica (quartz) *Silicatoses - from the linked silica (silicates)* *Asbestosis* *Caolinosis* *Talcosis and others.*	*Aluminosis - respirable particulate aluminium or Al_2O_3* *Berylliosis - from beryllium aerosols* *and other*	*Mineral powders:* *Talcasbestosis* *Silicotalcosis* *Pneumoconiosis in stove ripairers* *Pneumoconiosis in stove builders and oth.* *Pneumoconiosis of metal-containing dusts (Pneumoconiosis in electric welders)* *Pneumoconiosis of corundum Pneumoconiosis in dental technicians and other*	*Anthracosis - from high carbonization coal with different quartz content* *Apatitosis - from apatite powder (rarely diagnosed)*	*Clean siderosis of pure iron respirable dust (relatively rare)*	*Baritosis - pure powder barite (rare)*

cal field, studying the pneumoconiosis, was the Lyon Congress in 1929 and the Johannesburg Conference on Pneumoconiosis (1930), which adopted the first clinico-radiomorphologic classification underlying pneumoconiosis diagnostics.

Silicosis is a fibrous lung disease caused by inhalation and deposition of free crystalline silica (quartz).

Silicosis is one of the most common occupational pulmonary diseases in the world, especially in the countries of Asia, Africa, Eastern Europe and South America. According to WHO data, the incidence of tin mines in Bolivia in 1987 is 7.6%, the mines in Korea - 3.5%, the gold mines in Egypt - 43.3%, the lead mines in India - 30.4%, mines and metallurgy in China - 3.5% and 5.5%, the ceramic industry in Brazil - 3.7%, all risky industries in Japan - 17.4%, the stone and ceramics industries in Thailand - 21% and 9 , 3%, gold, copper and chrome mines in Zimbabwe - 20%. Silicosis is less important for Western Europe and the United States. Data from the silicosis divisions in Bulgaria for a five-year interval (1985-2000 period) show that the total number of silicosis + silicotuberculosis patients ranges from 4566 to 5472 and the endangered ones from 11238 to 24011 **(Table 2)**[1].

Gradually, the cases of silicosis from underground work on dry or dry and water drilling in mines for extraction of ores, anthracite and black coal in Bulgaria are exhausted. New cases of silicosis and silicotuberculosis in the future are expected from underground and overground extraction and processing of non-ore minerals, production and use of refractory, facing, grinding materials, mosaics, in glass, ceramic, faience, metallurgy, machine building, use of sand streamers, production and processing of monuments and others. Depending on the specifics of the powder and quartz content, different forms of silicosis are expected. Increasingly, there will be cases of reticular and reticulo-micronodous silicosis from underground mines developed after a long latency period and prolonged exposure to low concentrations of free crystalline silica.

Tab. 2. Total number of silicosis, silico tuberculosis and controls (endangered of silicosis) in Bulgaria - 1985 - 2000 year

Diseases	Year				Average for the period
	1985	1990	1995	2000	
Total silicosis	4447	4195	3904	3828	4159
Total silicotuberculosis	1025	946	858	738	921
Total silicosis + silicotuberculosis	5472	5141	4762	4566	5081
Total controls	11238	10650	24011	20000	18684

*An **etiological factor** for the emergence of silicosis is the free crystalline silica (quartz). There are three basic forms of free silica: 1. crystalline, 2. micro (crypto-) crystalline and 3. amorphous. Quartz is a crystalline form of silicon structured by tetrahedra. The chains of Si-O-Si are at the base of the crystal structure. Fibrogenic effects also have high temperature modifications of crystalline and amorphous silica produced by heating from 870 ° to 1470 ° C and above 1470 ° C (crystobalite and tridimide). Practically important for the development of pulmonary fibrosis are fine (respirable) silicon particles with a diameter of 0.5 μm to 5 μm.*

Risk production processes for silicosis are:

- *mining, uranium mining and coal mining in Bulgaria (extraction of anthracite and black coal);*
- *transport and hydro-construction and extraction of non-ore minerals;*
- *use of natural abrasive, and grinding materials;*
- *sandblasting for various industrial purposes;*
- *production and use of refractory materials in metallurgy (iron-melting, agglomeration, ore preparation);*
- *ferro-alloy production, foundries;*
- *glass industry, manufacture of porcelain and stoneware products and others.*

*An important role **in the pathogenesis of silicosis** play the following factors:*

- *size, shape, specific surface of quartz particles, tendency to agglomeration;*
- *duration of exposure to dust;*
- *dust concentration in the air of the working environment;*
- *amount of powder and latency in the lungs;*
- *- individual anatomical features of the lower respiratory tract of the exposed;*
- *possibilities of lymphatic transport and ability of the mucociliary escalator to self-purify;*
- *spent and accompanying pulmonary inflammatory processes;*
- *individual features of the immune system;*
- *genetic predisposition.*

Modern perceptions of the pathogenesis of silicosis emphasize the cellular and immune mechanisms of quartz action.

Key points are:

[1] The undisputed hidden morbidity and illness of silicosis and silicotuberculosis in recent years is due to the incomplete registration of former workers leaving home, relocation by place of residence, financial, organizational and other reasons of subjective and objective nature.

- *clustering of alveolar, interstitial macrophages, blood monocytes;*
- *polymorphonuclear cells, lymphocytes, few eosinophils and basophils around the dust particles in the alveoli;*
- *elimination of a minimal part of them by absorption by macrophages and purification by means of the mucociliary escalator;*
- *eliminating part of the respiratory quartz along the lymphatic pathway and transporting to regional lymph nodes;*
- *transporting the respirable quartz particles from the macrophages to the interstitial of the lungs where the pathogenetic mechanism is realized at the cellular level;*
- *phagocytosis of the dust particles absorbed by the macrophages, covering a portion of them with a protein coat in the first 2-6 hours;*
- *subsequent toxic injury and death of phagocytes;*
- *lysing the protein coat around the quartz particles from the released cell enzymes at the death of the macrophages and taking the particles of new generations of macrophages;*
- *removal of superoxide radicals (predominantly O^{2-}, H_2O_2), interleukin-1 (IL-1), tumor necrosis factor alpha (TNF-α), leukotrienes from macrophages;*
- *activation of T-lymphocytes, unlocking a cell-mediated immune process, and cytokine release of T-lymphocytes - interleukin-2 (IL-2) and γ-interferon;*
- *re-activation of macrophages by IL-2*
- *changes in humoral immunity: blocking T-suppressor function of lymphocytes, unlocking an autoimmune process;*
- *the supposed adjuvant effect of quartz leading to an autoimmune process;*
- *fibroblast proliferation and collagen formation;*
- *possible decreased ratio of helper to suppressor (Th/Ts) in peripheral blood and pulmonary tissue, resulting in decreased cell-mediated immunity: increased B-lymphocyte activity, autoantibodies production;*
- *reduced activity of natural killer cells (NK): reduced response to mitogens, chronic immunosuppression, impaired regulatory mechanisms resulting in pneumoconiosis development;*
- *predicted genetic predisposition to silicosis - genes close to HLA-B locus;*
- *involvement of other genetic mechanisms and role of programmed cell death (apoptosis);*
- *impact of frequent inflammatory lesions of accompanying or complicating silicosis chronic bronchitis or other non-specific or specific pulmonary inflammatory processes leading to secondary collagen formation, and facilitating the faster progression of the silicotic fibrosis process.*

Pathomorphology of silicosis. *The basic morphological substrate of silicosis is the silicone node with a diameter of 0.3 to 1.5 mm, located initially in the interstitial peribronchialy, paraseptale and sub-pleural.*

The silicotic nodes gradually increase and show a tendency for conglomeration. In the central zone of the silicone node, collagen fibers are formed with a predominantly concentric disposition exhibiting a tendency to the hyalinization. Rarely, collagen fibers are unevenly positioned. At the periphery of the nodules are found macrophages and less - plasmocytes and lymphocytes located in close proximity to the central zone. The large conglomerate nodes are composed of a hyaline-free collagen without a distinctly concentric disposition. The presence of centrally located focal necrosis is most commonly associated with granulomatous inflammation, tuberculosis or atypical mycobacterial infection. In acute silicosis, the morphological substrate is silicoproteinosis with the presence of macrophages in the lesion and hyperplasia of adjacent pneumocytes of the second type.

There is a variant of low-silica mixed pneumoconiosis and a predominant expansion of interstitial fibrosis with single nodous elements, taken as mixed silicosis, diffuse-sclerotic silicosis or mixed mineral pneumoconiosis.

There are three basic classifications of silicosis - X-ray, clinical-radiomorphological and classification by passing.

Radiographic classification of silicosis - ILO International Classification of Radiographs of Pneumoconioses, Geneva, 1980 (ILO'80).

This classification is used for qualitative and quantitative description of X-ray findings in pneumoconiosis, including for silicosis. With ILO'80, three main groups of x-ray findings are described:

small irregular (strial) shadows of pulmonary parenchyma:

- s - small irregular shadows up to 1.5 mm wide. **(Figure 1)**;
- t - small irregular shadows with a width of 1.5 to 3 mm;
- u - small irregular shadows with a width of 3-10 mm;
- *small oval (micronodous, nodous) shadows of pulmonary parenchyma;*
- p - small oval shadows with a diameter of up to 1.5 mm. **(Figure 2)**;
- q - small oval shadows with a diameter of 1.5 mm. To 3 mm. **(Figure 3)**;
- r - small oval shadows with a diameter of 3-10 mm. **(Figure 4)**.

There are also intermediate finds for small oval and small uneven shadows (eg p/q, s/t, etc.) as well

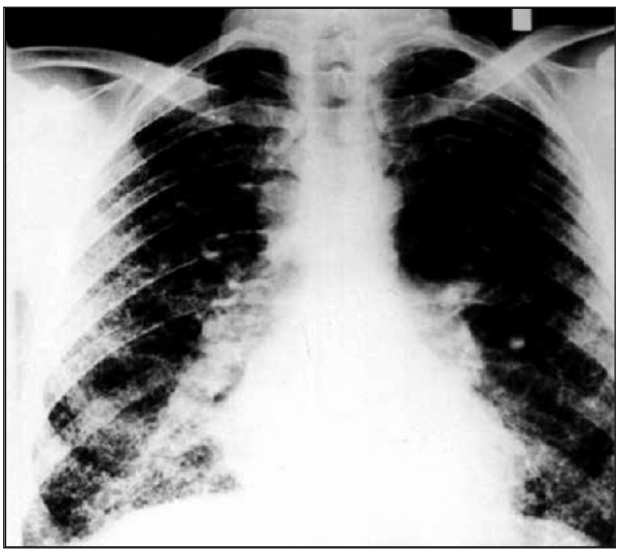

Fig. 1. s - Small uneven shadows of category 3/3 (control "L") - reticular silicosis

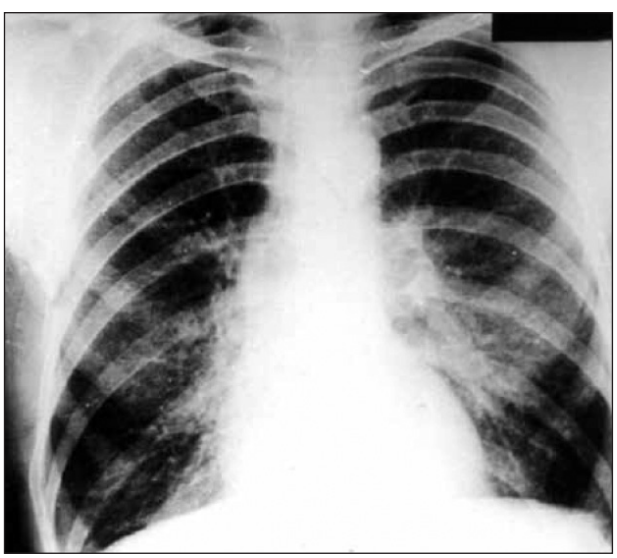

Fig. 2. p - small oval shadows of category 3/3 in medium pulmonary fields - silicosis I stage

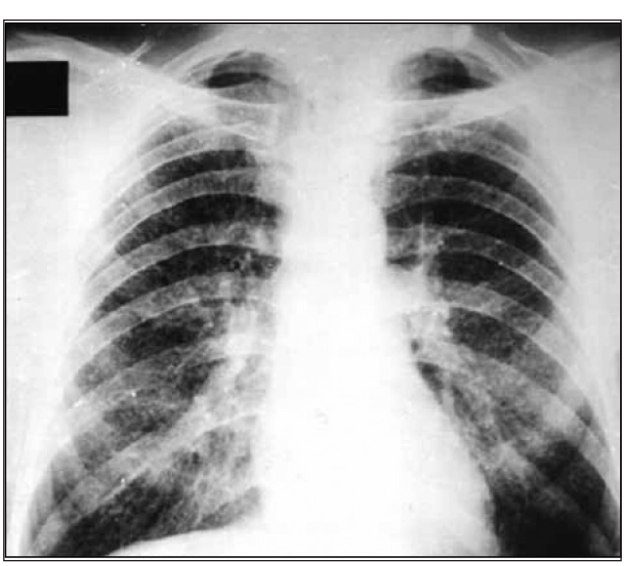

Fig. 3. q - small oval shadows of category 3/3 in all pulmonary fields - silicosis II stage q type

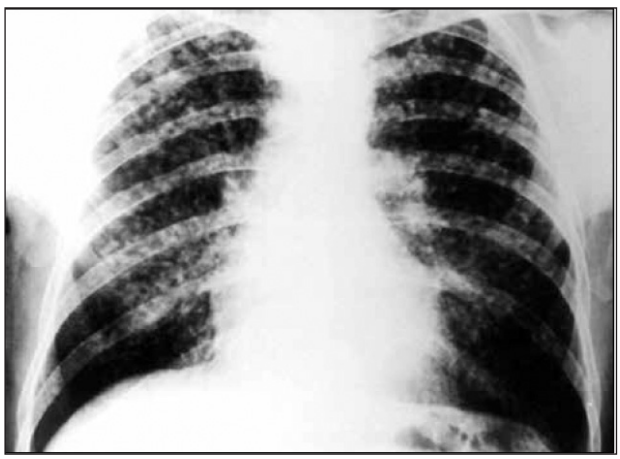

Fig. 4. r - small oval shadows of category 3/3 in all pulmonary fields - silicosis II stage r type

as mixed finds (p/s, p/t, r/s, t/q).

Progressive massive fibrosis:

- A-type progressive massive fibrosis (PMF) - solitary or confluent small oval shadows, occupying an area of 1 cm to 5 cm in diameter **(Figure 5)**;
- B-type PMF, with a diameter of 5 cm to a diameter equal to the upper 1/3 of the right pulmonary field **(Figure 6)**;
- C-type PMF with diameter larger than that of B-type PMF **(Figure 7)**.

Pleural thickenings (plaques) are labeled with PL and pleural calcifies with PLc. Their spread is reflected by numbers (1 to 1/4 of the lateral contour of the chest wall, 2 - 1/4 to 1/2 and 3 - over 1/2 to the chest wall), and their thickness - by letters (a - up to 5 mm, b - 5 mm to 10 mm, c - over 10 mm).

The X-ray density (number of small oval and unequable shadows per unit area) is described using a three-step density scale, reflected in the categories

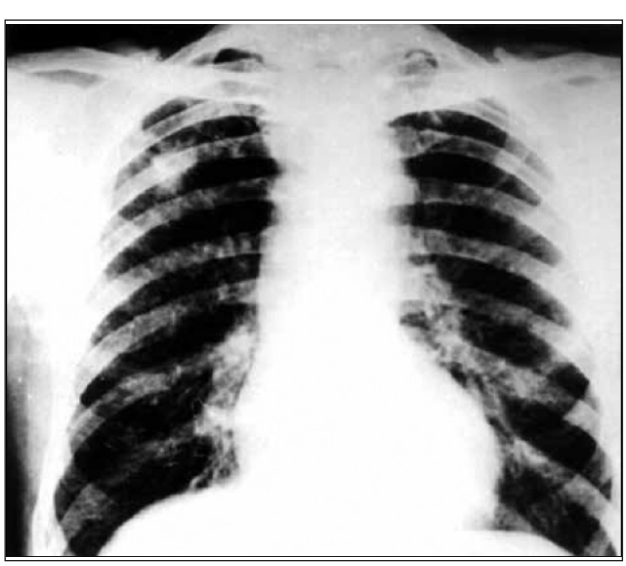

Fig. 5. Progressive massive fibrosis (PMF) type A - silicosis III of type A

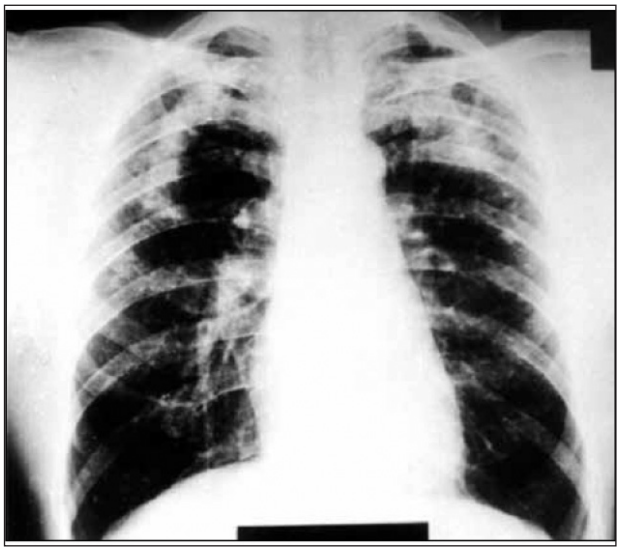

Fig. 6. Progressive massive fibrosis (PMF) of type B - silicosis III stage of type B

- category 1 (Cat 1), cat 2 (Cat 2) cat. 3 (CAT 3). There is also a 12-degree density scale used for precision studies (Cat 0 / -, Cat 0/0, Cat 0/1, Cat 1/0, Cat.1 / 1, Cat.1 / 2, Cat 2/1, Cat 2/2, Cat 2/3, Cat 3/2, Cat 3/3 and Cat 3+).

Existing additional X-ray findings from pulmonary parenchyma and pleura are described with certain alphanumeric characters. The most common are:

- ax - clustering of small oval shadows;
- em - pulmonary emphysema;
- bu - emphysema bulls;
- ca - lung cancer;
- cn - small shadows with calcium density in the pulmonary parenchyma;
- co - changes in the configuration of the cardiac shadow;
- cp - pulmonary heart;
- cv - cavity shadow (cavern);
- es - increased hilus shadows with circumferential or full calcifies-type "egg shells" **(Figure 7)**;
- fr - fracture of a rib;



Fig. 7. Progressive massive fibrosis (PMF) of C type - silicosis III stage C type

- ho - "honey-comb" lungs;
- ih - nosharp outline of the heart contour;
- kl - Kerley's keys;
- od - other pulmonary X-ray findings;
- pi - inter-lobar or mediastinal splicing;
- px - pneumothorax;
- rl - Kaplan's syndrome;
- tba - signs of active pulmonary tuberculosis and
- tbn - inactive pulmonary tuberculosis and others.

Clinical-radiomorphological classification.

According to this classification, the pneumoconioses are divided into three manifest clinical stages:

- *1st stage silicosis* **(Figure 2)** - mainly p-spadows from Cat. 2, located bilaterally in middle and partial in lower pulmonary fields to the parietal or unequable shadows with superexposed p-shadows Cat. 1/1 and enlarged hilus shadow;
- *Phase II silicosis* **(Figure 3, Figure 4)** - bilaterally scattered across all lung fields to circumferentially symmetrical small oval shadows predominantly of q- and/ or r-type with medium to more pronounced x-ray density category;
- *III silicosis stage* **(Figure 5, Figure 6, Figure 7)** - presence of bilateral symmetrical shadows type "PMF" - A, B or C.

There are two preclinical states:

Control "Z" - powder exposing former or current workers without visible X-ray changes in pulmonary parenchyma and

Control "L" - dust exposed individuals with minimal uneven shadows in middle and lower pulmonary fields and enlarged hilus shadows.

Classification of silicosis by passing:

Acute silicosis. It occurs in massive quartz exposition (sandblasting, milling, manual preparation and use of quartz for various purposes, guniting, massive dust impact on dry drilling in underground mines). It runs progressively and quickly.

Chronic silicosis. It occurs with prolonged dust exposure to relatively low quartz powders or a short-term massive quartz exposure in the past. This is the most common form of silicosis in recent decades. *It is divided into:*

- *Reticulo-micronodose* form of silicosis (*silicosis I stage* in clinico-radiomorphological classification);
- *Nodose form* (*silicosis II stage* - according to clinico-radiomorphological classification);
- *Silicosis type of progressive massive fibrosis* (*silicosis III stage* - clinicall-radiomorphological classification);
- *Diffusion-sclerotic forms of silicosis* (diffuse

growth of fibrous tissue in the lungs without clear formation of nodes and tumorous masses, with a vague distinction from the surrounding parenchyma);

- *Late forms of silicosis.* Occur after 5 years or more of dust-free period - from quenching quartz exposure. They still prevail among the newly discovered cases of silicosis but have significantly decreased due to the depletion of the reservoir of former underground miners who have worked in dry or dry and water drilling of blast holes;
- *Mixed mineral pneumoconioses of quartz-containing powders are equivalent to the term "mixed silicosis"* of some Bulgarian authors. They are caused by powders with a low quartz content (up to 2%) and the addition of other minerals to the dust from the working environment;
- *Kaplan syndrome (a combination of silicosis, predominantly anthracosylicosis with rheumatoid arthritis). In some cases, arthritis may be pre-existing.* Radiologically, it is manifested with large node shadows (with a diameter of more than 1 cm) scattered across all the lung fields bilaterally. It resembles collagenosis. Progressing fast.

Clinical picture of uncomplicated and complicated silicosis

Clinical subjective pulmonary symptoms and physical pulmonary phenomena in silicosis are unspecific and are determined by:

- type of disease (acute silicosis, chronic silicosis and chronic late forms of silicosis) by the degree of pulmonary fibrosis;
- from existing complications with specific and non-specific chronic pulmonary inflammatory diseases;
- the age of the patients (more pronounced in late forms of silicosis);
- the smoking and alcohol use factors;
- the incidence of inflammatory lesions of the accompanying silicosis of chronic bronchitis.

* More severe clinical symptoms, more pronounced physical pulmonary phenomena and functional respiratory disturbances are detected in the so-called "diffuso-sclerotic" forms of silicosis.

Acute silicosis

Subjectively acute silicosis is manifested by pronounced dyspnea, cough, rapid progressive respiratory and heart failure. It has been lethal for two or three years. Clinical pulmonary symptoms predominate X-rays manifestation. Radiographically base located, X-ray shadows, passing briefly into cloud infiltrates. Higher hilus shadows are found.

Chronic Silicosis

In uncomplicated conventional reticulo-micronodose, micronodose and nodous forms of silicosis, the symptoms are:

- easy fatigue;
- periodic tightness in the chest;
- lack of air.

In complicated conventional chronic silicosis (PMF) with/without accompanying chronic pulmonary or heart disease:

- shortness of breath;
- easy fatigue;
- breasts in the chest;
- coughing
- sputa

In late forms of chronic silicosis (occurring 5 years and more after the discountinuation of dust exposure), the more pronounced subjective pulmonary symptoms are due to longer smoking and long-term use of alcohol:

- cough;
- sputa;
- breasts in the chest;
- easy fatigue;
- shortness of breath.

Haemopthoae is a rare symptom of pure silicosis. It is usually seen with silicosis complicated by bronchiectasis, silicotuberculosis, accompanying lung carcinoma, thromboembolism.

Silicotuberculosis. The subjective clinical symptoms in late and conventional forms of silicotuberculosis resemble those of silicosis *(but in an active tuberculosis process, additional intoxication events are identified)*:

- weakness;
- anorexia;
- weight loss;
- very easy fatigue;
- subfebrility;
- intermittent elevation of temperature;
- sweating;
- flu-like symptoms;
- upper dyspeptic, less common - lower dyspeptic syndrome and other symptoms typical of the tuberculosis process.

Physical phenomena in chronic silicosis

Uncomplexed conventional chronic silicosis at the initial stage lacks physical pulmonary abnormalities or they are scarce.

Late forms of silicosis are usually accompanied by a number of complications or accompanying illnesses due to the older age of the patients (over 50 years). Physical pulmonary findings are richer and more diverse than conventional chronic silicosis. *There are:*

- emphysematously altered chest;

- Sonorous percussion tone;
- Weak vesicular breathing
- Exacerbate vesicular breathing with accompanying crepitations.
- **Dynamics of the silicotic process in chronic silicosis.** In late forms of silicosis, the transition from silicosis to silicotuberculosis is shorter and more pronounced.

Complications of chronic silicosis

- chronic bronchitis;
- pulmonary emphysema;
- complication with tuberculosis (silicotuberculosis);
- complications with atypical mycobacterial infections;
- complications with mycotic infections (most commonly aspergillosis in immunocompromised individuals);
- restricted pulmonary fibrotic areas outside of silicotic fibrosis;
- secondary bronchiectasis;
- bullous emphysema;
- frequent pneumonic processes;
- pleural adhesions;
- pleurisy (in active silicotuberculosis);
- rarely - partial pneumothorax due to existing pleural adhesions;
- chronic respiratory failure; - chronic cor pulmonale;
- right heart failure.

Most common combinations of chronic silicosis with other pulmonary and extra-lung diseases:

- with lung carcinoma (predominantly the causal relationship between quartz dust exposure and silicosis, and lung carcinoma);
- with rheumatoid arthritis - Kaplan's syndrome;
- with scleroderma;
- with systemic lupus erythematosus and dermatomyositis.

Functional characteristics of silicosis

Most authors consider silicosis to have minimal functional deviations in its discovery. With the onset of progressive massive fibrosis, progressive deepening of respiratory failure occurs in a long delay in the occurrence of hypoxic manifestations and in the absence of hypercapnia (the occurrence of hypercapnia is recorded in terminal stages of the disease).

Chronic late forms of silicosis show a predominantly restrictive, mixed, and rarely - obstructive respiratory deficiency at the time of detection, and *conventional chronic silicosis* is manifested by a restrictive and obstructive ventilator defect. Ventilation changes in late forms of silicosis are also associated with a number of additional factors such as age, smoking, passing or accompanying silicosis, chronic non-specific and specific inflammatory processes.

Late chronic silicosis shows more frequent manifestations of respiratory failure at the time of disease detection.

In case of conventional chronic silicosis, beginning at the time of dust exposure, there is a faster progression of respiratory failure over time. This is due to younger age at a more pronounced reactivity of the body and a higher quartz depot in the lungs.

Our studies of changes in blood pressure (PaO_2, $PaC0_2$, O_2Sat, BE) indicate that they are not particularly informative for assessing respiratory function in uncomplicated silicosis.

The study of the ventilation index for micronodose, nodose silicosis and progressive massive fibrosis (silicosis III stage) showed statistically significant changes in PEF and FEF 50% in reticular silicosis and in the group of micronodose, nodous silicosis and progressive massive fibrosis (PMF) - more expressed in the second group. There was a decrease in FEV_1, PEF and FEF50%, age-dependent and decrease in FEV_1 as the duration of powder exposure increased.

Our studies of pulmonary perfusion changes in various forms of silicosis (performed with ^{99M}Te) showed an insignificant predominance of pulmonary perfusion disorders in cases of progressive massive fibrosis (100%) and significant perfusion disturbances in accompanying silicosis chronic bronchitis, pulmonary emphysema, pneumosclerosis, CPI-I degree CPI-I-II dg. and CPI-II degree.



Fig. 8. HRCT of the lungs in the silicosis I stage, reticulum-micronodose form - small-spotted shadows (p-type) of category 2/2, located dorsally

It is considered that pulmonary high-resolution computer tomography (HRCT) to allow for a more objective assessment of pulmonary X-ray changes, as well as better correlated with functional ventilator changes than conventional pulmonary X-ray **(Figure 8)**.

Diagnostic methods for silicosis

- *Powder-hygienic assessment of air in the working environment* (sufficiently long-term work exposure to excess free crystalline silicon dioxide).
- *Subjective pulmonary symptoms,* even minimal, and physical pulmonary phenomena in quartz exposed workers are a signal to search for silicosis.

Silicosis-suspective cases are directed to diagnosis for specialized clinics.

- *The X-ray method is essential in the diagnosis of silicosis.* Conventional pulmonary radiography with special technical requirements and reading according to the criteria of the ILO International Classification of Radiographs of Pneumoconioses, 1980, is performed. The quality of two-sided anterior-front tomograms (9, 11 cm) for the visualization of uneven, oval shadows or shadows of the "progressive massive fibrosis" type.
- *Additional imaging methods are:*

1) computer tomography (CT) and

2) high resolution computer tomography (HRCT) of the lungs. They are recommended for difficult diagnosis cases.

- *Functional breath test system for silicosis:*

1) breath ventilation, capacity and flow ventilation-vital capacity (VC), forced vital capacity (FVC), Tifno index (VC/FEV_1), extreme expiratory debit (PEF), debits ($FEF_{25\%}$, $FEF_{50\%}$, $FEF_{75\%}$ of the FVC curve) in order to specify the type and degree of ventilator disturbances

2) diffusion capacity study (DLco) and its membrane component (Dm) to determine the amount of disturbed diffusion of gases through the alveolar-capillary membrane;

3) airway resistance study (Raw); and

4) blood gases and alkaline - acid samples in arterial blood (Ph, PaO_2, $PaCO_2$, BE, O_2Sat).

- *Other paraclinical diagnostic methods are:*

BSR; haematological; biochemical indicators; bacterial studies of sputum, broncho-alveolar lavage (BAL), pus from a focal point taken by a transthoracic biopsy, hemocultures (for aerobic / anaerobic bacterial flora, Mycobacterium tuberculosae, atypical mycobacteria, some mycotic bacteria, etc.) - via direct bacterioscopy, seeding culture, by Bactec technique; skin test for atypical mycobacteria; adenosine deaminase (ADA) for a silicotuberculosis susceptibility; immunological tests (Manthoux 5 TE, ELISA - for testing of antibodies to tuberculous mycobacteria in serum, BAL and others, blast transformation test, PCR - test - testing of anti-DNA antibodies against tuberculosis mycobacteria, serological tests for mycosis, HIV and other microorganisms).

- *Additional instrumental techniques:*

Echocardiography, thoracoscopy, thoracotomy, fibrobronoscopy with BAL, trans-bronchial pulmonary biopsy (TBPB), transthoracal aspiration biopsy (TTAB), pleural biopsy (PB) and cytological, histological, mineralogical, bacteriological and, other studies of materials from invasive techniques.

- *Study of cardiovascular system* for complications with cor pulmonale, as well as accompanying other cardiovascular diseases.

X-ray diagnostic criteria for silicosis:

- *Control Z* - powder exposed individuals without significant X-ray traces
- *Control L* - powder exposed persons with s-, t-, s/t - uneven shadows of Cat. 1/0 to Cat. 3/3 +, sometimes - small oval p-type shadows from Cat. 0/1 and Cat. 1/0, often - enlarged hilus shadows (hi).

1. Silicosis I stage - reticulo-micronodose form - uneven shades of s-, t-or s/t - type of Cat. 2/1 to Cat. 3/3 +, with superposed p-small oval shadows of Cat. 1/1 and upwards, disposed symmetrically to partial in medial, partial - lower or partial - sub - clavicles, enlarged, deformed, non - uniform hilus shadows (hi);

2. Silicosis I Stage micronodose form - small oval shadows of p-type Cat. 2/2 and upward in middle, partial - lower or partial subclavicles areas, located on both sides, enlarged and deformed hilus shadows (hi), peripheral emphysema, sometimes light basal or subcluvicle emphysema (em).

3. Silicosis II stage, nodose form - q-, r- or a combination of both sometimes - with the presence of p-shadows scattered across all lung fields at the usual X-ray oval shadow density - Cat. 2/1 and upwards, presence of less pronounced irregularities (s-, t- shadows), enlarged and deformed hilus (hi), periphocal emphysema, basal and sometimes supreme sub-clavicle emphysema (em) and/or bullous emphysema (bu).

4. Silicosis III Stage - type progressive massive fibrosis (PMF) or tumorous silicosis - bilateral symmetrical shadows of A, B, C - type, pulmonary emphysema (em), distorted and extracted mediastinal shadow and trachea (di), often adhesions in bone-diaphragmatic (PL), presence of bullous emphysema (bu), transverse diaphragmatic domes, presence of other additional X-rays, depending on the working dust exposure or other pulmonary lesions added, often enlarged right heart contours (cp). *In case of doubt about the addition of another process, in case of differentiation with another pulmonary disease, in reticulomic ronodose silicosis, for early diagnosis of borderline p 0/1, p1/0 forms of silicosis, the HRCT of the lungs is a much more informative imaging than the conventional pulmonary X-ray.*

Silicotuberculosis is characterized by the follow-

ing basic X-ray traits:

1) unilateral (asymmetric) changes;

2) added soft-foci infiltrative shadows;

3) binding of the infiltrative shadowing with the upper pole of the hilus to the affected side;

4) retraction of the affected thoratic half;

5) cavity shadows located rearward;

6) the cavity shadow is often without drainage bronchus;

7) pleural adhesions with blocking of the diaphragm on the affected side;

8) tentative drawing of the diaphragm domes on the affected side;

9) metatuberculosis calcium outbreaks from a tuberculosis process;

10) interlace of X-ray signs in a mixed silicotuberculous process (silicosis III stage with fibrous-cavernosal and caseous tuberculosis with / without cavity shadow).

Differential diagnosis of silicosis. Good information on dust production is required.

Differential diagnosis of clinical disease is performed with all inflammatory and non-inflammatory lung diseases.

Differential diagnosis in x-ray is roughly divided into:

Differential diagnosis with all diseases occurring with small oval spots:

1. Other pneumoconiosis variants (coal pneumoconiosis, kaolinose, talcose, mixed pneumoconiosis, pneumoconiosis of metallic and metallic powders and other

2. hypersensitivity pneumonitis

3. pPulmonitis

4. pulmonary tuberculosis

5. subacute and chronic haematogenous disseminated pulmonary tuberculosis

6. sarcoidosis

7. disseminated septic pneumonia

8. disseminated pulmonary Metastases

9. idiopathic pulmonary fibrosis - Hamman-Rich syndrome

10. pulmonary proteinase

11. primary and secondary pulmonary haemosiderosis

12. histocytosis

13. all disseminated forms of lung mycosis etc.

Differential X-ray diagnosis in progressive massive Fibrosis (PMF from A, B and C-type):

1) other pneumoconioses with PMF (PMF coal coagulation, kaolinose, other mixed mineral pneumoconiosis);

2) peripheral lung carcinoma and other benign pulmonary neoplasms (adenoma, neurinoma, hamartoma);

3) round croup pneumonia;

4) pulmonary gangrene;

5) round viral pneumonia;

6) tuberculosis;

7) silicotuberculome;

8) lung echinococcus;

9) gum-infiltrative (pulmonary form of syphilis III stage);

10) lung abscess (filled);

11) pulmonary aspergilloma filling a residual pulmonary cavity;

12) filled-in solitary pulmonary cyst;

13) rheumatoid lung and others.

Medication treatment for silicosis includes:

Protective treatment of silicosis to delay or prevent the onset of silicosis. Prophylaxis has been done with:

1) Aluminum citrate inhalation, performing BAL in healthy dust exposed workers (to eliminate quartz, inflammatory cells and cytokines leading to collagenation).

Primary etiopathogenesis (antifibrous) treatment (to stop the progression of the disease). The following preparations were used:

In Asia (mostly China), hundreds of herbal and herbal preparations based on the alkaloid bis-benzylisoquinoline (tetrandrin, cepharan, varbamine) have been tested, the effect of which is associated with stopping fibrosis progression but not fully proved.

According to experiments in Europe (1997), bis-benzylisoquinoline has a cytotoxic and immunosuppressive effect on macrophages, so its use is not recommended.

In the early 1970s, anti-malarial preparations based on quinol-piperazine and quinol-piperazine-hydroxyphosphate showed inhibition of fibroblasts-treatment used in China and in our country. No data are currently available on the use of these preparations for this purpose.

The most used in the world until recently was the preparation polyvinyl-2-pyridine-nitrogen oxide (in parenteral and inhaled form - Kexipink). Its effect is associated with blockage of superoxide groups on the surface of quartz particles. Less known are the Chinese preparation Xifukain and the Russian bacterial anti-inflammatory agent Terilidin.

The antioxidants *(Prednisolone, Glutathione, N-acetylcysteine, Vitamin E, combination of Prednisolone, Vitamin E, Heparin; Vitamin E and Zinc combination)* have been tested in humans and in animals, and have shown neutralizing effects on elevated lipid peroxidation, characterized with the overproduction of superoxide radicals, playing an important role in the formation of collagen and the pulmonary

fibrosis. *Bulgarian zinc aspartate Oxyrich* stops the progression of pulmonary fibrosis in primary forms of silicosis due to antioxidant and immunomodulating effect.

Recent research has focused on direct mechanisms of pulmonary fibrosis formation by:

1) substances directed against growth hormones;

2) cytokines; and

3) antioxidants.

Secondary pathogenetic treatment focuses on already formed pathogenetic mechanisms of advanced silicosis and its complications.

Scheme for the treatment of an inflammatory lesion of accompanying silicosis chronic bronchitis:

1) Antibacterial treatment of the added bacterial infection.

2) Bronchodilator treatment, including xanthine derivatives, beta-agonists, atropine derivatives, combined preparations.

3) Corticosteroid treatment - in case of impossibility of disobstruction with bronchodilators.

4) Mucolytics - N-Acetylcysteine, Ambroxol, inhalations (heat-wet with NaCl, $NaHCO_3$).

5) General stranght givings and vitamins.

Treatment at exacerbated chronic respiratory deficiency in silicosis-sheme:

1) All previous stages of bronchitis treatment

2) Oxygen therapy (PaO_2 below 50 mm Hg)

3) Heparin prophylactically - 10,000 IU s.c. In the abdominal wall (for the protection of pulmonary thromboembolism)

4) Antiaggregants - Aspirin, Antistenocardin

5) In high hematocrit (over 60%) - blood letting and saline substitution

6) In cardiac decompensation - eventual administration of cardiotonics and diuretics

7) Of COPD and arterial hypertension, to exclude any use of β-blockers from the treatment regimen of silicotic patients with bronchial obstruction.

The peculiarities of the silicotuberculosis process require precision with regard to the duration of treatment (at least 8 months), the tuberculostatic combinations (better - quadruple) and their mode of administration (daily). There is a real danger of the presence of resistant tuberculous mycobacteria and ineffective de-bacillation.

The non-medication treatment includes:

1) general strengthening motional and diet regimen

2) use of food rich in vitamins and antioxidants

3) prevention of inflammatory impulses through sanatorial- and climatotherapy

4) physical therapy

5) respiratory rehabilitation.

Prognosis. For the progression of the disease is bad. Mortality is higher than that of the general population.

Prophylaxis of silicosis includes:

Primary sanitary-technical prophylaxis consisting of: effective ventilation, wet boring, personal protective equipment, dustless technologies.

Medical prophylaxis includes:

1) Preliminary and periodic medical examinations of exposed workers at a certain interval.

2) Do not allow people with chronic pulmonary and cardiovascular diseases to work with dust. Diet B is recommended - professional protective nutrition.

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T. Burilkov, Kr. Dinkova

5.2 SILICOTUBERCULOSIS

Tuberculosis is the most common complication of silicosis. In 1970, over 20% of patients with pneumoconiosis in Bulgaria were with silicotuberculosis and their lethality was 7.2% versus 1.8% for patients with pure silicosis and 0.8% for persons with other occupational pulmonary diseases. By improving working conditions in the mines and controlling the tuberculosis epidemic, the incidence of silicotuberculosis is significantly reduced. Today, we observe it in inspected, tuberculin-positive individuals over 50 years with advanced silicosis. Except for acute silicosis, this complication is more frequent in contact with tuberculosis bacilli excreters, under the influence of adverse factors (alcoholism, diabetes, malnutrition).

It is **ethiopathologically** proven that the silicotic tissue in the lung predisposes to the development of tuberculosis. In a significant number of cases, silicotuberculosis begins as a mixed process or is applied to reactivated old tuberculosis outbreaks. The two ingredients are difficult to distinguish not only from the dissecting table, but also histologically.

Clinical picture. The initial stage is usually uncharacteristic, with scarce symptoms - complaints of easy fatigue, increased tiredness, subfebrile temperature, productive cough, or bleeding, shortness of breath. These complaints are generally attributed to the established silicosis. In the case of poor professional history, the opposite is observed - the late forms of silicosis found in prophylactic X-ray examinations of former miners are being treated for tuberculosis. Mixed forms of silicotuberculosis often produce characteristic clinical manifestations only after cavern formation.

Paraclinical examinations. X-ray diagnosis is essential for the diagnosis because clinical manifestations may be missing or covered with silicosis. Any violation of the symmetry, distribution, and uniformity of the silicone nodes in both chest halves makes it necessary to think of overstated tuberculosis. It is characterized by apico- caudal development, whereas silicosis begins symmetrically in the mid-lateral lung fields. The unilateral fusion of blurred shadows in conglomerates with narrowly defined borders and their association with the hilus with a lymphatic pathway is highly questionable for silicotuberculosis. Also associated with this disease are parietal pleural adhesions, poor bullous emphysema, presence of cavity shadows with draining bronchus, tendency to cirrhotic constriction with mediastinum, trachea or diaphragm adduct.

An important role is played by the repeated study of sputum for tuberculosis bacteria. When finding mycobacteria in sputum-without cavern, a lymfobronchial fistula is sought from reactivated old tuberculosis outbreaks in the hilus lymph nodes.

The presence of accelerated BSR, moderate leucocytosis, or monocyte and tuberculin hyperergasia helps diagnose. The blast transformation of lymphocytes after provocation with tuberculin can be beneficial.

Diagnosis. It builds complex on the above signs and includes cases where there is no possibility of differentiating the two components. Then we talk about a mixed form of silicotuberculosis and refine the phase of the tubercular component - degradation, infiltration or consolidation without signs of activity. When differentiated after diagnosis, we describe in brackets the ingredients, e.g. Silicotuberculosis (second stage silicosis, infiltrative-pneumonic tuberculosis 1 k/0 in decomposition phase, BK /+/).

Differential diagnosis of silicosis and silicotuberculosis is difficult. In advanced silicosis, emphysema bulls around and in the fibrous areas are often accepted for caverns, despite their characteristic features - thin walls, plurality, lack of draining bronchus, wall infiltration, or fluid level.

Treatment. With modern anti-tuberculosis agents it is promising but harder and longer than that of pure tuberculosis. Operative treatment of resistant cases requires assessment of respiratory function not only now but also in the future in view of the expected evolution of silicosis.

Labour expertise. In the period of long-term stationary treatment and saturated chemotherapy in the home, the patients are incapacitated. After stabilization of the process, due to an increased tendency to relapse and limited recovery, the patients remain under observation in a second dispensary group and are recruited for 5 years. In determining the disability group, the incidence and severity of tuberculous and silicotic changes, the effect of treatment and the degree of functional impairment are taken into account.

Prevention. It is done above all by observing hygiene standards for air pollution, elimination of people with inactive tuberculosis outbreaks in the

professional selection and effective treatment of infectious forms of tuberculosis. An important role is played by chemoprophylaxis with HICK in silicosis patients, especially in the presence of risk factors or a lowering of the standard of living.

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T. Burilkov, Kr. Dinkova

5.3 ASBESTOSIS

The term "asbestos" encompasses a large group of hydrated needle-like silicates with a common property to decompose into fine and relatively flexible fibers. Industrial application finds the chrysotile serpentine (approximately 90%) and the crocidolite amphibians ("blue" asbestos), amosite and the produced in our country anthophyllite and tremolite.

The application *of asbestos in the industry is determined by the valuable combination of thermo-, noisy- and electro-insulating properties.*

The main ***exposed groups*** *are workers from the asbestos-textile and asbestos-cement industry, insulators, shipbuilders, furnace repairers and individual workplaces in the plastics, chemical and automotive industries.*

*Currently, asbestos (carcinogen IARC group I) in a number of products is replaced by less dangerous synthetic glass and ceramic asbestos needles or other materials. Since 1980, world asbestos production has declined steadily, as well as its use in Europe and North America, but has been growing in Africa, Asia and South America (a cheap material for construction, water and sewerage supply). The world's largest producers of asbestos are Russia, Canada and South Africa (in 1976, world production of asbestos was 5.7 million tons, in 1986 - 4 million tons and continued to decline).**

Asbestos minerals cause a variety specific **pathological effects** on humans:

- pulmonary asbestosis;
- pleural plaques;
- asbestos exudative pleurisy;
- lung cancer in asbestosis or after exposure to asbestos;
- mesothelioma of the serosal envelopes - pleura or peritoneum;
- asbestos warts type "foreign body" of needles in the skin.

PULMONARY ASBESTOSE

Pathogenesis. The rate of precipitation of needle particles in air is proportional to the square of their diameter until their length is of no importance (needles of 30 μm or more may reach the alveoli, whereas for isometric silica particles it is less than 5 μm). The needles hardly change their direction in the bronchial airflow - they are deposited mainly in the pulmonary base, and less in the upper parts. Fibrosis is the result of both mechanical irritation and phagocytic damage with subsequent enzymatic activation of fibroblasts and the onset of autoimmune processes.

Pathological anatomy. Pulmonary asbestosis begins as an alveolite characterized by the accumulation of macrophages, asbestos needles, and so-called. "Asbestos bodies". These are elongated formations of central mineral fiber and a segmented protein envelope with a positive color reaction for iron. Asbestos bodies are not evidence of disease but are pathognomonic for contact with asbestos. Over time, the alveolite finds its way into diffuse fibrosis with predominantly basal localization.

Clinical picture. The earliest symptom is dyspnea in physical effort. A typical physical finding is the crackling wheals in the lungs, combined with cyanosis and drum fingers. From this triad the most com-

* In Bulgaria and in several neighboring Balkan countries there are asbestos fields of industrial importance, which determine the so-called endemic asbestosis in farm workers, manifested by the formation of hyaline and calcified pleural plaques. In the eastern regions of Bulgaria and Belasitsa, over 1200 cases have been reported.

mon are the cracklings, referred to as "sclerophonia".

Clinical picture. In case of doubt about asbestosis, according to an accepted international standard, the X-ray examination includes an overview of the thorax and a l-profile with an angle of 45° instead of the usual 60°. Strial fibrosis is present mainly in the bases, sometimes accompanied by pleural plaques. Testing of ventilator function displays discrete distortions of a restrictive type that are increasing over time. Unlike silicosis, functional respiratory disturbances in pulmonary asbestosis outstrip the appearance of an X-ray finding. Exploration of the phlegm gives a characteristic finding - "asbestos bodies" and needles, also hemosiderin. Broncho- and the scintigraphic findings in the bases show bronchial or vascular branching in the form of "broom" and thus confirm the presence of basal fibrosis.

ASBESTOS PLEURAL PLATES

Pathogenesis. Asbestos needles penetrate the lung into the pleural cavity through the so-called retrograde lymphflow. From there, they are also eliminated via the lymphatic pathway through the parietal pleura. Prolonged retention of the needles there leads to hyaline growth, especially in the weakly vascularized areas.

Pathological anatomy. Asbestos-induced hyaline thickens of the pleural pleura are identified, where calcium is gradually deposited due to poorly blood supply. They are localized most often in the centrum tendineum of the diaphragm, the chest wall and the pericardial pleura.

Pleural plaques are clinically established after a latency period of at least 15 years after exposure to asbestos. They are diagnosed radiographically **(Figure 1)** or by ultrasound - the latter method has an advantage over the period prior to calcium deposition. In most cases, the plaques do not cause significant complaints and cause mild restrictive ventilator disorder, unless combined with pulmonary fibrosis. In the endemic regions in Bulgaria, 123 cases of pleural asbestos plaque have been identified (Burilkov, T. 1983).



Fig. 1. Diaphragmatic, pericardial and chest wall asbestos plaques

ASBESTOS TUMORS

Asbestos causes two different groups of tumors with different pathogenesis. ***Asbestos lung cancer*** is associated with pulmonary fibrosis. For this reason, its localization is predominantly in the foundations, but and multiple outbreaks of malignant degeneration are not rare. Smoking stimulates tumor risk. The disease occurs after a latency period of over 20 years from the onset of asbestos exposure. The clinical finding is not specific except the combination of pulmonary asbestosis and tumor. The basis for prophylaxis of this tumor is the prevention of pulmonary asbestosis by observing hygienic requirements. In the endemic regions of Bulgaria in exposed individuals with pleural plaques, but without or with minimal pulmonary fibrosis, there is no increase in pulmonary cancer.

Mesothelioma of pleura and peritoneum after asbestos exposure. The spontaneous frequency of mesothelioma is estimated at about one case per million inhabitants per year. Around asbestos mines and businesses, especially when using crocidolite ("blue" asbestos), have a credible increase in this rare tumor. Its pathogenesis is related to the presence of needles with certain parameters - long, thin and able to stay in the tissues for decades without breaking apart. Synthetic fibers with the same properties introduced into the pleural cavity of experimental animals also lead to mesothelioma.

Pathologically, we have mesothelial cell growth to a mass of rubber-like consistency that presses the lung. Metastases in remote organs are not common. Characteristic is the long latency period, which may exceed 30-40 years, especially in the case of low asbestos exposure. Clinically dominated by complaints of increasing dyspnoea and cachexia, chest pain, persistent blood pleural effusion.

Radiologically and in computed tomography **(Figures 2 and 3)** there is a massive parietal over-

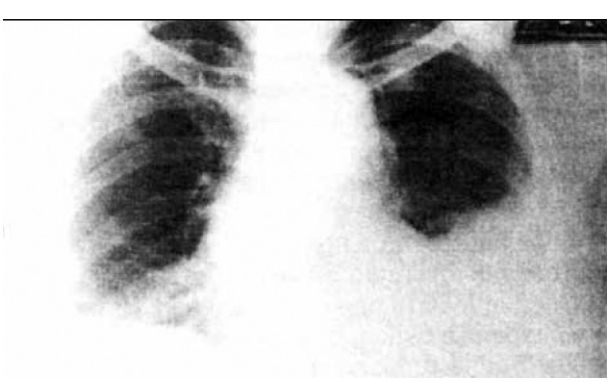

Fig. 2. Initial asbestosis with pleural mesothelioma

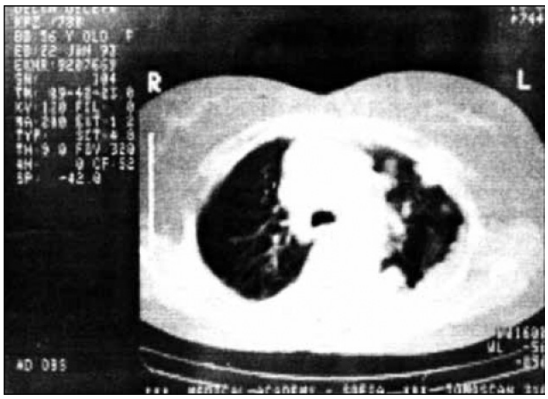

Fig. 3. Asbestos pleural mesothelioma

shadow that presses the lung. Peritoneal mesothelioma shows abdominal painlessness swelling, weight loss, drowsiness. Tumor mass is **palpated**. Diagnosis of mesothelioma is based on asbestos-related data, especially crocidolite or amosite, over a latent period of over 20 years, and a characteristic puncture finding.

Prevention. Due to the strong carcinogenic effect of crocidolite, its use in many countries, and in Bulgaria is forbidden.

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5.4 SILICATOSES

The term **silicatoses** is a common term for occupational pulmonary diseases caused by the inhalation of silicates - compounds of one or more metal ions with silica **(Table 1)**.

Approximately one third of the mineral breeds that occupy a significant proportion of the earth's soil and rock masses are silicates. ***The mineralogical varieties of silicates*** *are largely based on their crystalline structure, and the particle shape defines them as* ***isometric powders*** *(close sizes in the three dimensions). This gives some authors the opportunity to differentiate them into a group of non-asbestos silicates, unlike asbestos or* ***anisometric powders****, where in one dimension the size is significantly different from the others.*

The majority of non-asbestos silicates are foliar or lamellar ***(Figure 1)****.*

The specific identification of silicate minerals in pulmonary tissue can be accomplished by various techniques. Light microscopy with special coloring methods objectively reflects parenchymal changes occurring in response to swallowed particles and often gives evidence of the nature of dust exposure. Silicate

Tab. 1. Classification and composition of silicate minerals with pathological action

MINERAL	METAL CATIONS
Layered silicates	
Talc	Mg
Mica (muscovite)	K, Na, Al
Vermiculite	Al, Mg, Co, Fe
Kaolin	Al
Diatomaceous earth	
Montmorillonite	Ca, Na, Mg, Fe
Sepiolite	Mg
Attapulgite	Mg, Al
Skeletal silicates	
Feldspars	
Albite	Al, Na
Orthoclase	Al, K
Zeolite	Na, Ca, Mg, Al
Chain silicates	
Valastonit	Ca
Orthosilicates	
Mulit	Al

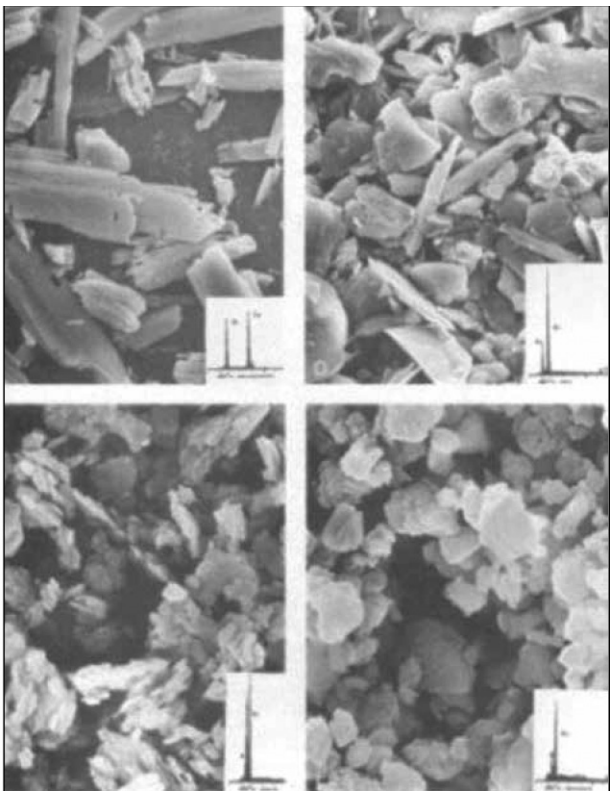

Fig. 1. Silicate minerals:
C. Valstonit D. Talc E. Kaolin F. Bentonite

minerals in the tissue sections look like polygonal particles, plates or needle-like structures, colorless or pale yellow to brown, but always biconvex. Phase-contrast light microscopy identifies fibers with 0.2 μm in diameter. Scanning electron microscopy visualizes smaller particles, and elemental components can be identified with X-ray dispersion analyzes.

Biological effects. *A variety of biological effects characterize silicates - from inert dust depots and harmless granulomas to diffuse fibrosis. While asbestos and similar needle minerals are highly aggressive compounds causing diffuse pulmonary fibrosis, non-needle silicates cause the formation of "foreign body" granules (kaolin, nefelin, olivine, mica, etc.) or are practically inert compounds (bentonite, cement, glass wool). However, silicates often contain quartz or asbestos impurities, or their thermal treatment is associated with the formation of high-temperature quartz, the action of which on the body is characterized by a mixed and varied pathology that has been expressed in terms of mixed pneumoconiosis, mixed silicosis, silico-silicatoses, etc. It is generally accepted that the possibility that the majority of non-asbestos silicates used in industry can cause disease is limited. It grows only in response to high exposure levels, especially in the presence of quartz and needle-like silicates. Although relatively rarely diagnosed, some of the pathological reactions caused by these powders are specific and are not found in subjects exhibited only with quartz and asbestos.*

TALCOSIS. This is the name of pneumoconiosis combining symptoms similar to silicosis and asbestosis (depending on the composition of the powder) and the specific clinical and morphological features of lean talc lesions. Talc is hydrated magnesium silicate ($Mg_3Si_4O_{10}(OH)_2$) with scaled form, but technical talc generally contains asbestos, mica, serpentines and quartz impurities.

Workers are exposed to the paper, paint and pharmaceutical industry, rubber and battery production, as well as workers involved in the production and processing of gypsum.

The pathogenetic mechanisms of action are different for the individual components of the powder aerosol. Pure talc causes the formation of mild peribronchial fibrosis from focal to more diffuse in workers with prolonged exposure to high concentrations of talc. The deposition of talc in the parenchyma provokes focal clumps of the type of "foreign body" granuloma. A quartz impurity in the talc or the content of crystobalite in its calcination up to 1200 ° C causes the formation of silicate granulomas and diffuse interstitial fibrosis of nodular type. Morphological features such as interstitial fibrosis, "asbestos bodies", pleural swabs and plaques are characteristic of the action of asbestos (tremolite, anthophyllite) impurities. Consequently, in the pooled term talcosis, different palomorphological findings are combined but with similar clinical symptoms.

The clinical picture is characterized by a slow onset of shortness of breath after prolonged periods of work, coughing and mucus expectoration. In advanced stages, particularly complicated by infections, the clinical symptom of pulmonary emphysema and pulmonary heart with respiratory and heart failure is manifested - constant dyspnea, cyanosis, drum fingers.

The physical finding is scarce, represented mainly by basal crevices. Significant ventilator disorders are absent in the initial stages, while diffuse interstitial fibrosis is accompanied by restrictive ventilator disorders. The X-ray traits represent different forms of pulmonary damage corresponding to the talc composition **(Figure 2)**. The nodose form of fibrosis, with reticulo-nodal shadows in the middle fields and the tendency to progression and fusion is characteristic of quartz impurity in the gypsum. The presence of diffuse interstitial fibrosis and pleural plaques is associated with an asbestos impurity. In the extraction and processing of pure talc, or in professional contact with medicinal or cosmetic talc, talc granulomas of the "foreign body" type have been observed in the pulmonary interstitium, in the absence of subjective complaints and ventilator disorders for a long time. Experimental and epidemiological studies sug-

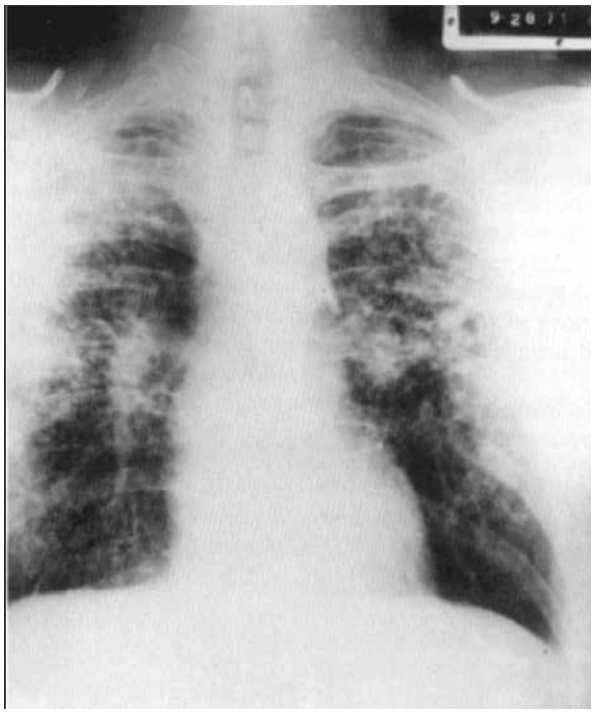

Fig. 2. X-ray of a worker exposed to cosmetic talc for 27 years

gest that pure talc is considered to be less fibrous in man - the disease occurs after 15-20 years of work and progresses slowly.

Treatment is symptomatic.

Labour-expert assessment is based on functional disorders in the respiratory and cardiovascular activities.

Prophylactic measures, in the case of quartz or asbestos impurities, are consistent with those for biologically active powders.

KAOLINOSIS. The impact of dry clay dust on its extraction, crushing, preparation of articles and their treatment is attributed to the group of pneumoconiosis, but in the majority of cases it is about moderate interstitial or small-pulmonary pneumofibrous changes without progression.

The most common silicosis in this group is kaolinosis caused by inhalation of kaolin - a group of clay materials, with a major representative - kaolinite ($AL_2O_3.2SiO_2.2H_2O$).

Kaolin has a wide application in the ceramic and porcelain-faience industry, paper, paints, rubber, medicine and cosmetics. The largest developed field in Bulgaria is in the region of Kaloinovo; other kaolin-related precipitates, white earth, bentonite, some of which have a significant quartz impurity, are also obtained.

Epidemiological studies on workers from the production and processing of kaolin in our country identify diseases of pneumoconiosis with a frequency of 1 to 9%, occurring after prolonged exposure to dust.

Technically pure kaolin produces poorly expressed fibrosis, with numerous inclusions of "foreign body" granulomas.

The clinical picture is poor in subjective symptoms, in the absence of early stages of functional breathing disorders. More substantial ventilator disturbances are possible in the formation of fibrous clusters with peak position and concomitant bronchitis.

X-rays are observed in small-scale shadows, in the context of reticular perialveolar and peribronchial fibrosis, with no progression, to diffuse fibrosis with alveolar obliteration or "honeycomb" type, at severe disease rates, when morphological findings correlate with kaolin content in the lungs **(Figure 3)**.

Significant quartz impurities in the powder pose a high risk of pneumoconiosis, which occurs more recently in the silico-silicatosis type. Thus, in the production of fire-resisting materials, pneumoconiosis from chamotte (containing 20% or more SiO_2) is particularly prevalent. The development of the disease is preceded by prolonged work experience over 10 years and rarely exceeds the first stage of interstitial-focal fibrosis. The histological finding is represented by peribronchial macules and nodules and shows significant kaolinic deposition in complicated pneumoconiosis. Only in individual cases, with massive dust exposure, heavier forms, similar to silicosis, occurring with granuloma confluency and tumorous transition have been observed.

Treatment is symptomatic. Systematic measures for sanitary-technical **prophylaxis** are required, limiting dust emissions, including hydrocycling processes. Medical prophylaxis is aimed at early diagnosis of dust diseases and timely rehabilitation.

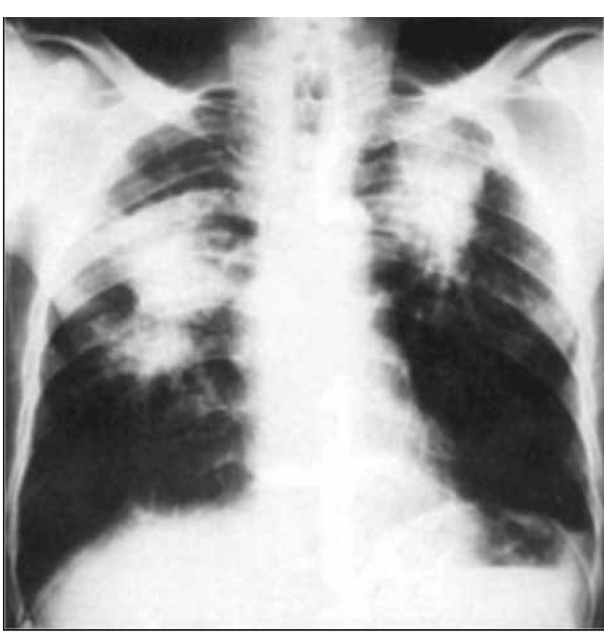

Fig. 3. Roentgenography of a kaolin worker with 34 years of professional exposure

NEPHELINE PNEUMOCONIOSIS. The disease is caused by nefelin - an aluminum silicate extracted in a ore deposit on the Kol'ski peninsula (Russia). Used in the production of aluminum and phosphorous fertilizers (apatite). In prolonged exposure it causes benign interstitial fibrosis of reticulated and spotty-striped shadows. The course of the illness is slow, rarely complicated.

APATITOSIS. Apatite is a powdered substance derived from nepheline, which has significant X-ray contrast. By chemical composition, it is phosphate anhydride, with calcium, iron and fluorine impurities.

Primary forms of apatitis have been observed in our country in workers in apathetic port silos after a labour experience of 8 to 10 years.

There is no specific clinical symptomatology and significant ventilator disturbances. X-ray changes are represented by intense, fine-grained shadows included in a fine mesh pattern. A characteristic finding is the presence of single, well-defined round shadows in the middle and lower lung fields of the "pea-disease" type. In some cases, changes in the bone structure of the pelvis and the ribs associated with the fluorine content are described.

OLIVINE PNEUMONOMICOSIS. Olivine - orthosilicate of magnesium and iron is used in the fire-resisting and ceramic industry, is present in the composition of blasting slags and some alloys.

Olivine pneumoconiosis occurs in the type of small meshy fibrosis of interstitial type, with small foci in the middle and lower pulmonary fields, often accompanied by pleural adhesions. Cellular-dust foci contain, besides olivine and coal and iron pigments, giant cells, collagen fibers. The disease occurs as a benign process, and regression of X-ray morphological changes is observed.

Olivinosis is relatively rare and not very relevant in our country.

MICA PNEUMOCONIOSIS. Having the properties of heat- and thermo resistance, mica is widely used in electrical engineering, for windows of furnaces and ovens, as insulating material and others. Mica is a complex compound of flaky aluminum, magnesium and sodium silicates - muscovite, biotite and phlogopite.

Mica-induced coniosis is a mild form of interstitial fibrosis with a benign course.

In **the clinical picture**, the symptoms of atrophic catarrh of the upper respiratory tract and the bronchial physical findings, with a progressive dyspnoea, prevail.

A characteristic finding in pulmonary parenchyma is "micous corpuscles", similar to asbestos, with a positive reaction to iron. Pleural plaque findings related to the non-isometric platelet type of the particles or to an unrecognized asbestos impurity are also described.

The treatment is sanatorium-resort and the working capacity is preserved.

The role of sanitary-technical **prophylaxis** is leading.

CEMENT PNEUMOCOMINOSIS. The cement industry, which uses powdered materials (clays and limestone, with gypsum, trace, etc.), is associated with intensive dusting.

The main Portland cement materials also contain SiO_2 with high hardness in the structure of quartz sand, quartzites, etc., with the free SiO_2 content in the fine powder being up to and about 2%. Probably due to the relatively stable content of SiO_2, portland cement does not cause pneumoconiosis, and the morphological substrate of the disease is predominantly dust granulomas in the absence of fibrosis. High dust concentrations cause pulmonary bronchitis, but epidemiological studies have a lower incidence than expected, due to the good solubility of cement in the biological media.

Cases of bronchial allergies and contact cement dermatitis of eczematous type are due to a chromium impurity in the cement. More puzzolan and fast bonding cements containing more quartz are more aggressive.

Professional contact with asbestos cement provokes broncho-pulmonary and pleural pathology described in the asbestosis section.

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5.5 ANTHRACOSIS

Coal pneumoconiosis (anthracosis, anthracosilikosis) is specific to a disease caused by the inhalation and retention of coal dust in the lungs and the response of the lung tissue (this pneumoconiosis must be distinguished from the lungs in coil miners which includes chronic obstructive pulmonary disease).

In the production of coal dust with different fibrosogenicity, the response of the lung tissue is heterogeneous. Three major variants of pulmonary events are distinguished:

- *Collagen coal pneumoconiosis (nodose form and progressive massive fibrosis);*
- *Non-collagen (chronic obstructive pulmonary disease) caused by coal dust;*
- *Pure silicosis or anthracosylicosis - with a high content of quartz in the coal mining powder.*

The risk of the occurrence of coal pneumoconiosis is determined by the nature of coal:

- *Coal with high carbonation (anthracite, bituminous, brown) is the most fibrousogenic effect.*
- *The percentage of free crystalline silica (quartz) in the fines fraction of the powdered aerosol plays an important role - most often about 5-6%.*

Pathogenesis of collagen coal pneumoconiosis. Major pathogenetic moments are:

- opsonization and phagocytosis of the macrophage coal particles;
- clustering in the localized particles of macrophage, blood mononuclear cells, polymorphonuclear cells and others;
- activation of macrophages.

It is believed that O, H_2O_2, NO, cytokines, proteases and antiproteases play an important role in the remodeling of the cells in the respiratory trakt.Superoxide radicals (O, H_2O_2) may be of a cellular and extracellular origin.

Participation in the formation of pulmonary fibrosis of cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukin-8 (IL-8), produced by blood monocytes activating a cell-mediated immune response.

Studies in vitro and in vivo with coil powder show impaired leukocyte factors such as leukotriene B_4 (LTB_4), platelet derived growth factor (PDGF), monocyte chemotaxis protein-1 (MCP-1), tumor necrosis factor - alpha (TNF- A), neutrophil adhesion factor and intracellular adhesion molecule (ICAM-1).

The coal particles may produce other factors with the possibility of modeling the lung cells and / or extracellular matrix: fibroblast chemoattractants such as transforming growth factor - beta (TGF - β), plate-

let-derived growth factor (PDGF) and fibronectin, and several stimulate or inhibit fibroblast proliferation factors (tumor necrosis factor - alpha (TNF-α), transforming growth factor - beta (TGF-β), platelet-derived growth factor (PDGF), insulin growth factor (ILGF) and prostaglandin - E_2(PG-E2).

Presumably participation in a utoimmune pathogenetic mechanisms.

It is suspected a role of HLA antigen expression in the formation of progressive massive fibrosis.

Pathomorphology. *There are two major forms of collagen anthracosis:* 1) simple coal pneumoconiosis and 2) progressive massive fibrosis.

The initial lesions in coal-simple pneumoconiosis are coal macules (black areas of 1 to 4 mm in size), located bilaterally symmetrically in the lungs, most intensely in the peak zone. They are surrounded by focal emphysema in a perimeter about 1-2 mm. Fine reticular fibers are found in the macules. Usually there is no collagen. Elastinic fibers disappear in the emphysematous sections. Common are vascular changes associated with coal macules and interlobular pleural thickenings. *Massive progressive fibrosis* is represented by massive lesions, often involving more than one lobe. At the center of the lesions there is a cavity filled with homogeneous black matter. The walls are smooth or with fibrous drags on the surrounding tissue. The vessels are destroyed. The lesions detect protein of unknown origin, calcium phosphate and coal dust.

Clinical picture. *The subjective symptoms* of coal pneumoconiosis are not pathognomonic.

Simple coal pneumoconiosis is manifested with scarce symptoms:

1) easy fatigue in physical effort;
2) weight in the chest.

Progressive massive fibrosis is characterized by:

1) cough;
2) black slime sputum;
3) shortness of breath.

Chronic dust bronchitis and emphysema caused by exposure to coal dust are characterized by:

1) permanent cough with expectoration
2) significant airway obstruction
3) dyspnea;
4) signs of respiratory failure.

X-ray changes in coal pneumoconiosis precede respiratory disturbances (VC and FEV_1). They completely resemble silicosis finds.

Simple coal pneumoconiosis is manifested radiographically (ILO'80) with scattered oval (nodosal) shadows of varying sizes (p - up to 1.5 mm, q - 1.5 mm to 3 mm, r - 3 mm to 10 mm) and often - perifocal emphysema.

Progressive massive fibrosis (ILO'80) is characterized by: "progressive massive fibrosis" **("PMF")** shades of varying diameter (A-shadows - 1 to 5 cm, B-shadows - 5 cm, to the diameter of the upper 1/3 on the right pulmonary field, C-shadows - with a diameter larger than that of the B-shadows).

Functional ventilatory disorders. *In simple coal pneumoconiosis,* functional deviations are:

- small airways disease with a reduction in maximum expiratory flows ($FEF_{25\%}$, $FEF_{50\%}$ and $FEF_{75\%}$) from the forced vital capacity curve (FVC);
- normal or slightly reduced diffusion capacity (DL_{CO}, D_M);
- altered ratio between dead space and total lung capacity (VD / VT).

"PMF" type coal pneumoconiosis is characterized by:

- reduced vital capacity (VC);
- reduced diffusion capacity (DL_{CO}, D_M);
- reduced lung complaint (CL).

Ventilatory defects in *chronic pulmonary bronchitis* are:

- decreased forced vital capacity (FVC);
- decreased forced expiratory volume for one second (FEV_1).

The progression of simple coal pneumoconiosis to *"progressive massive fibrosis"* is usually eight or more years.

The most common complications of coal pneumoconiosis are *a combination of rheumatoid arthritis - Kaplan's syndrome* (two-sided scattered nodose shadows, underlying rheumatoid nodes, progressive course, poor prognosis).

Diagnosis and differential diagnosis of coal **pneumoconiosis** is similar to silicosis.

Treatment is similar to silicosis. *Chronic pulmonary bronchitis* is treated in the conventional way for the treatment of bronchitis.

Prevention - as with silicosis.

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5.6 METALCONIOSIS

This name combines injuries from inhalation of metals in the form of pure or mixed powders. Respiratory organ damage has its own pathomorphology and clinical-radiographic characteristics.

CONIOSIS OF THE IRON

The variety of observed injuries necessitates the distinction of individual forms:

1. Siderosis

At the end of the 19th century, Zenker first described siderosis. A major risk group under modern conditions exists in electric welding or gas cutting where the iron evaporates and condensation aerosols are formed. The risk group is silver polishers using iron oxides.

Pathoanatomical in lung interstitium there are iron particles, which are predominantly concentrated in macrophages and lymphatic pathways. The iron content exceeds 15 times the norm in dry lung tissue. Siderosis is a pneumoconiosis of the so-called pulmonary thesaurosis and may develop back after discontinuation of exposure.

Diagnosis is based on its characteristic occupational history, preserved respiratory function, and X-ray picture, which is characterized by spotted shades of p-type, sharply outlined and more intense.

Employability. It is preserved in uncomplicated cases. Change of work is required for workers with X-ray morphological changes.

2. Siderosilicosis

The hazardous professions of this disease are associated with iron extraction, mostly in the form of hematite and in some processes in the industry, where an iron aerosol containing a significant amount of quartz (casting cleaners) is mechanically formed.

Patho-anatomical alterations are associated with the biological action of quartz rather than with the available iron compounds. Diffuse fibrosis predominates. The lung has a reddish-red color.

Clinical picture. There are complaints of easy fatigue, shortness of breath, coughing. The sputa emit may be reddish in color.

X-ray picture is characterized by interstitial fibrosis. This determines the development of obstructive or mixed ventilator disorders. The course of the illness is slow and occurs of over 10-15 years.

The so-called "Labrador lung" (in the Labrador iron mines) is due to the impurity of anthopholity asbestos. In this case, hyalinic pleural plaques are added to the picture of mixed pneumoconiosis.

Siderosilicosis can be observed in ochre miners and grinders and other earth paints - siena and umber. In this case, mixed silicosis is due to the quartz content of the clay. X-ray changes are in a gentle spotted nature on the backdrop of a generally reinforced meshed lung pattern.

ALUMINIUM PNEUMOCONIOSIS (ALUMINOSIS)

The main forms here are:

1. Bauxite pneumoconiosis (Shiver's disease - first described in 1947).

It is encountered by workers engaged in the extraction and processing of ore.

Pathogenesis. The intensity of lung processes caused by fibrous powders depends on the chemical composition, the crystal structure and the properties of the surface and the dust particles. This also determines the importance of the high temperature modifications of quartz - crystobalite, present in bauxites.

Pathologically, the lungs are rigid, with a bluish color. Pleural adhesions and bulls can also be found.

Clinical picture. The usual complaints are found - shortness of breath, sometimes paroxysmal, easy fatigue, non-systemic cough. Anamnestic evidence of severe chest pain, sudden increase in breathlessness and cyanosis, is pneumothorax.

Radiologically, diffuse fibrosis is detected.

2. Aluminum lung

The disease occurs in workers in contact with a finely dispersed aluminum powder - in the grinding of duraluminous panels and joinery, in the warfare (flammable bombs) and as a dye.

Pathogenesis. Aluminum and its compounds lead to primary bronchopathy with a latency period of months, but can be up to years (15 years). It is characterized by diffuse interstitial fibrosis with widespread wrinkles, emphysema development and spontaneous pneumothorax tendency.

Clinically, complaints include dyspnea, at first on exercise, and then at rest, which is accompanied by coughing and expectoration.

Radiological changes are of type "s" and "t", pneumothorax, due to the bursting of emphysematous bulls. There is a mixed type of ventilator malfunction. A late complication is the chronic pulmonary heart. Typically, the listed findings can occur quickly (after several months of practice).

PNEUMOCONIOSIS OF SOLID MATERIALS

The new studies consider the cobalt contained therein to be used in metal alloy steels. Of great interest is the large use of cobalt alloys in jet engines and the production of ferromagnets. Pure metal is used for staining glass and glazes.

Clinical picture. Complaints occur after exposure over 10 years. They are nonspecific - cough, expectoration, dyspnoea. They resemble chronic bronchitis accompanied by persistent conjunctivitis and paraclinical with hypergammaglobulinaemia.

Radiologically, there are enlarged hilus shadows, reticulonodous shadows with pre-selection in the middle and basal lung fields. The cardial contour may be flat.

Functional examination of external breathing determines it from a restrictive type, and then from an obstructive, accompanied by disturbances in gas exchange.

The presence of cobalt ions in the cooling fluid when milling the metal can lead to the picture of **exogenous allergic alveolitis**. A positive epicutaneous test with cobalt was established.

BERILIOSE

Industrial applications find beryllium fluoride, chloride, nitrate and sulphate - in the production of steels, fluorescent lamps, X-ray tubes. Its most extensive application is in nuclear energy.

Pathogenesis. Today, it is associated with immune mechanism unlocking. Antigens (easily soluble protein-bound beryllium compounds) lead to IV type-delayed type hypersensitivity reactions. This brings the chronic forms of berylliosis closer to the group of collagenoses.

Clinical picture. The similarity of systemic disease with chronic berylliosis determines its interpretation with detailed occupational history, even after discontinuation of exposure. In connection with the development of emphysema the leading symptom is dyspnea. Consequently, coughing, broken gas exchange, overloading the small circle of blood circulation leading to a pulmonary heart.

Radiological changes are not typical. Increased roots, enlarged pulmonary pattern are seen, gradually blurred shadows thicken to form "honeycomb" or diffuse, scarry fibrosis.

Diagnosis. It is based on proven professional contact with beryllium, immunological data for increased IgA, beryllium test for blast transformation of lymphocytes, epicutaneos test with 1-2% solution of beryllium fluoride, sulphate or nitrate (positive for contact persons), slowly developing diffuse fibrosis.

Differential diagnosis is made with sarcoidosis.

Treatment. It is symptomatic and does not lead to permanent remission. Corticosteroid preparations are used. Recently, "pulse therapy" - a short course with high doses to avoid complications.

STANNOSIS

The disease develops in inhalation of the condensation aerosols of tin - by melting the enriched ore and pouring the pure metal.

The clinical picture and **functional** studies show no changes. They contrast with the rich X-ray find - the lung are sharply delineated in thick, miliary shadows with significant frequency.

MANGANESE PNEUMOCONIOSIS

As an etiological factor, manganese plays a role in the extraction and processing of ores (meaning their composition) and in the production of ferro-manganese.

The mechanism of pathological reactions is closely related to protein structure degradation, sulfur-containing amino acids, enzyme activity and metabolism of biogenic amines, in particular neurotransmitters due to destruction of dopaminergic neurons.

Clinically, a cough with moderate expectoration, obstructive or mixed type ventilatory failure is observed.

Changes in functional indicators point to small airways obstruction. In the case of underground workers the concentration of trace elements in biological fluids is increased - manganese, lead, copper, zinc and iron.

Radiological changes of type "s" and "t" were detected, and over 10 years of reticular shadow with micronodose inclusions "p 1". Changes by the respiratory organs are characterized as toxo-irritative bronchitis. They are due to early lymphatic transport of the aerosol to the bronchopulmonary lymph nodes. Bronchitis and peribronchitis develop with predominantly basal localization and peak emphysema.

Manganese pneumoconiosis is associated with neurological manifestations (including early parkinsonism).

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5.7 MIXED PNEUMOCONIOSIS

PNEUMOCOMOINOSIS IN FURNACE REPAIRS

The disease occurs in workers from the metallurgical furnace, glass and other industrial furnaces, the construction of new thermal insulations. Pneumotropic damages - high dustiness, toxo-irritative gases and overheating microclimate - are the leading roles. The inhaled powder aerosol contains free crystalline silicon dioxide, asbestos, chamotte, dinas.

Pathogenesis. Two strong fibrousogenic components - quartz and asbestos, play a role in the development of mixed pneumoconiosis.

Clinical complaints are limited to easy fatigue, dry cough, and shortness of breath with physical effort. They occur after the impact of the harmful factors over 15 years.

The X-ray finding is two types: when the basic component of the inspired aerosol is asbestos, the basal stripped shadows predominate, and when exposed to refractory dust - nodose with a tendency to confluence. With computer tomography, hyalinic, but rather calcified, pleural plaques can be detected.

The evolution of this pneumoconiosis is slow. Typical here is the appearance of early hypertension, which is associated with the overheating microclimate. Given the slow evolution of the disease, the significance of the cocarcinogenic action of asbestos for the development of lung tumors has not been proven.

PNEUMOCONIOSIS IN ENERGY REPAIRS

The risk group here is working with asbestos-containing materials by spraying (injecting asbestos, cement and water glass as a pressurized mixture). Heating increases the fibrous- of silica and quartz silicates and lowers that of asbestos.

Clinical picture. It does not differ from that of the furnace repair workers.

Radiological (changes are caused by the action of asbestos) - stripped shadows in the two lung bases, hyaline pleural plaques, unclear diaphragmatic and cardiac contour.

POLYVINYL CHLORIDE PNEUMOUCONIOSIS

First polyvinylchloride pneumoconiosis (PVC) is described by Zsende et al. In 1970.

In a production environment chronic exposure occurs in the production of PVC products - fibers, electrically insulating materials, tubes, resins. The peculiarities of the technological process of the different productions determine the difference in the harmfulness. It is assumed that the combined effect of dust and toxic agents increases the appearance of changes in the respiratory system.

Clinic. Chronic exposure to PVC powder during resin production is associated with the development of pneumoconiosis at over 10 years. The subjective and physical symptoms are scarce and often absent. Examination of the external breathing function did not show significant changes in pulmonary volumes. A decrease in FVC and FED_1 was observed.

X-ray changes are characterized by the appearance of gentle, predominantly stripped and rarely micro-nodal shadows that do not undergo further development. The X-ray picture corresponds to I or I-II degree of pneumoconiosis.

Vinyl chloride as a monomer systematically injure the skin of the hands, as in scleroderma (Raynaud's syndrome, bone cysts) and the liver in which it initiates a rare tumor - angiosarcoma.

There is also an association between exposure to PVC and lung carcinoma.

THESAUROSIS

In the systemic use of a hair spray, the main ingredient polyvinylpyrrolidone (PVP) describes this type of pneumoconiosis.

A small percentage of consumers of different sprays without PVP react with bronchoconstriction or a typical asthmatic attack by inhalation of the carrier gas freon.

FOUNDRY PNEUMOCONIOSIS

In the foundries, the workers are in contact with a mixed powder aerosol containing iron, quartz and silicates, which are in varying proportions. At the greatest risk here are the arc- welders and the gas-welders.

Pathological anatomy. Reticulin and collagen fibers in the lung are radially arranged in a star formation. The development of fibrosis is associated with the quartz component.

Clinic. A picture of mixed pneumoconiosis is added to that of chronic bronchitis. This is due to the high dustiness, the overheating microclimate and the toxo-irritative gases.

PNEUMOCONIOSIS IN GRINDERS

A mixed aerosol that contains metal particles and abrasives (when grinding metals with synthetic abrasives - electrocorundum, carborundum) can lead to the development of mixed pneumoconiosis. It is observed during an internship of 15-18 years.

Radiological changes are of the reticulum-micronodose type.

BARITOSIS

Injury of barium sulphate powder with quartz admixture develops the picture of mixed pneumoconiosis. Barium is used in painting in the form of white pigment - lithopone, in vulcanization, in the textile and porcelain industries, in X-ray contrast studies. The highest risk is the production of pure barium or its milling to lithopone.

Radiologically there are strong contrasting stripped shadows in the lungs. Termination of manufacturing contact leads to the clearance of the pulmonary parenchyma.

The treatment of all listed forms of metal and mixed pneumoconiosis is symptomatic, depending on the stage and severity of the disease.

Particular attention is paid to the prevention of respiratory failure, overdosage on respiratory tract infections and right heart rate overburden.

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5.8 ORGANIC POWDER

Organic powders are divided into: non-living - irritating or toxic and allergenic, and live - powders with bacteria and powders with fungi and spores.

The effects of organic powders on the respiratory system may be irritation of the bronchial mucosa, allergic and immune reactions of the "foreign body" type with formation of granulomas or for fungal infections, respectively. Allergic reactions to their metabolic products. In any case, an individual assessment of the work-hygienic factors and chemical-bacteriological analysis of the powder to solve the relationship between the dust profession and the pulmonary disabilities is required.

The following types of pulmonary lesions are distinguished in the respiration of plant dust depending on their mechanism of action:

1. Reflex bronchospasm (with or without bronchial hypersensitivity) as a result of mechanical irritation from the dusts.

2. Allergic bronchospasm, usually accompanied by bronchial hypersensitivity, which may be of the first type (IgE antibodies) of the third type (IgG antibodies) or of the fourth delayed type (sensitized lymphocytes), existing separately or in combination.

3. Pharmacological bronchospasm (with or without bronchial hypersensitivity) as a consequence of direct (non-immunological) secretion of mediators such as histamine, serotonin, kinins, etc.

4. Acute inflammatory bronchospasm caused by bacteria and viruses contained in plant dust.

Agricultural plant materials and products such as hay, straw, cereals, feed, etc. are good substrates for development at the appropriate temperature and humidity of a number of molds (microscopic fungi). The most important for pulmonary pathology as allergens are molds: Alternaria, Aspergillus, Penicillium, Termoactinomyces, Cladosporium. In the lungs, spores or parts of their mycelium can cause true mycoses - proliferation of the mold in the lungs, with subsequent sensitization of the organism; bronchial

* Professional pathology, ed. Alexieva Tz., Kiriakov S., Med. and phys., 1982

asthma or asthmatic bronchitis - direct sensitization to a mold without the development of mold in the lungs; granulomatous lungs - formation of specific granulomas with sensitization of the organism to the natural mold. Here is the typical professional allergic disease "farmer's lung", also called "threshers disease", "harvest fever" and others.

The "FERMER LUNG" was also known to B. Ramazzini (1713). The spores of actinomycetes Micropolispora Faeni and Termoactinomyces vulgaris are the cause. Most affected are breeders, folders, millers and others. The morphological substrate of the disease is interstitial or granulomatous inflammation of the lungs. An exogenous alveolite develops, for which "farmer's lung" is a classic example.

Clinical picture. After a few hours of latency, shortness of breath, fatigue, cough with scarce mucus expectoration and increased temperature, sometimes with fever and night sweats, appear. Discontinuation of contact with the harmful material leads to healing for several days or weeks. There are also severe acute cases with fatal outcome. Prolonged contact leads to recurrent pneumonia with development of pulmonary fibrosis and respiratory-heart failure.

In addition to the typical history, X-ray findings, serological tests, skin and respiratory tests, pulmonary biopsy are important for diagnosis.

The functional, acute phase of **allergic exogenous alveolitis** (hypersensitive pneumonitis) is manifested by a reduction in respiratory volume, oxygen diffusion capacity, alveolar arterial blockade, hypoxia. Our data in patients with exogenous allergic alveolitis in the fibrous stage show marked hypoxia with a tendency to hypercapnia and a minor decrease in act. blood pH, due to the compensatory increase of the excess of bases (BE) and standard bicarbonate (SB).

X-ray in acute forms, a fine-grained diffuse dissemination, more pronounced for the foundations, is found in the case of sharp forms. Often, the picture is similar to confluent infiltrates. Chronic forms are characterized by a rough, reinforced mesh-striped pattern, emphysema and pulmonary heart.

Two types of antibodies are formed in the body. Circulating class IgG antibodies have a major pathogenic significance for the disease, so it is related to type III allergic reactions. Parallel to these antibodies, typical anaphylactic antibodies - IgE class reagins are also formed during the course of the disease.

In skin tests 4-5 hours after intradermal administration of the antigens, a reaction occurs histologically as an Arthus phenomenon. Respiratory tests with allergens are of limited use, because after 5-6 hours they provoke a "farmer's lung" attack.

Diagnosis criteria for exogenous allergic alveolitis are:

I. Compulsory criteria:

1. Confirm the presence of exposure by proving antigens.

2. Respiratory or general symptoms 4-12 hours after exposure.

3. Detection of antigen-specific humoral or cellular sensitization.

II. Additional criteria:

1. Typical disorders of pulmonary function.

2. Characteristic radiological changes in the lungs.

3. Positive inhalation provocative test with the specific allergen.

Diagnosis can be confirmed by histological or bronchoalveolar secretion analysis.

Treatment requires removal of the patient from the harmful environment and the specific desensitization that prevents the occurrence of severe pulmonary sclerosis with its consequences.

BISINOSIS. It is caused by the inhalation of cotton, linen or hemp powder. The workers are most at risk in the primary processing of the material, where the dustiness is high. Smaller is the risk in spinning and weaving workshops.

The bisinosis pathogenesis has not yet been elucidated. Cotton powder contains histamine liberators and serotonin. Histamine is released from the lungs without prior sensitization. Constiratory action on the airways is associated with increasing histamine release and can be prevented with a small dose of antihistamine. The histamine liberator hypothesis explains the more acute breathing effect of the textile powder, but not the slow progression of the bisinosis to the chronic stage in some workers. Isolated allergens from the fibers of the textile fibers themselves, which are an E-enzyme resistant glycoprotein fraction. It is believed that the underlying effect of the glycoprotein allergen is Maillard's reaction with lysine-saccharides links formation. Antibodies in serum against cotton have also been demonstrated, with no significant relationship between them and the symptoms of the bisinosis.

Clinical picture. In the early stages, the bisinosis is characterized by chest tightness on the first working day after the rest (around the end of the working day or after leaving the workplace). If the disease progresses, tightness is accompanied by difficulty breathing, which goes on during the other days. Occasionally, the worker may experience symptoms on each working day with constant acute intolerance to the dust, which is necessary to remove from the professional environment. In the final stages, the disease is not distinguished from chronic bronchitis and emphysema caused by non-professional

factors (indicative in these cases are the data from the professional history of chest complaints in the past, expressed more strongly at the beginning of the week). Radiologically, no changes specific to bisinosis (a specific pathological finding in the lungs are also not detected - it is usually the same as for chronic bronchitis and emphysema).

Bisinosis has an **acute stage** that can progress to chronic irreversible disease. At the acute stage along with the symptoms on the first working day after the rest, there is usually a decrease in FEV_1. This can also be found in dust-exposed workers without complaints, more on monday than on other days. FEV_1 reduction during the workday is a characteristic phenomenon and initially is reversible. Epidemiological data indicate that workers who regularly receive acute bisinosis symptoms are at a higher risk of developing **chronic disease** than those who have never had such complaints.

There are three stages in the development of the bisinosis:

I stage - weight in the chest, shortness of breath, dry cough, rapid fatigue, mostly on the first working day of the week.

Stage II - these complaints increase in strength and duration, asthma-like crises are added.

Stage III - chronic spastic bronchitis with recurrent exacerbations leading to expressed emphysema.

In workers from the flax and hemp industry, besides these symptoms, febrile states, fever, have also been observed. In hemp processing, the temperature may reach 40 ° C, accompanied by headaches, chills, nasal bleeding, vomiting and diarrhea. These phenomena can last from 2 to 5 days. Sometimes there is also a general weakening, feeling dizzy and sex disturbances - reduced libido, metrorrhagia, abortion - so-called "hemp atrophy".

Skin testing by flax and hemp skin allergy tests show a positive reaction in about 50% and 51% of those tested, while a cotton allergen responds positively to a smaller number of exposed individuals - about 20% (more important is the inhalation challenge test with acetylcholine and cottonseed extract, with changes in FEV_1, eosinophils and blood gases).

Leading place in **prophylaxis** is the reduction of dustiness. Prophylactic importance is the proper medical selection of workers (with no evidence of chronic pulmonary disease and changes in respiratory function), prophylactic periodic examinations and studies for early detection of changes in the respiratory system.

PNEUMOCONIOSIS FROM CORN POWDER. It is caused by prolonged inhalation of cereal powder. Affected agricultural workers, millers, sillage- and port workers.

The impact of particulate matter leads to chronic granulomatous pneumonia terminating with pulmonary fibrosis. **Pathologically** anomalous formations are present in the lungs, hilus sclerosis and bifurcation lymph nodes, non-specific "foreign body type reaction, and so-called "corn corpuscles", in which protein envelope contains iron. Some authors treat them as a tissue reaction of the type of exogenous hypersensitive alveolitis.

Clinically, the disease at the onset of dust exposure often occurs with the so-called "corn fever" that lasts for 2-3 days and is characterized by fever, shaking, coughing, nausea. Later these ebullitions are repeated only with a significant dust load, and instead there is a permanent cough with expectora-

Tab. 1. Professional lung injuries from some organic powders

Name	Etiology and pathogenesis	Endangered professions	Clinical presentation and diagnosis
Wood dust and timber	cork powder (moldy)	production and processing of cork	hypersensitivity alveolitis, the presence of precipitins
Koniosporiosis "lung of maple barkers"	spore	fuller on the bark of maple	as "farmer's lung"
Sequoiosis	mushroom sawdust	processing of California tree	pronounced shortness of breath, serum antibodies from isolated mushrooms
Disease of poltry-farmer	antigens of serum proteins, egg proteins, feathers and droppings of birds, especially pigeons	poultry-farmers, especially pigeons	as "farmer's lung", manifests itself usually six hours after inhalation of antigens
Disease of mushroomers	spores and metabolic products of molds	workers in artificial mushroom cellars	as "farmer's lung"
Disease of pepper spliters		workers from the production of peppers	allergic hypersensitivity in the pulmonary alveoli - toxomycosis
Disease of cheese producers	spores of Penicillium casei	workers in dairies, cheese washing	usually delayed reaction type III, but at atopics - delayed reaction type I "asthma cheese producers"

Bagasosis	Thermophilic actinomycetes	Processing of waste from sugar cane (bagasse)	Fever, cough, shortness of breath, radiological shadows or planar infiltrates in the lungs, proof of relevant precipitins
Disease of silage fillers	fungal allergies	Workers employed in the preparation of silage	Febrile condition, phenomenon of chr. bronchitis and emphysema, may pneumosclerosis
Plant pulmonary granulomatosis	cellulose of powder from seeds of peas, beans, lentils	Workers in contact with the seeds of legumes	Such as acute bronchopneumonia, miliary dissemination radiographically in the lung fields
Disabled from dust of the grass sainfoin	dust of grass sainfoin containing acetylcholine or similar active substances	production of ropes and carpets	Shortness of breath, bronchopneumonia

tion and an exacerbation for years dyspnea. Sometimes pneumonia, dermatitis and increased allergic reactivity can be detected.

Radiologically there may be evidence of pulmonary fibrosis in cases of prolonged work experience.

Sanitary-technical equipment includes improvement of ventilation and mechanization of the working process.

DISABILITIES IN WORKERS FROM THE FLOUR MANUFACTURING. While the grainy powder can lead to pneumoconiosis, the flour attaches to the bronchi and causes chronic inflammation of the lining followed by atrophic changes. Evolution is slow and benign if bronchial spasm does not develop (gluten of flour or grain beetle may cause primary bronchial asthma). Allergic reactions can also be caused by Penicillium, Aspergilius, Acarina and other of contaminated flours.

Initial damage to bakeries is the development of rhinitis and bronchitis (at 9-10 years of exposure), then bronchial asthma and end of emphysema and pulmonary heart. X-ray examination usually reveals normal lung transparency and often increased pattern in the lung basis. Changes in external breathing (FEV_1, PEF, VC), according to some authors, are the initial signs of the harmful effects of flour powder. An acetylcholine inhalation test (for the detection of latent obstructive functional disorders) is recommended. Etiological significance includes skin-allergic and especially inhalation tests with the corresponding allergen. The demonstration of specific reagins (IgE) in serum of patients by radio-allergosorbent test (RAS) is a reliable method of etiological diagnosis in flour production workers.

Etiological treatment is specific desensitization. If necessary, patients are readjusted.

There are also many organic powders causing occupational respiratory trauma. It is known, for example, the sensitizing effect of some plant seeds - cotton, linseed, soybean, castor.

A number of occupational groups of workers are exposed to proteins of different origins - in the production of enzymes, fodder, yeast, leaven, antibiotics, insect farming.

Allergic diseases can also be caused by organic plant protection products. There are also diseases of the respiratory system of tobacco and other organic dust **(Table 1)**.

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5.9 PROFESSIONAL CHRONIC BRONCHITIS

The definition of chronic bronchitis (CB) is based primarily on clinical criteria including ***chronic coughing with expectoration in different daytime for at least 3 months in 2 consecutive years.*** This definition also introduces new requirements for CB determination - is there chronic airway obstruction; with what localization and degree; excluding other diseases with chronic cough and an expectoration. **In professional CB**, it is essential that the factors in the work environment are crucial to its manifestation.

CB is characterized by constantly growing, high prevalence. Globally it affects 12-25% of the adult population.

Accurate determination of the incidence of occupational CB is mainly hampered by its multifactorial conditionality. It is therefore one of the most disputed nosological units for inclusion in the Official list of occupational diseases (Convention 121, published by the Committee of Experts at the ILO 1980). Recent epidemiological studies by our and foreign authors have

shown that CB is more credible among some labour contingents. Studies in our country show that manganese workers have a frequency of 6.5 times that of controls. The CB rate for iron and steel workers ranges from 15-29% for men and 5-21% with women, without dust risk workers only at 6 to 13% (1990).

Therefore, more and more authors have subscribed to the view that, under strictly defined conditions and specific procedures, CB can be considered as an occupational disease. CB is listed in occupational disease lists (tables) except in our country (2008) and in countries of the European Community and the CIS.

Unprofessional etiological factors that enter into complex relationships with professional factors (when acting together) are:

1. Exogenous factors:
- smoking - a leading exogenous factor for the development of CB;
- viral and bacterial infections;
- environmental pollution;
- various exogenous allergens;
- low social status.

2. Endogenous factors:
- Immune deficiencies of Ig-A, α_1-antitrypsin;
- congenital abnormalities of bactericidal activity of macrophages and polynuclear leukocytes;
- AVN - non-selective;
- syndrome of non-moving cilia;
- growing age.

The main **etiological factors for a professional CB** are:

A) Powders - inorganic and/or organic together with the added microbial flora.

B) Toxic-chemical factors in the form of gases, vapors or condensation aerosols, which can be systematized in 4 groups:
- fast volatile organic compounds such as: acetaldehyde, acrolein, formaldehyde, paint solutions, phosgene etc.;
- hardly volatile organic compounds such as: dimethylsulphate, epoxy resin hardening agents, some isocyanates, naphthalene, paraffhenylenediamine, phthalic acid, solids paints, etc.;
- easily volatile inorganic substances such as: ammonia, chlorine, ozone, hydrogen fluoride, nitrous gases, sulfur dioxide, hydrogen sulphide, acid and alkali vapors, etc.;
- poorly volatile inorganic compounds such as metal powders, nickel smoke, cobalt, zinc, platinum and their compounds.

C) Meteotropic factors when operating in overheating or overcooling microclimate, ionizing radiation, etc. Their self-importance in the professional bronchitis etiology is not great.

Pathogenesis. The bronchial tree's ability to respond to multiple professional factors is the same. The main pathogenetic mechanisms are:

1. Disturbed local and cleansing protective function due to destruction of the ciliary epithelium and blocking of immunological and phagocytic protection.

2. Some powders and gases can model bronchial hyperreactivity (BHR) by blocking β2-adrenergic bronchial receptors or reflexively by direct vagal receptor irritation.

3. Along with its irritant action, some powders, especially organic and a number of chemicals, also have an immune-allergic or histamino-liberate non-allergenic effect.

4. Different etiological factors lead to tubular gland hypertrophy and hyperplasia of the bronchial gland, followed by hyper secretion and dissecretion (increased volume and altered bronchial secretion) and subsequent development of mucostasis and bronchial obstruction.

Pathology. The following major pathological changes were observed:

1. Proximal in larger bronchi - hypertrophy of the tubular gland with Raid index increase from 0.26 to over 0.59, hyperplasia of the acinar cells and numerical increase of the cupular cells with metaplasia of the multilayered cylindrical ciliary epithelium (replacing it with a multilayer flat epithelium).

2. Distal in small bronchus (less than 2 mm in diameter) - inflammatory infiltration of the bronchial mucosa, submucosal swelling and thickening of the basal membrane and later atrophy of glandular structures.

Obstructive and later destructive changes in the bronchial tree cause difficulty in passing the air through the bronchi. The subsequent alveolar hypoventilation and disturbance of the physiological ratio of ventilation/perfusion leads to limiting gas diffusion with subsequent arterial hypoxemia. Later in the evolution of CB and combining it with destructive changes in the pulmonary parenchyma, conditions for arterial hypercapnia and development of respiratory failure (RF) are created.

Classification. According to the etiological classification of chronic professional bronchitis, it is:

1. Powder
2. Toxic-chemical
3. Meteotrope
4. With mixed etiology

Depending on the way of its occurrence it is:

1. Primary - formed after prolonged exposure of the damaging factors of the working environment.

2. Secondary - when accompanying or compli-

Tab. 1.

Symptoms	Clinical signs	Laboratory Values	Other methods
Cough, expectoration, shortness of breath, tiredness	Dry and / or wet thorax wheezing, increased front-rear diameter, cyanosis, tachycardia, hepatomegaly, edema of the lower extremities.	BSR, hemogram, sputum for banal flora, BGA, SA tests (in allergic forms) with oflife and proff. allergens	Radiographic studies of lung and paranasal cavities, ECG EhoCG, FEB, broncho-provoc. tests, bronhological researches

cating another occupational pulmonary disease (eg pneumoconiosis, toxic pneumosclerosis) or as a consequence of acute occupational intoxications involving the respiratory tract.

Clinical picture. In the course of professional chronic bronchitis, there is nothing strictly specific that distinguishes him from the unprofessional.

The main clinical symptoms are:

1. Cough - early onset in patients with CB, manifested mainly in the morning and / or in contact with production damage. Depending on the disease phase (remission or exacerbation), the cough changes its appearance, strength and character.

2. Expectoration in qualitative and quantitative terms also strongly depends on the stage of the disease.

3. Dyspnoea and easy tiredness are later manifestations. The breath originally occurs at higher physical stresses, and consequently smaller, as in the end of the disease it is also at rest. If there is an allergic component, it may be of a leaping nature.

The physical examination may initially detect an increased anterior-back diameter of the chest with limited respiratory motility, wheezing - small moist non-bubbling in non-destructive forms and dry with different acoustic characteristics in the obstructive. In more advanced forms of occupational aetiology, chronic pulmonary heart (CHD) and central cyanosis respiratory failure can be identified.

The main clinical forms are chronic non-obstuctive bronchitis and chronic obstructive bronchitis.

With chronic exposures and frequent exacerbations, *chronic deforming bronchitis* followed by bronchiectasis is reached.

Depending on the etiological factor - concentration, dispersion and duration of action, as well as individual characteristics, certain nuances are observed in the course of chronic professional bronchitis:

- Powdered bronchitis of inorganic powder usually develops slowly, proceeding with a poorer clinic (as dry bronchitis) and for a long time the patients have compensated respiratory function and preserved working capacity.
- Powdered bronchitis from organic dust can occur both as a normal CB (with normal bronchial reactivity) and as asthma-like bronchitis with bronchial hyperreactivity, coughing and whistling repetation of a paroxysmal nature and unful expectoration.
- Toxic-chemical bronchitis occurs after a shorter exposure period. They end up with a richer clinic, often with functional complications, and more quickly lead to complications and disability. When it is caused by water-soluble gases, vapors and aerosols (chlorine, ammonia, acids, bases) primarily damage the mucous membrane of the URT, trachea and large bronchi. More difficult and insoluble (ozone, sulfur, beryllium, manganese, nitrogen oxides, gasoline, phosgene) affect the lower compartments, bronchiols and even the alveolar wall.
- Meteotrophic bronchitis factors affect directly on the bronchial mucosa and indirectly by creating conditions for frequent exacerbations.
- Mixed etiology CB are the most common forms, as in most occupations the risk factors are combined.

The clinical diagnosis is based on a thorough history and objective study of some laboratory, X-ray and functional studies **(Table 1)**.

It is important to know that:

1. X-ray examination of the lung has little information and serves mainly to exclude other diseases masked or associated with CB.

2. X-ray examination of the paranasal cavities, serves to detect any inflammatory changes present in them.

3. When diagnosis is made, both the etiological form and the clinical form of the CB (via FID), the developmental phase (remission, exacerbation) and any related **complications** (RF, CHD, bronchiectasis) must be determined.

Differential diagnosis. It is done with pulmonary emphysema, bronchial asthma, bisinosis, localized or spread pulmonary fibrosis and granulomatous processes, interstitial lung edema in left ventricular CF.

Diagnostic criteria for determining the occupational nature of CB.

The following basic criteria are used:

- Labour-hygienic - to prove the etiological factor, its intensity and duration of impact;
- Exposure period - impact of at least 10 years;
- Positive collective (epidemiological) test demonstrating a credible increase in CB in a given occupational group;
- Absence of smoking - exceptionally, only cas-

es with a duration of smoking below 3 years and an intensity of less than 10 cigarettes per 24 hours (smoking is demonstrated by quantification of the nicotine metabolite in urine-cotidine);
- Absence of chronic lung disease (in particular, CB) before commencement of work experience;
- Absence of inflammatory diseases of an infectious nature affecting the UAP - the exception is only where the profession predisposes to such (eg. low temperature at underground work and elevated humidity).

If there is a suspected presence of an allergic component accompanying CB, the following criteria are added:
- positive skin allergy and serological (immunological) tests to professional allergens
- positive broncho-provocative test to mediators and allergens
- positive exposure and elimination test

Treatment. In principle it does not differ from that of the unprofessional CB. Significance is attributed to the so-called etiological treatment - adequate rehabilitation (in case of functional complication of CB), in case of dequalification is determined the% of the lost working capacity, and in the case of the younger persons - the possibility of re-qualification. Proven allergic pathogenesis of CB requires compulsory rehabilitation, with a specific hyposensitisation with the professional allergen.

The pathogenetic treatment is aimed at combating infectious exacerbations, bronchoconstriction and altered rheological properties of bronchial secretion.

Bronchodilator treatment includes the use of various in structure and mechanism of acting drugs: xanthine derivatives (novfilin), inhalatory sympatho-mimetics (salbutamol, fenoterol, salmeterol, etc.), inhalant cholinolytics (ipatropium bromidum), and in asthmatic bronchitis and inhaled corticosteroids (beta- methazone, fluticasone). Therapy should be controlled and optimized according to FEB results.

Mucoscretory treatment is intended to liquefy the hyperviscosity of the bronchial secretion. It is achieved by *adequate hydration* (intake of increased amounts of fluids), inhalation affect with aqueous solutions of sodium bicarbonate and administration of *mucolytics and secretagogues* (bromhexin, mucosolvan, acetylcysteine, etc.). Different physiotherapeutic procedures (vibratory and percussion chest massage, postural drainage, inhalatory administration of mucolytics) are also found in treatment. An essential element of the complex treatment is the climatotherapy in suitable sea and mountain resorts, healing gymnastics with breathing gymnastics. Proof of complications - chronic pulmonary heart and/or respiratory failure - requires the administration of cardiac glycosides, vasodilators, diuretics and oxygen therapy (dosed and controlled according to BGA).

Labour expertise. In the absence of functional respiratory complications, persons are able to work, and in case of exacerbation of CB, they are temporarily incapacitated. In case of frequent exacerbations, temporary labour is recommended, and in case of functional breathing disorders (RF), with or without cardiovascular disorders, the% of the disability is determined. Cases of asthmatic bronchitis are permanently incapacitated for the profession without regard to functional breathing values.

Prevention. Sanitary-technical prophylaxis consists of measures whereby production deficiencies reach norms.

The medical prophylaxis of professional CB requires strict adherence to the rules for prior medical examinations in order to prevent people with chronic respiratory diseases from being at risk work environment. Quality medical check-ups and timely medical treatment with labour readjustment.

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5.10 OCCUPATIONAL DISEASES OF URT

The upper respiratory tract (URT) acts as a conditioning device for the inhaled air, which heats, moisturizes, cleanses mechanically and chemically, and retains and disposes of many microbial agents. An important role in performing these functions is the presence of chemo-, baro-, thermal and olfactory receptors in the nasal mucosa, the structure of the nasal cavities, the cavernous formations, the rich innervation of the nose and the swallow (n. trigeminus, n. olfactorius, n. vidianus, ganglion sphenopalatinum), defensive reflex mechanisms for elimination - sneezing, cough, increased secretion, spasms of the throat, etc., creation of reflexes through the center of breathing and regulation of the quantity of passing air and its physico-chemical properties.

In case of damage to the protective mechanisms of the nose, its filtration capacity is reduced sharply. In atrophic processes, the mucous membranes thinnish, atrophy the glands and nerve endings, reduce the number of cilia, suffer drainage, filtration and phagocytic role of the nose, and conditions for the occurrence of occupational diseases from physical, chemical and other injuries.

The lymphoid throat ring is actively involved in immunogenesis by the production of lymphocytes in the tonsils. Its superficial layout ensures close contact with harmful factors and plays an important role in the defense mechanisms of the body. Chronic tonsillitis as an infectious-allergic disease leads to allergy to the whole organism - physiological sensitization, e.g. with regard to hemolytic streptococcus becomes pathological and leads to frequent recurrences. URT occupational diseases are not always directly related to the length of service. They may occur due to physical (meteorological, ionizing radiation), toxic chemical (metals and their compounds, acids, bases, organic compounds, plastics, pesticides), biological (pathological bacteria, fungi, viruses) and allergic factors of work environment.

Diseases of URT by meteorological factors (high and low temperature, increased humidity or dry air, increased or decreased atmospheric pressure, etc.) Threatened professions include workers in metallurgy, machine building, mining, bakery, agriculture, etc.

Pathogenesis and clinical picture. At elevated temperature, the body reacts with capillary dilation, accelerated blood flow, increased sweating. On the part of the URT, nasal mucosal dryness, enlarged capillaries and haemorrhages are evident, there is no inflammatory process of the URT. Often in the case of bleeding, first aid is required through a front nasal tamponade.

When the body cools in the nose, the cavernous bodies expand, the conat becomes pale, and nasal frost, sinusitis, pharyngitis, tonsillitis, laryngitis often occur. Treatment is anti-inflammatory and symptomatic.

Diseases of URT in ionizing radiation. Radiation disease is complex damage to the body, which occurs acutely or chronically. On the part of the URT, bleeding from the nasal mucosa, gums, throad, which often lead to secondary infections in this area, is typical. The treatment includes local administration of Vit. C, A, B complex.

Diseases of URT from powder substances. In the production environment there are various dust factors that pass through and influence the URT:

- *inorganic:* metal (lead, cadmium, manganese, copper, zinc, iron), mineral (cement, sand, lime, gypsum etc.);
- *organic:* plant (cotton, flax, hemp, tobacco, flour), animal (wool, silk, leather);
- *mixed*.

For occupational diseases of solids a significant role play the dust size.

Pathogenesis. Dust is the most common cause of occupational diseases in the URT. The influence of dust on the URT depends mainly on the amount of powder aerosol, the size of the dusts, the degree of dispersion, the chemical composition thereof etc. The powder, depending on its nature (metallic, mineral, plant, animal, etc.), irritates the lining mechanically, chemically, via the infectious route or combined and causes inflammatory, vasomotor and secretory reactions. The mucus loses its protective properties, edema occurs, secretion increases, nasal cavities swell, worsening blood circulation, difficulty breathing. Changes that occur arise into chronic and develop a productive, hyperplastic process. The connective tissue grows most in the area of nasal cavities, the mucous glands atrophy, reduce the vessels of the cavernous bodies, the cylindrical epithelium passes flat and the atrophy rhinitis picture is formed. Erosions and necrosis with subsequent septum perforations develop.

Often the inflammation goes down and covers the mucous membrane of the throat. It is hyperemised, with enlarged blood vessels covered with a tough

fluid, hypertrophic changes occurring, which may be diffuse or cover only the lateral parts - lateral pharyngitis, the back - granulous pharyngitis. In late stages, the lining atrophies, becomes smooth and shiny.

Changes from the throttle go from lateral changes with dystrophy to complete atrophy. The mucous and vocal cords connections are hyperemic, edematous and with pachydermic changes. In advanced stages of longer work experience, the lining of the throat is dry, often covered with scabs.

Clinical picture. It does not differ significantly from that of non-professional aetiology. There are some differences depending on the length of service, the type of powder, the age of the patient, etc.

Events start with acute snuffle, often with subsequent inflammation of the nose cavities - difficulty breathing, sneezing, dryness, mucus secretion. This picture gradually subsides and passes into chronic - developing productive hyperplastic rhinitis. Nasal obstruction often disappears, but it is repeated again. With the development of atrophic rhinitis, dryness in the nose and throat increases, weakens the sense of smell. Occasionally there are changes in the nasal skeleton - perforations that only affect the cartilage and manifest with characteristic breathing. In the case of nasal cavities, rhinolitis is often observed - concrete inorganic elements in the middle nasal passage, resembling a cast (impress of the paranasal relief). Inflammatory changes in the nose and nose-swallow descent and cause pharyngitis and laryngitis. Workers complain of dryness, burning, pain, feeling of a "foreign body" in the throat, dry cough.

When cadmium powders are affected, there are atrophic changes in the lining of the nose, the mouth and the taste papilla, often erosions with calcium deposits. The picture of hypertrophic processes under the influence of zinc dust with subsequent atrophy and perforations of the septum is also characteristic[1].

The damage from plant dusts to URT is most often characterized by the catarrhal forms of rhinitis, granulosa and lateral pharyngitis, chronic tonsillitis, sometimes accompanied by allergic components.

Treatment. Local therapy is the leading - nasal drops and ointments, iodine-glycerin solutions, inhalations with broncholytic and antihistamine preparations.

Preventive measures include:

- sanitary-hygienic - improvement of technological processes, production ventilation, sanitary control;
- medical-healing - pre-medical selection, excluding workers with chronic rhinitis, nose septum dislocation, polyposis, pharyngitis, laryngitis, tonsillitis; periodic prophylactic examinations;
- personal protective equipment - anti-dust masks, respirators, etc.

Diseases of URT from gases and vapors. In the case of inhalation of gases and vapors, the ability to dissolve in liquids (water) plays an essential role. Various external and internal factors (temperature, air humidity, body condition - general and local: curved nasal septum, chronic rhinitis, pharyngitis, tonsillitis, laryngitis) also play a role in the onset of injuries. The general toxic and local disease effects depend on the concentration of the vapor, the nature of the action, the time of exposure.

Mineral acids in the form of aerosols lead to chemical irritation of the lining of URT, as hydrochloric acid damages predominantly URT due to its marked hydrophilicity, nitrogen acid is less soluble and mainly damages the lungs, while the sulfur acid damages equally in all parts of the respiratory system. In acute lesions coagulation necrosis of the lining occurs, manifesting as inflammatory changes of hemorrhagic nature in the nose, larynx and throat. Chronic catarrhalis, hypertrophic or atrophic changes are more common. Sodium and potassium bases have a predominantly local, chronic, irritating effect on the lining of the URT, resulting in chronic inflammation - catarrhal, hypertrophic, atrophic.

Clinical picture. Acute disabilities are rare, often due to industrial accidents, low qualification of workers. The mild forms are manifested by rapid passaged burning, irritation in the nose and throat. Medium heavy are expressed in painful cough, painful swallowing, bleeding and difficulty in breathing. Severe forms are characterized by burns of the mucosal lining of the URT and are experiencing by difficulty breathing due to edema of the throat. Tracheotomy is often required.

Chronic injuries often occur in the form of dryness, burning in the nose and throat, nasal bleeding, irritable cough, hoarse voice, etc.

Treatment. It is carried out in outpatient conditions for light forms with inhalations and gargles with alkaline solutions, analgesic drugs and antibiotics for inflammatory complications.

Severe forms require rapid removal from the harmful environment, cardiotonic and analgesic medications, oxygen passing, and hospitalization in the resuscitation and intensive care units.

Prevention. Includes sanitary-hygienic controls, personal protective equipment, professional selection, dispensary treatment of workers with chronic disabilities at the URT.

Diseases of URT from organic substances and compounds. Occupational harm to the chemical in-

[1] Some peculiarities are observed in the case of damage to mercury (mercury vapors) from the URT, so-called Cusmaul pharyngitis with copper red shine, accompanied by sinusitis and lymphadenitis.

dustry includes gasoline, benzene, phenol, acrylonitrile, ethylenediamine and others organic substances, often combined with variable microclimate, dust, noise, vibrations. Threatened professions are mainly fitters, repair workers, apparatus workers, and others.

Pathogenesis. Under current conditions, work in environments with high concentrations of toxic substances is rare (failures). The prolonged action of toxic substances in low concentrations is essential. Morphological changes are mainly localized in the lining of the lower nasal cavities and in the palate tonsils, initially affecting the epithelium of URT, later reacting the vessels in the subepithelial layer with subsequent destruction.

Clinical picture. Damage to the URT is non-specific. Complaints include dryness, burning, nose and throat irritation, feeling of a "foreign body" in the throat, hoarse voice. Often, inflammation of the URT is also developing - catarrhal and atrophic forms of chronic rhinitis, rarely pharyngitis and laryngitis.

The length of service determines the localization of the pathological process. Short-time workers have disabilities mostly on the nose, and with a prolonged period of service they are downward in defeats.

Treatment is predominantly topical. Inhalations are administered in catarrhal forms with alkaline-salt solutions, corticosteroids, in hypertrophic are preferred with sodium bicarbonate, and in atrophic - with iodine-glycerin solutions.

Prevention. It includes medical-prophylactic measures: professional selection of workers (excluding atrophic diseases of the URT, allergic diseases of the URT, curved nasal septum, etc.), periodic medical examinations.

Diseases of URT from overtension. Voice function has a leading role in the way of communication and as a tool of labour. Professions where voice is used as speech in unfavorable conditions of production (at elevated or reduced temperatures, dust, gases, excessive noise, etc.) are endangered. Professions where voice is the main tool of work (actors, teachers, etc.) and professions that have undergone special vocal training and used their singing voice are endangered.

Pathogenesis. Strong load makes it difficult for the vocal vibratory muscles, as fatigue occurs expressed, in the hypotonic state of the vocal muscles. At first, changes are an expression of easy voice tiredness due to functional disturbances of central origin or peripheral at the level of the loud musculature of the throat and are of a transient nature.

Changes were observed in laryngoscopy and laryngostroboscopy (an increase in the amplitude of movement of the vocal cords was observed - a hypotonic state). In the more advanced forms, objective morphological changes of the vocal cords are established in the form of permanent paretic states, "singing nodes", fibromas and others.

Permanent tissue changes primarily affect the edges of vocal cords by thickening their mucous membranes in certain places. The most recent neoplasms of this genus are the "noduli cantatorum".

Rarely, the fibroids affect the vascular system with the appearance of submucosal haemorrhage, which is resorbable and develops a rich vascular network and a conjunctival stroma (teleangiectatic form).

Clinical picture. Initial changes in the timbre and the density of the voice occur. It becomes hoarse and adds noises. As the process progresses, the changes become lasting and are objectified with the morphological changes of the vocal cords. The voice loses its power, becomes dysphonic to a different extent to aphonic. Breathing disturbances begin.

The objective manifestations of the developing hypotension are visualized in a laryngoscopic study. There is incomplete adduction of the vocal cords in the middle, followed by hypothrophytes of the vocal muscles with thinning at theirs rim, increased blood supply, resulting in redness and droplets of mucus.

The nodules develop usually on both sides, on a broad base, reaching in size to a bean, have an off-white color and locate between the front and the rear two-thirds along the edge of the vocal cords.

Fibroma develops unilaterally, on a foot or wide basis, reaches a pea, has a red color and a smooth surface and locates in the anterior half along the edge of the vocal cords.

Treatment. Initial stages of dysphonia require complete vocal rest for several days with subsequent orthophonic exercises, medication treatment with group B vitamins, atriphos, nivalin, novocain blockade in the Zhaharin-Hed zones for larynx, etc.

Nodules and fibromas are removed by microsurgery, and then a strict regiment of voice-rest and orthophonic exercise regime is required.

Prevention. An important role in the professions of singers, teachers, lawyers, speakers and oth. is the need for not voice overloading by intensity and duration of use, which requires the creation of a loading regime, the application of vocal technique, the use of amplifying equipment, etc.

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6 OCCUPATIONAL OVERTENSION DISEASES

6.1 DISEASES OF PERIPHERAL NERVOUS SYSTEM OVERTENSION

Peripheral nervous system (PNS) may be affected by a variety of pathological processes of different aetiology: infectious-allergic, toxic, metabolic, traumatic, vascular-ischemic, genetic insuffiently, blastomic, etc. More and more often in the pathogenesis of these diseases are some professional factors that may also have a leading role in the causal process - chronic systemic microtrauma, long-term overtension of the same kinematic chains, cold impact, forced working position, rhythm imposed, etc. Very often, the occupational disabilities of the PNS go along with the damage to the musculoskeletal system or are their effects.

Relatively more frequent occupational diseases of overtension, microtrauma and other factors are cervical radiculitis, brachial plexitis, neuritis of *n. medianus* and *n. ulnaris*, lumbosacral radiculitis, polyneuritis and polyneuropathies, including upper limb vegetative, primary form of shoulder-arm syndrome, angioneuro-dystonia, and angio-neurodystrophies.

In cervical radiculitis (ICD-X-54.2), the most common etiological factor is osteochondrosis. Relatively rarely, the cervical root is damaged by common infections (influenza, angina, etc.). A major pathogenetic role in osteochondrosis plays the mechanical factor as well as the occurrence of vascular-ischemic and allergic-inflammatory changes in the affected cervical roots.

The clinical picture of professional cervical radiculitis is not different from that of another aetiology. Only the gradual development of the disease and its frequent combination with forearm myositis and shoulder periarthritis could be noted. The determination of the causal link with the profession is justified by this peculiarity, as well as by the nature of the workload, the localization (pre-ilection) and the length of service. The presence of cervical osteochondrosis does not contradict the relationship between the diagnosed illness and the occupation if it is found in persons no older than 40-42 years (this is based on a number of studies proving that working conditions - physical load, working posture, significantly affect the incidence of osteochondrosis).

The cervical radiculitis with the symptoms of lower cervical and 1th thoracic root (C5-T1), forming the brachial plexus and upper limb nerves, are common in manual labour. Pain is a leading symptom in the clinical picture. They also appear at rest and movement, they have different widespreadness, strength and character. They are caused by paravertebral pressure in the supraclavicular fossa as well in the roots of the nerve stems. The symptoms of stretching are particularly remarkable this of *Remack*. There are also root-type sensory outcomes such as thumb hypesthesia (C6 dermatom), to 4th and 5th fingers (C8 dermatom) or between them (C7 dermatom). Heavier forms of cervical radiculitis also affect muscles. If the 5th and 6th cervix roats (Duchenne Erb type) are affected, weakness and hypotrophy occur in the muscles of the shoulder and arm. When 7th cervix root is affected, the muscles innervated by n. radialis are damaged, and C8-T1-root (type *Aran Duchenne*) - predominantly muscles innervated from the medial and ulnar nerve - mainly tenar, hypotenar and interoseous muscles. Periosteum and tendon reflexes become weaker. The course depends entirely on the causes of the disease - i.e. vertebral pathology associated with cervical radiculitis usually occurs more tightly, lasts weeks, even months and often recurves.

Brachial plexitis (ICD-X54.0) can be caused by severe physical labour, causing stretching or compression in the shoulder joint area. Endangered professions include - carriers, blacksmiths, persons working with low-frequency and strong backlash pneumatic appliances (miners, vibroconcretors, concretiers, stonecutters, woodcutters, etc.).

Clinical picture. The main sign is the severe pain

in the shoulder area and neck. It quickly develops and fails sensory and motor symptoms throughout the arm and shoulder belt - muscle weakness, pronounced hypesthesia, pain in palpitation and pression, vegetative symptoms (cyanosis or redness, swelling, cooling, sweat, etc.). Stretch tests are highly positive. Tendon and periosteum reflexes become weaker or disappear. Rapid and muscular atrophy develops. Running here also depends on the main etiopathogenetic factors and is sometimes quite delay - up to months.

Labour expertise. If there is only cervico-brachial split neuralgia, after 10-12 days of treatment, it can be translated into lighter work for 1-2 months. After that, work can be continued in the profession. Rational labour readjustment is imposed only in cases of recurrence. It is also mandatory in the case of cervical radiculitis and brachial plexitis. In such patients, re-employment must be preceded by long-term treatment. On rare occasions, there is a permanent partial loss of working capacity.

Differential diagnosis of cervical radiculitis and plexitis is done with cervical pain syndrome or cervicalgia due to irritation of the endings of the sinuvertebral nerve in the area of the fibrosis ring in the interdiscal hernia. And there is also pain in the neck, shoulder and scapular area, which often occurs with a sharp head movement and often spreads to the elbow and wrist. Unlike radiculitis, there is no sensory disorder. The myalgia, which is most common in the cervix-shoulder zone, is runed in root symptoms. It usually occurs with muscle tension and cold. Myositis (the chronic form of myalgia) is characterized by a recurrent course. The affected muscles are painful under pressure, and they often find a variety of hard and painful nodes. In the differential diagnosis, pseudostenocardial syndrome should also be considered, which is observed not only in the neck but also in the thoracic osteochondrosis. Clinically, it is manifested with complaints that are very reminiscent of angina pectoris - gradual or paroxysmal pains in the heart area or behind the sternum of sympathetic nature that continue for hours, and in some cases even days, and are not affected by the administration of nitroglycerin **(Table 1)**. Difficulties sometimes create the humerus-scapular periarthritis, which is also manifested with pain in the shoulder area, and often occurring all over the hand. Typical for him is the pain caused by the pressure on the tubercles of the shoulder bone, proc. coracoideus and the upper extrem-

Tab. 1.

Indicator / symptom	Shoulder-hand syndrome	Myocardial infarction	Cervical-thoracic radiculitis
Pain			
Localization	Shoulder, arm, shoulder,	heart zone	Chest, spine, shoulder
Irradiation	neck, head, heart area	Shoulder, arm	In the course of the C3, C6 roots
Begining	Gradually, very rarely sharply	gradually and sharply	Suddenly - after colds, physical over-intension
Character	Diffuse, protopatic	acute, sharp	Gradually - rarely Constant with periodic enhanced
Behavior of the patient	Calm, antalgic posture of the hand	Anxiety, fear, protective reflex posture, hand over heart zone	Limited movement in cervical-thoracic zone of spine
Pupils	Normal	Mydriasis left	normal
Disease reflexes	Absent	Weakened upper abdominal reflex left, pseudobulbar reflexes	At first lively, and later weakened arm reflexes
Sensory	Painful points above the upper shoulder and scapular angle	Hyperalgesia in the area of Saccharin-Head for heart	Painful points in the area of the roots, hyper- or hypoesthesia in the area of radiculitic pain
Arterial pressure	Unchanged	reduced	unchanged
Pulse	rhythmical	arrhythmia	rhythmical
Heartbeat	Normal tones, without noise	Deaf tones, sometimes faint sound of pericardial friction	Normal tones, without noise
EKG	No change	Typical of myocardial infarction changes	No changes
Blood	No change	Leukocytosis, changes in LDH, CPK, GGTP	No changes, in infectious radiculitis has leukocytosis
Urine	No change	Sometimes reflex urinary retention	No changes

ity of the trapezius muscle (radiography is of great importance for diagnosis). In case of persistent pain in the cervix, it is always necessary to think of cervical vertebrae metastasis, primary spinal cord or root tumor, tuberculous spondylitis, spinal arachnoiditis, etc. These diseases are usually preceded by a long period of progressive pain and paresthesia. Correct diagnosis is facilitated by X-ray examination. Root injuries also occur with traumatic lesions of the spine. Some congenital diseases of the spine in the neck area also have differential diagnostics (the diagnosis of these diseases is also above all radiological).

Lumbosarral radiculitis (ICD-H54.1). Like cervix, they are found in a large number of hard-working workers, but with a load in the lumbosacral region and lower limbs. Except for an accident at work, the main etiological factor is the microtrauma, which is two types: external-multiple weight lifting, small thrusts, stretching at a significant frequency, and internal-long-term muscle contracture imposed by the forced posture. A vertebral pathology with radicular manifestations is obtained. According to modern perception, the most common cause of lumbosacral radiculitis is the disc hernia - 70%, most commonly between L5-S1 vertebrae. Followed by discopathy, spondylosis, spondylarthritis, and colds. In pathogenesis, the anatomical peculiarity of the L5- root is particularly important, it is the thickest of all roots, but it passes through a narrow, intervertebral opening.

Clinical picture. The main symptom is the acute stiff pain, usually one-sided with irradiation to the seat and legs. Failure proceedings of motor, sensitivity and vegetative function subsequently occur in a slow run. They respond to the affected roots in the disc hernia often the L5-root. The stretching symptoms are positive, the painful pressure at the points of the Vale is strong. In the disc hernia vertebral syndrome with the symptom of the "button" is pronounced. In lumbosacral radiculitis as well as in the cervix, vertebral pathology does not always correspond to the clinical picture- at severe discopathy, a clinical picture may be missed, and in contrast - to mild spondylarthrosis, the pain may be strong, and along with failing neurological symptoms - as other factors, colds, focal infections, allergic reactions, etc., that doubtful the professional origin of the disease. The course of lumbosacral radiculitis usually lasts for several weeks. Only in severe forms of disc hernia leading to permanent waste sensory and motor changes with n. paresis of peroneus, pelvis-uretic disorders, etc., the disease lasts for months. In such cases, a permanent partial loss of working capacity is required. There are also single cases of occupational neuropathy of *n. peroneus* when working in a squatting or kneeling position (parquet workers, roof builders, vegetable growers).

Differential diagnosis. First, a distinction has to be made with the lumbar disc hernia, in the clinical picture of which the root symptomatology occupies a central position. It is characterized by an acute onset after abrupt movement, vertebral syndrome, monoradicular symptomatic, reduction of pain after physical rest and recurrent flow. Radiologically, total or partial downsizing of the affected disc, local osteophytes, and sealing of the disc-limiting surfaces of the adjacent vertebrae is often detected. In the so-called lumbago, it is also clinically manifested by pain syndrome arising after physical exercise (prolonged bending and straightening, weight lifting), but unlike radiculitis, there is no root symptom. Neuritis of the femoral nerve, in which the sensory disorders have no root, but peripheral-trunk nature on the anterior surface of the thigh and the inside of the lower leg. Neuralgia of the external skin-thigh nerve is characterized by the presence of numbness and hyperaesthesia with elements of hyperpathy in the upper two thirds of the fore-outer side of the thigh, which are enhanced by walking and prolonged standing. Ankylosing spondylitis (Bekhterev's disease) in its early stages is often with rooted pain and is difficult to recognize. The main clinical sign is the constant pain in the waist, which paroxysmaly intensified. The X-ray image, which represents the spine as a bamboo stick, is very characteristic. Distinction from coxitis and coxarthrosis is not difficult. Hip joint movements in them are very painful and limited, especially rotation and thigh removal. Coxitis may have elevated temperature and accelerated BSR. Radiography of the joint clarifies the diagnosis. Sacroileitis is characterized by spontaneous pain localized in the lumbosacral joint, most strongly manifested in the sitting position. Here too, radiography helps clarify the diagnosis. Certain diseases of the vertebrae (spondilites, cancer metastases, primary tumors) have to be taken into account - and here the x-ray is decisive. Congenital abnormalities (sacralisation, lumbarization, spina bifida, scoliosis, etc.) occur asymptomatically or occur with unspecified back pain and relatively weak root syndromes (they are also extremely radiologically recognized). In the differential diagnosis, vascular diseases of the lower limbs (thrombophlebitis, endarteritis, varices, intermittent atherosclerotic claudication, etc.) should be considered. In them the pain has no root, but more diffuse and often indefinable character. Reflected pain in the waist area in some surgical, internal and gynecological diseases (appendicitis, renal and ulcer disease, prostate adenoma, adnexitis, parametritis, post-operative scarring in the pelvis, etc.) should also be considered. And in them neurological status

is lacking in morbid deviations.

Compression mononeuritis

The most common form of mononeuritis is n. medianus neuropathy less frequently in *n.ulnaris*, and even more rarely in *n.radialis*. The professions in which these diseases are observed are very diverse. All of them are characterized by rapid and intense wrist movements, and often by pressure on the palms.

Carpal tunnel syndrome (ICD-X-56.0). Pressure of *n.medianus* in the carpal canal may be due to trauma, edema, fracture, rheumatoid arthritis, tendinitis, etc. The clinically manifested manifestation is the atrophy of the thenar. Anesthesia of *n. medianus* innervated fingers is often observed, which is initially intermittent in contrast to anesthesia in cubital canal syndrome. There are severe second and third finger pains, sometimes hyperaesthesia in the *n.medianus* area. In atypical cases there may be pains throughout the wrist, which include the forearms, night paresthesias (pathognomonic signs characteristic of the early morning hours) and hyperhidrosis of the palms. Usually the pain does not reach the shoulder joint.

Pronator syndrome (ICD-X-56.1). *N.medianus* can sometimes be squeezed into the *m.pronator teres* area and as a result form the clinical picture of the pronator syndrome. His knowledge is important for the differential diagnosis of carpal tunnel syndrome. The similarity between the two syndromes is due to common signs: anesthesia and paraesthesia in the fingers innervated by *n. medianus*, weakness, and later tenar musculature hypothrophy, wrist and forearm pain, microcirculation discrete disturbances with sympatheticotonic overload. As a difference, it is noted that in the pronator syndrome the Phallen test and reversal test are negative, there may be a delayed neural conduction that does not affect the wrist. The treatment here is very often operational.

Supinator Syndrome (ICD-X-56.3). The motor branch of the radial nerve-*r.profundus n. radialis* innervate the wrist and finger expanders except for *m. ext. carpi radialis* and *m. branchioroidis*. His lesion (supinator syndrome) leads to a partially hanging hand or to a paralysis of *n. radialis* without a hanging arm. The passage of *r. profundus* through *m. supinator* allows in rare cases to be considered as a site of injury, resulting in a purely motional paresis of *n. radialis*. As a rule, progressive paralysis of the innervation of the hand and fingers, outstanding in the foreground to the symptomatic (the paresis of *n. radialis* without a hanging arm). The paresis are accompanied by pain in the proximal third of the forearm, due to overload/overtension, which can be emitted by *dorsum manus*. Movement in the elbow increases the complaints. Etiological can be considered: tumors, chronic mechanical damage from overtension in certain professions (pianists, tennis players, etc.), after injections in *m. brachioradialis*, fracture of the ulna with dislocation of the head radial ahead. In a radial epicondylitis, a supinator syndrome may develop.

The diagnosis can be supported electromiographically. Due to the difficulty of localizing the pain, the supinator syndrome may be mistaken with a number of other pain syndromes in this area, even sometimes with psychogenic pain complaints in the same area. Treatment is primarily operative.

Ulnar canal syndrome (ICD-X-56.2). *N.ulnaris's* compression is much less common than *n.medianus's* compression. It is most commonly associated with traumatic genesis or the presence of abnormal structures such as ganglion or muscle abnormalities that narrow the *Guyion's* canal.

Clinically, sensory and motor disorders are observed. Sensory disturbances can be expressed to varying degrees and cover the small toe and the ulnar half of fourth finger. Motor disorders predominantly include hypopothenaric muscles and are expressed as muscle weakness, sometimes as paralysis.

Cubital canal syndrome (ICD-X-56.2). Different mechanisms can chronically damage *n.ulnaris* as it passes through the cubital area by squeezing or friction - anatomic hand features, direct or indirect trauma, periodic nerve shifts in hand movement, swelling, muscle abnormalities. The pain in the clinical picture is dominated, which can cover the entire upper limb, both distal and proximal - there are paresthesia and anesthesia of the fingers involved. Unlike ulnar canal syndrome, anesthesia and sting in fingers are progressive and continuous. Sensory disturbances in cubital canal syndrome include, in addition to the fingers, hypothenar and ulnar half of *dorsum manus*, as the latter symptom may be decisive for the rejection of ulnar canal syndrome in differential diagnosis.

Radial canal syndrome (ICD-X-56.3). The emergence of the radial canal syndrome, unlike the other syndromes described above, can only be associated with some local anatomical abnormalities and alterations that compress *n.radialis*. As a trigger factor on this basis, over-pressure in the working conditions and more precisely some chronic repetitive, stereotypical movements of the wrist and forearm, can importance. In the clinical picture, the most common symptom is the pain that extends to the extensor elbow area and can be radiated to the wrist and *dorsum manus*. It has a troublesome, painful character and is exacerbated by certain movements - wrist flexion and ankle pronation.

Shoulder-arm syndrome (ICD-X-M89.0).

Threatened occupations: textiles, carpets, typewriters, drivers, spinners, hard labour workers, bookbinders, printers with intensive loading of the kinematic chain arm-shoulder and chronic high frequency traumatism. It is most common between the ages of 35 and 45 (the most active period in the volume and intensity of the workload). A significant proportion of women almost 1: 3 (male: female) suggests a possible role of hormone-endocrine factors (preclimax and climax).

The clinical picture shows a different degree of disease alteration. Initial manifestations may be slightly pronounced - paraesthesias and a vague feeling of discomfort in the shoulder. There is objectively palpated soreness in the area of the shoulder and scapula, particularly the pressure of the *incisura scapulae*. Gradually overwhelmed pains and dysfunction in the shoulder, as the pains are of a typical vegetative nature - diffuse, undefined localization, protopathic, with broad irradiation in the direction of the hand distally and to the scapular area, neck, head. Typical is their appearance in the second half of the night. There are paraesthesias, and sometimes anesthesia, mostly on the fingers. Circulatory and trophic disorders are expressed in swellings, skin moistening, color changes such as paleness, flushing or lividity, sweaty palms. Later, muscle hypotrophy and contracture of the forearm flexors and consequent long-term contractions of the fingers, reflect the severe nerve-vegetative disorders.

Characteristic of the course of the disease process is its **phasic nature**. The first stage may have an acute onset with marked prevalence of pain or develop gradually for 2 to 6 months with painful dysfunction of the shoulder and widening swelling with stiffness of the hand and fingers. The next, second stage also lasts between three and six months and can be seen as a backward development because the pain and dysfunction in the shoulder area is noticeably weaker, and the swelling of the hand is noticeably dropping (but improvement is apparent). With the entry into the third stage, the trophic disorders in the area of the arm stand out. Limited flexion and stiffness of the fingers persist, and in some patients there are muscle contractions of the flexors and consequent long-term contractions of the fingers, which sometimes resemble the Dupuytren's contracture. There is a decrease in skin temperature and changes in the color of the swollen tissues - sometimes to paleness, and in other cases to the lividity. Atrophies are associated with differently expressed retraction of the subcutaneous tissue and the muscles of the hand and fingers.

The recovery and rescue processes are protracted and can last from 3 to 12 months. Full functional hand restoration is sometimes seen in about 2 years.

Differential diagnosis of shoulder-arm syndrome with professional genesis should be made with medication-toxic clinical forms caused by acute or chronic chemical intoxication in drug treatment, e.g. tuberculosis, epilepsy, and the like (isoniazid, phenobarbital). Cases of shoulder-arm syndrome in acute vascular disease with hemiplegia have been reported.

Treatment. In severe cases with pronounced pains, admission may be required until they are taken.

Medicinal treatment - in the first stage with acute or sub-chronic course of manifest sympathicotonic phenomena, capillary spasm, rarely and differently expressed inflammatory effects, may have vasoactive and rheology-improving drugs: *Radecol, Ronicol, Dusodril, Pentoxyphillin, Complamin, Tiklid and others*. For the sake of sympatholytic action, vegeotropic agents and vegetative harmonizers such as *Bellergamin, Bellergal, Vegezin, Ipronal in combination with Diazepam, Seduxen, Rudotel, Tranxene, Xanax*, are most commonly used.

Vitaminotherapy is widely used. The combination of vit. A with vit. E (in the *Geritamin* preparation), vitamins B_1, B_2, B_{12} (as well as the newer preparations - *Milgamma, MilgammaN, Thiogamma, Benfogamma*). The usual analgesics - *Analgin, Aspirin, Paracetamol and combined preparations - Benalgin, Tempalgin, Saridon*, are included in the therapy. Due to the metabolic disturbances and the possibility of osteoporosis changes in chronic development, anabolic and calcium preparations are applied.

In the treatment plan, anti-inflammatory drugs can also be taken with the NSAIDs - *Indomethacin, Sulinac (Clinoril), DiclofenacNa (Voltaren, Feloran), DiclofenacK (Cataflam, Aulin, Niflural*, etc.) Neurological therapy - the use of *Novocain, Novoplex, Lidocain* intramuscularly, intradermally or in layering.

6.2 VEGETATIVE POLYNEUROPATHY FROM OVERINTENSION OR MICROTRAUMATISM (ICD-X-90.0)

In general, polyneuropathy combines diseases of the peripheral nervous system of a dystrophic nature caused by exogenous or endogenous factors (infections, toxic, malignant, dysmetabolic, genetics. Multiple damages to the peripheral nerves (predominantly to their distal parts), where there is no evidence of an inflammatory process, are expected. However, their difficult differentiation from inflammatory polyneuropathies has given some authors reason to speak of vegetative polyneuritis.

The vegetative pathology of the upper extremities of overtension encompasses a group of diseases affecting the extreme vegetative periphery and, most importantly, the neurovascular network of the wrists. Depending on the preferential involvement of the peripheral structures, they are divided into neurovasal and neurotruncal.

Etiology. *Neurovegetative lesions of overtension are observed in professions mainly with a uniform manual work requiring rapid and frequent movements at physical overtension. Professionally threatened are most often bookbinders, cardboard workers, milkers, carpet-weavers, knitters, ironers, polishers, grinders, as well as stenographers, telegraphists, typists, pianists, violinists. For the development of the disease, the irrational organization of labour, the inappropriate physiological amplitude, the disturbed optimum of working movements, the static and dynamic overtension and especially the increased traumatisation of the peripheral neurovegetative and vascular substrates play a significant role.*

Pathogenesis. *Permanent mechanical microtrauma lead to recurrent microreactions of neuro-vascular net - vasospasms or vasodilatation due to blood flow impulses on the vascular wall. Consequences of these are abnormal vascular reactions, pathological axon reflexes, chain-amplifying, permeability disorders, swelling, tissue dystrophy. The ultimate result is the deformation of the capillary wall, the changes in the capillary net, well perceptible by capillaryscopy and capillarygraphy.*

Clinical picture. Subjective syndrome occurs with paraesthesias (thirst, tingling), pain, sweating, coldness, swelling of the fingers and wrists.

Objective finding:

- vegetative and vascular changes, hyperhidrosis, change in skin color (lacy pattern, marbled), puffiness of palm and fingers;
- reduced sensation of touch and distal (polyneuritic) type pain for upper limbs;
- trophic disorders of the nails and the skin of the palms and fingers.

Objectivity is done by capillaryscopy or resp. capillarygraphy, skin thermometry, cold test with initial temperature recovery, dynamometry, white spot test (Lanyell-Lawastin), distal doppler flowmetry, X-ray of wrist's joints and fingers, electromyography, cold stress test, polarization current study, investigation of intensity of pain and more.

Stages of the disease. The mild degree is characterized by mild paresthesia pain, morning swelling and stiffness of the fingers, humidity of the palms, objective signs of angiospastic syndrome of a transitory nature.

The middle stage is characterized by pronounced and persistent symptoms. Paresthesias and pains intensify, swelling and hyperhidrosis become visible, the lace pattern is pronounced. They appear and attacks from "pale" fingers. Muscle strength is reduced, distally increasing hypoesthesia is convincing.

Severe stage is characterized by worsened, permanent subjective complaints. To the objective finding, talking about predominantly anthony or even vasoparesis, trophic disturbances are added. The attacks of "dead" fingers increase.

In the **differential diagnosis**, the other manifestations of vegetative disabilities in occupational disease - vibration disease, cold polyneuropathies and professional toxic factors (lead, vinyl chloride, mercury, organic compounds, etc.) must be taken into account at first. The consequences of a number of systemic disorders (collagenosis, dysmetabolic polyneuropathies, endocrinopathies, atherosclerosis, etc.) should be distinguished. It is known that the vegetative nervous system can be damaged in all forms of collagenosis, but it is most often afflicted with nodular periarteritis, lupus and dermatomyositis, and rarely with scleroderma, rheumatoid arthritis and true rheumatism. Dysmetabolic polyneuropathies that occur as a result of impaired metabolism in some somatic diseases: diabetes, renal and hepatic failure, endocrine disorders, etc., should also be considered. Apart from the endogenous toxic factor in the pathogenesis of some dysmetabolic polyneuropathies (diabetes, liver), B_1 vitamin deficiency is attributed, and in others (amyloid, porphyrin, etc.) - genetic factors with a clear or unspecified metabolic

defect. Alcoholic polyneuropathy is of particular interest due to its marked increase in recent years due to the widespread prevalence of alcohol abuse. In pathogenesis, an important role plays the deficit of (vitamin B_1, B_6 and PP) as well as impaired absorption of proteins and carbohydrates due to gastrointestinal lesions. As polyneuropathies, the early stenosis processes of the vessels (atherosclerosis, obliterating endarteritis) can also be manifested.

Epicondylosis, dystrophic changes in the spine (spondylosis, spondylarthrosis and osteochondrosis) also give vegetative disorders in the upper limbs. Polyneuropathies may be confused with cases of acrocyanosis and erythropelalgia.

Treatment. In the early stages of spasticity, vasodilators: *vasolat, nicotinic acid, papaverine, and in some more persistent cases - dillinal or dusodryl* are used. In the dilation phase ephedrine, vasotone is used. Against permeability disorders, *routascorbine, calcium, combined with vitamin C, peflavit* is administered. In all phases, B vitamins are indicated. The *Dibazole* is used as a neuronal stimulant, especially in the vasoparalytic phase. Novocaine drugs are used as trophotropic drugs. Physiotherapy includes kinesitherapy, electrotherapy (ionization and galvanization, ionophoresis with iodine, calcium, novocaine), ultraviolet irradiation, diathermy, hydrotherapy, massage, gymnastics, passive movements to activate peripheral circulation.

Employability.

Slight degree - the ability to work is preserved. If necessary, temporary disability up to 20 days.

Middle degree - temporary disability up to 30 days, followed by 6 months of retraining or requalification (in individual cases - disability group).

Severe degree - compulsory reemployment. Retirement from work experience (disability group in the absence of such possibility).

6.3 DISEASES OF OVERTENSION OF THE MUSCULOSKELETAL SYSTEM

In the group of occupational diseases of overtension, the nosological forms related to the defection of various parts of the musculoskeletal system (MSS) are significant.

The prevalence of musculoskeletal pathology is related to the degree of use of systematic manual labour in a number of manufacturing industries, characterized by high intensity, numerous, frequent and monotonous recurrent upper limb movements, conveyor-type rhythm, static overtension of the kinematic chain hand-arm.

These overtension factors, however, are conditional nature, individual reactivity is important.

Numerous studies of researchers not only in our country are dedicated to musculoskeletal disorders of overtension with a characteristic tendency of lack of specific nosological interpretation of most of these disorders, but instead are used generalized concepts such as RSI *(repetitive strain injury)*, *overuse syndrome, cummulative trauma disorder,* occupational cervicobrachial disorder, and others.

Workers in many professions and industries are especially vulnerable to the development of such disabilities: washing machines, ironing machines, packers, labels, boxes, typists, bookbinders, presses, carpets, pianists, violins, production counters, hand weavers, stampers, telegraphs, keyboards, draftsmen, hand-made cigarette packs, shoemakers, conveyor-makers, masonry makers, stoners, miners, spinners, hand knitters, grinders, hand-made polishers, painters, cutters, loaders, digger, crane-operators and others.

Spondylosis, osteochondrosis (ICD-X-M43.C also - M47.8). Degeneration of intervertebral discs is a result of age-related changes and external overloads. *Nucl. Pulposus* loses its elasticity, reduces its volume, affects the intervertebral joints and narrows the intervertebral spaces. Spondylosis develops as a result of a local degenerative process of the outer layers of the fibrosis ring. Spondylarthrosis is the most common in the cervical and lumbar segment.

Clinical picture. Degenerative changes in the spine may persist asymptomatically or may be manifested clinically with various manifestations and with periods of remission and exacerbation.

Lumbar osteochondrosis is common, chronic, with lumbago, lumboischialgia, and ischioneuralgia. The lower spine is subjected to heavy loads and volume of movements. Sharp movements, lifting weights favor the exacerbation of radiculopathy. People at risk such as hard labour workers such as dockers, loaders, carriers, metallurgists, rollers, excavators and exposed to general vibration. The effect of physical overload on structures rich in receptor terminations results in the occurrence of reflex syndromes. As a result, reflex muscular tension, reflex vasomotor or vegeotrophic disorders, persistent diffuse pain occurs. The clinical picture can also be seen

in the image of a discal hernia with root syndrome depending on the level of damage most common at L4, L5 and L5 - S1, due to the narrower space of the latter, the thicker roots emerging at corresponding holes and the increased load on this segment. Diagnosis is placed radiographically with radiograms in profile, front and oblique projections, the paraclinic shows deviations from the norm.

Professionally-determined degenerative changes in the spine have a gradual onset, occur with periods of remission, and exacerbations are associated with overtension, and radiological signs are early - they usually occur before the age of 40. They need to be distinguished from some other groups of degenerative diseases - congenital dysplastic malformations, affecting the disc under the influence of other causes - hernia, Schmorly corpuscles, *M.Baastrup*, inflammatory diseases of the spine, bone-tumors, metastases, *M .Paget* and others.

Treatment is primarily rest, thermal procedures, careful massage, non-steroidal anti-inflammatory drugs and local Lidocaine therapy - neural therapy. **Expertise** - The patient has to break away from work for at least 2 weeks or 1 month during treatment, then it is appropriate to be labour-readjustment at work with no overtension and forced posture for several months. The lifestyle is related to the regular healing physical drill (HPD), physiotherapy and massage.

Arthrosis and periarthritis (ICD-X-M16 and M18). It is the most common chronic disease of the musculoskeletal system. Normal degenerative changes in joints expressed in varying degrees in the elderly. In some professions, however, their earlier appearance and wear of joints are characteristic due to chronic microtrauma and overtension. Risk contingents for this are dockers, loaders, miners, stonemasons, blacksmiths. Deadlines for wear of cartilage and appear clinical signs of arthrosis vary widely depending on the degree of mechanical discrepancy - from several months to several years. This is important in the differential diagnosis of professionally determined arthrosis of other types of arthrosis and age-related changes. Professional arthrosis usually affects the congested joint, for example, gonarthrosis in parketters and mosaics; omarthrosis and arthrosis of the elbow joints of concretors, turners, miners, interphalangeal arthrosis of knitters, milkers, pressers, miners, etc. Age arthrosis usually occurs after the age of 40 slowly, gradually with moderate degenerative joint changes affecting more joints - polyarthrosis. Distinction from inflammatory diseases of the joints - arthritis. Differential diagnosis is also made with some out-joints illnesses such as radiculitis, periarthritis, etc.

Treatment and labour expertise. Treatment with non-steroidal anti-inflammatory drugs and physiotherapy is carried out, after which it is appropriate to work without loading the affected joint.

Periararthitis. Humeroscapular periarthritis (ICD-X-M75.0) is a collective concept of a number of diseases in the area of the shoulder joint - including capsular, insertion, tendon stenosis muscular lesions, lesions of periarticular bursae - deltoid bursitis, calcural bursitis, subacromial bursae. Each form has its own topographical and pathological anatomical features, but similar clinical picture and disorder of hand function.

Risk contingents are operating under conditions of prolonged muscular tension on the shoulder girdle and increased volume of movements in the shoulder joint area - endangered occupations include weavers, blacksmiths, fitters, turners, shipbuilders. Clinically, it is manifested with a gradual onset, soreness in the shoulder, strengthening with rotation in the shoulder joint, lifting of the arm above the horizontal level, with irradiation of the pain to the neck and the scapula. Some movements, especially internal rotation and retroposition, are limited, while the pendulum movement back and onward along the body is preserved. In advanced cases, the limitation of movement is greater to the extent of complete immobility in the joint, and this favors the development of hypothrophies in adjacent muscles, bone osteoporosis, trophic disorders.

Diagnosis is determined clinically and radiographically. Radiographic features are common in these cases calcium deposits in the subacromial and subdeltoidal bursa and other periarticular tissues, destruction and sclerosis of the large humeral tuber (periarthritis, however, may be clinically manifested and without radiological features).

Treatment. Suitable HPD, physiotherapy and non-steroidal anti-inflammatory drugs. Appropriate rehabilitation is due to the tendency of periarthritis to chronic course and exacerbation.

Tendomyositis (myo-tendinosis) (ICD-X-M 60.8). The damage is in the muscle-tendon transition, in the middle third of the forearm, most often affects the hand extensors, among the occupational disabilities of the MSS is the highest incidence - in our country 58% of all diseases of the MSS for the period 1986-1988.

Risk contingent - hand knitters, fitters, seamstresses, typists, locksmiths, turners, bricklayers and others.

Clinically, it is manifested with constant soreness in the forearm, chronic pain syndrome, increased pain in active movements, or palpatory pressure in the area of the muscle-tendon connection, swelling, increased muscular consistency, increased density

and crepitations. Diagnosis is based on the clinical picture and EMG data for damage to affected muscles. Differential diagnosis - with conditions associated with myalgia and muscle weakness, e.g. systemic diseases of collagen and connective tissue - most often polymyositis, rheumatoid polyarthritis, rheumatic polymylagia, menopausal arthritis, some neurological diseases - peripheral nerve lesions, compression syndromes (supinatorial and pronatorial), cervico-arthrosis with root irritation, Pancoast- Tobias syndrome, Parsonage-Turner syndrome, muscular dystrophies, damage to certain drugs, and local muscular lesions associated with frequent healing manipulations (intramuscular injections).

Myositis (ICD-X-M60.8, M61.0). This group includes inflammatory processes in the muscle interstitium and is manifested by exudation, cell proliferation and sclerotic alterations. They are associated with unfavorable work and chronic microtrauma. The most common changes are in the shoulder muscles, the flexors and the extensors of the wrist and fingers. With more marked changes, seals of varying degrees and sizes, mostly with oval shape - so-called myogeloses, are formed (some authors separate them as a self-esteem disease - myositis ossifying). Myositis also occurs in athletes. The clinical picture of myositis is dominated by pain symptom.

Tendovaginitis, insertionitis. Dystrophic and inflammatory diseases and tendon processes, outside of the transition to the muscle or the insertion part, are manifested with extensive clinical symptoms. From the etiological point of view, particular attention is paid to the effects of traumas and microtraumatism in working conditions.

The disease of de Quervain (MKB-X-M65.4, M70.9) was described in 1885 and means still stenotic ligamentitis, styloiditis, chronic tendovaginitis. Disease is etiologically associated with prolonged microtrauma, combined with overtension, and is often seen in tailors, pianists and other occupations. Clinically characterized by slow, gradual onset, spontaneous pain in the distal part of the forearm and the area of *proc. styloideus radii* emitted to the toe. The trigger point is *proc. styloideus radii*, and the movements that cause pain disturbances are thumb abduction and ulnar abduction of the hand in the wrist. The grasping ability of the hand with the thumb is disturbed.

Treatment. In case of failure of conservative treatment and physiotherapy, surgery is usually performed with good effect.

Epicondylitis (ICD-X-M77.1). The most common diseases in the elbow joint region are the radial and ulnar epicondylitis.

Radial epicondylitis - the lesions are expressed in single or multiple tears at the locus of the extensor muscles of the forearm, caused by forced forearm movements, mainly supination and pronation, as well as when the extensor muscles are placed in a tense position. It is also often seen in tennis players ("tennis elbow"). It is clinically manifested with pain in the area of the lateral epicondyle, radiating from the dorsal side of the forearm. Here are some functional tests to prove weakness in the extensor muscles of the forearm - a Thompson test, a chair lift test, or a telephone (practiced by French authors), where sudden pain occurs in the lateral epicondyle. The illness may resolve for 2 weeks in mild cases, but sometimes the symptoms may last up to 2 years or more. Conservative treatment is with novocain local blockades, physiotherapy, massage of affected muscles, immobilization in gypsum dressing for about 6 weeks.

Aseptic necrosis (ICD-X-M93.1). Among the professional osteonecrosis, the most common are those of some of the bones of the wrist - os lunatumand os scaphoideum (naviculare).

Aseptic necrosis of *os lunatum, M. Kienbök-Preiser, lunatomalacia*.

Risk contingent - turners, miners, woodworkers, crane workers, working with vinyl chloride (toxic damage), i.e. at work primarily associated with a load in the *os lunatum* area of the wrist resulting in pathological changes. There is a breakdown in blood supply and bone nutrition and dystrophic changes therein. Clinically, it develops with moderate to severe pain in the wrist joint, increasing in motion and pressure in the area of os *lunatum*. Often, active and passive volar and dorsal flexion are limited. Radiologically, aseptic necrosis of the os *lunatum* is established - a focal point of enlightenment and bone sealing. Later, deforming arthrosis may develop in the carpal bone area.

Treatment. Prolonged immobilization - several months in gypsum bandage to the elbow joint, covering all fingers, physiotherapy, vitaminotherapy. Change of work - work without load on the wrist.

Aseptic necrosis of os *naviculare*. It is less common than lunatomalacia, usually associated with hard work and frequent movements in the radial carpal-joint, vibration - grinders, polishers, builders. It occurs with piercing pains, often at night, which initially occur gradually, but after about 2-6 months they become strong and permanent, with swelling in the joint, sometimes with deformity. X-rays are similar to lunatomalacia.

It is necessary to distinguish them from the primary traumatic fracture of os naviculare - the fracture does not cover all the bones, and there is a formation of adjacent pseudarthrosis. Treatment and rehabilitation - as in lunatomalacia.

6.4 COORDINATION DYSKINESIAS

Coordinator dyskinesias are diseases with basic syndrome motor disorders, selectively affecting only specific professional movements requiring precision and control that become awkward or impossible.

The etiology of coordinate dyskinesias is not fully understood as professionally determined diseases in most cases are etiologically associated with overtension of certain kinematic chains committed to performing rapid, repetitive movements with great precision and fine coordination, performing rapid and accurate manipulations of workers in the case of shortage and increased responsibility. Threatened occupations include handwriting employees, secretaries, stenographers, keyboards, teletypists, telegraphers, musicians, singers, dancers, handheld counters, milkers and knitters, drivers and others.

The most common professional coordinated dyskinesia is graphospasm, **writer's cramp**. "Clerk's cramp" are a type of focal dystonia and are included in the group of focal dystonias along with blepharospasm, oromandibular dystonia, torticollis and spastic dysphonia. As a relative share they are more than half of all forms of focal dystonia. Affect men and women equally often and develop between 30-40 years of age. Combinations between some of them, e.g. torticollis with tremor or with clerk's spasm or graphospasm with tremor.

Pathophysiological mechanisms are unclear. Although a known hereditary predisposition is suspected, the current understanding of these conditions is that they are a form of focal dystonia associated with task-specific dystonia. No organic brain damage is detected, but cortical neurodynamics is impaired - there is a pathological afferentation and efferentation from a modified motoric zone of the cerebral cortex that selectively engages the kinematic chain of the working hand, the dynamic stereotype is disturbed. Typical relapse of stress and improvement during silence and relaxation, also meaning have psychotrauma. Electrophysiological studies show abnormally prolonged activity of the muscle fibers involved in writing, but also increased activity of adjacent muscles not included directly in the task, as well as a change in the EEG amplitude of the record of the motor cortex zone, corresponding to the affected hand.

Clinical picture. The illness was first described in 1831 for long-term clerk workers. At first there is awkwardness, weight in the hand when performing precise movements, change in handwriting - the letters become distorted and illegible. Writing becomes painful, with pain and peculiar posture on the fingers of the hand - crushing the pencil strongly in a typical way. Occasionally, rising the tonic stress of the muscles of the 1-3 fingers of the hand and the perronator muscles of the wrist, sometimes to the athetosis-like cramps of the fingers, which can not be overcome voluntarily. Similar spastic forms of focal dystonia with other localization have been observed, though less frequently, in the occupational overload of other muscles or kinematic chains - eg laryngospasm with aphonia, spasm of m. orbicularis oculi in clockmakers, cramps of m. triceps surae in drivers and others.

Despite the violations of the precise specific movements that are characteristic of a professional activity, the other hand movements are retained. Disturbances, however, are observed in other types of activity, eg. related households and self-care that are similar in character, accuracy and precision to injured professional movements - holding a sewing needle and sewing, shaving with a razor, serving a spoon next to the mouth, knitting a knot, winding a clock, etc.

Diagnosis is based on the clinical picture, the absence of another organic neurological disease that could be the cause of the disorder, the data from the occupational program for prolonged work experience, and the performance of work related to systemic microtrauma and overtension of certain muscle groups. In an electromyographic study, the bioelectric activity driven by wrist flexors and extensors during writing shows a bursting of bursts at a frequency of about 5-8 Hz and alternation in the two muscle groups. Similar results are susceptible to the extra-pyramidal nature of the injury.

Differential diagnosis is made with other diseases of the nervous system and muscles - diseases of the peripheral nervous system (polyneuritis, radiculitis). In some other occupational diseases of the musculoskeletal system such as myalgias, myositis, epicondylites, there is also stiffness and awkwardness in the fingers of the hands (but there is also swelling in the wrist joint, crevices, radiographic evidence of epicondilitis). Chronic mercury poisoning, in which there is a fine, small tremor of the fingers,

can be misleading in its early stages, but the tremor also includes eyelids, tongue, lips, and the mercury test in biological fluids is crucial. Differential diagnosis should be made with organic lesions of the strio-pallidar system - torsion dystonia, hepatolenicular degeneration. Parkinsonism, essential tremor, cerebellar ataxia, etc., also with chronic mercury poisoning (fine tremor).

Treatment and rehabilitation. The complex approach is the removal of the etiological factor and, at the same time, the use of analgesic and sedative medication, appropriate relaxing acupuncture and physiotherapy, as well as various techniques of biological feedback by means of EMGmethodics. Diazepam, clonazepam, anticholinergic agents are used to treat spasms and pain. The prospective treatment with botulinum toxin A injected into the affected muscles is about 12 weeks effect. Appropriate recruitment and in severe clinical cases determine the degree of disability, as patients are fit for almost any other type of work.

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7 PROFESSIONAL INFECTIOUS AND PARASITIC DISEASES

Iv. Dikov

7.1 BACTERIAL AND VIRAL INFECTIOUS DISEASES

I. Bacterial Infectious Diseases

ANTHRAX

Anthrax is an acute infectious disease occurring most often as skin, more rarely as intestinal or pulmonary forms.

Etiology: Bacillus anthracis - spore-forming aerobic bacteria.

Epidemiology: Typical zoonosis - source are sick animals. One is infected with direct contact with them, more rarely when consuming meat from diseased animals or inhaling dust containing anthrax spores. This determines the occupational risk: farm workers - livestock breeders, shepherds, veterinary surgeons, zootechnics and processing wool, products of animal origin (bones, horns, hooves, etc.).

Pathogenesis: serous- haemorrhagic and necrotic inflammation develops at the site of penetration of the agent. Role is also the endotoxin.

Clinical picture: Incubation period 2 to 8-12 days. The most common is the skin form with the formation of a typical pustule with bluish-brown contents and subsequent central necrosis. There may be general toxo-infectious manifestations. The pulmonary form develops like severe haemorrhagic pneumonia with acute respiratory failure and the intestine as a severe haemorrhagic destructive enterocolitis.

Diagnosis is based on epidemiological data, occupation, clinical data, and is confirmed by the b. anthracis isolation of pustule, sputum, feces and blood. The skin form must be distinguished from staphyloderma (furunculus). The prognosis is serious, especially in the intestinal and pulmonary forms.

Treatment: compulsory isolation in the infectious ward. Etiological antibiotics of I order: penicillin - at a dose of 4 to 12 IU/day for a period of 7-10 days. Alternative preparations: cephalosporins of II-III p. Heterologous anti-anthrax antibacterial serum - 50-100 cm^3, by the Bezred method.

Prevention: with a spore vaccine JTI applied to risk contingents - veterinarians, zootechnics, etc.

BRUCELLOSIS

Acute infectious disease with polyorgan damage and tendency to protracted and chronic flow.

Etiology: Human pathogenic Brucella bacteria: Brucella melitensis, Br. abortus suis, Br. abortus bovis.

Epidemiology: zoonosis - the human infection is in contact with sick animals or the consumption of raw milk from them. Professionalism has the disease for agricultural workers, livestock farmers, meat industry workers, veterinarians and technicians. Laboratory infections are also possible.

Pathogenesis: after the initial phase of multiplication in the monocyte macrophage system, haematogenic dissemination develops with polyorganic damage mainly to the CNS, the locomotory system, the lymph nodes and the heart.

Clinical picture: Incubation period of 7 to 21 days. Toxico-infectious syndrome with an undulation temperature curve, generalized lymphonodulopathy, hepatosplenomegaly, marked tendency to protracted and chronic course. Common meningitis, polyneuritis, arthritis, endomyocarditis and other organ manifestations.

The diagnosis is based on epidemiological and clinical data and is confirmed by serological methods - the Wright and Huddlesson agglutination reaction. The isolation of the causative agent is possible but more difficult.

Prognosis - serious, especially in chronic form.

Treatment: hospitalization, bed rest. Etiological treatment with antibiotics - from I order: Doxycycline - 2 x 100 mg. oral, or Tetracycline - 3 x 100 mg. muscle or Tetraolean 3 x 100 mg/day for 10-12 days. It is good to combine with aminoglycosides: Streptomycin, or Chloramphenicol.

Several such healing courses should be conducted.

Prophylaxis: specific - with a live vaccine that has not yet been administered to humans.

ERYSIPELOID

An acute infectious disease with specific skin changes.

Etiology: Erysipelothrix rhusiopathiae suis.

Epidemiology: zoonosis with source of diseased animals - pigs. One is infected with direct contact with the diseased animal. Occupational risk is the pig-tenders, veterinarians and zootechnics. Contaminants and contact with fish are described, which may cause illness and work in fish processing plants.

Pathogenesis: In the place of the front door - usually the skin of the fingers develops a serous, rarely pungent inflammatory process. Hematogenic dissemination is rare.

Clinical picture: Incubation period of 1-3-7 days. Acute onset with a common toxigenic syndrome, edema and severe hyperaemia of a restricted skin spot, most commonly on the fingers. Pain expressed. The septic form is rare.

Diagnosis is based on epidemiological and clinical data.

Differential diagnosis with erysipelas, staphylococcus or streptoderma.

Prognosis - good. More serious - in septic form or rare complications such as endomyocarditis.

Treatment: can also be done at home. Etiological treatment with antibiotics - from I order: Penicillin - from 1 to 4 IU/day, for a period of 5-7 days. Alternative preparations: erythromycin or tetracyclines or chloramphenicol. Local treatment with compresses with Vishnevski ointment or rivanol solution.

Prevention: no specific. Personal hygiene and use of gloves when handling fish and meat.

LEPTOSPIROSES

Acute infectious diseases with general intoxication and polyorganic damage.

Etiology: Leptospirosis of fam. spirochetes.

Epidemiology: zoonoses. Source are animals and the mouse rodents. One is infected with bathing, wading in ponds contaminated with excreta of animals and rodents. Rare infection can also occur through conjunctivae. There are diseases among farmers, livestock breeders, slaughterhouse workers, excavators, miners and other underground workers, as well as veterinarians and zootechnics.

Pathogenesis: After insertion, leptospires disseminate haematogenically and cause polyorganic damage, primarily of the kidneys, the CNS and the liver.

Clinical picture: Incubation period from 6 hours to 20 days. Acute onset with common toxigenic syndrome, pronounced myalgias, two-wave temperature curve, hepatic lesion with jaundice, renal - acute renal failure, serous meningitis, hemorrhagic syndrome, rare skin rashes.

Diagnosis - based on epidemiological and clinical data, is confirmed by changes in the hemogram - BSR-induced leukocytosis at the onset of the disease and with serological response - lysis-agglutination (after the eighth day). Rather, leptospira in urine and blood (with direct bacterioscopy) can be proven.

Forecast: Generally good, more serious in icterohemorrhagic form.

Treatment: hospitalization, etiological treatment with antibiotics. From I order: Penicillin - from 4 to 12 IU/day for a period of 10 days. Alternative preparations: tetracyclines or chloramphenicol. Pathogenetic treatment of acute renal failure and hepatoprotective agents.

Prevention: no specific. Use of work clothing when working in water or in underground conditions.

TETANUS

Acute infectious disease involving NS and typical seizures.

Etiology: Clostridium tetani.

Epidemiology: Wound infection from tetanus spores in wounds with anaerobic conditions. Professional is the disease of agricultural workers, livestock farmers, gardeners, construction workers, excavators and others.

Pathogenesis: One of the ingredients of tetanus exotoxin - tetanospasmine has pronounced neurotropic activity, suppressing the retention processes in neuromuscular synapses due to increased glycine secretion.

Clinical picture: Incubation period of 1-4 days to several months. After prodromal signs from the wound - the entrance door, muscle stiffness develops with trizmus, risus sardonicus, abdominal rigidity, high fever, fulsome sweating, and finally with tetanous seizures.

Diagnosis - based on epidemiological and clinical data.

Prognosis - very serious. High lethality, especially in adult patients and those with a short period of time from the onset of the disease to the onset of first seizures.

Treatment: mandatory hospitalization in intensive care unit, relaxing regimen, feeding tube nutrition. Etiological treatment: with antibiotics from I order - Penicillin - 4-8-12 IU/day for a period of 10 days. Alternative preparations: cephalosporins, ampicillin. Second place as etiological treatment: hyperimmune human immunoglobulin at a dose

of 6 to 8000 U muscular or antitoxic heterologous anti-tetanic serum - 100-200,000 U, by the method of Bezredka. Pathogenetic treatment with oxygenation, sedative agents, neuroleptics, if necessary, and apparatus ventilation.

Prophylaxis: with specific tetanus toxoid - in the DTC vaccine in childhood and as monovaccine in adults over 60 years of age.

TULAREMIA

Acute infectious disease with a polymorphic clinic, with predominantly lymph node involvement.

Etiology: Francisella tularensis.

Epidemiology: zoonosis - humans get infected by direct contact with sick wild animals, rarely while domestic animals are manipulated with their skin. Rarely, contamination can also occur after a tick bite. Occupational nature is the disease for hunters, forest workers, leatherworkers and animal by-products, rabbit-farmers, and veterinarians and zootechnics.

Pathogenesis: Serous-purulent inflammation, especially lymph nodes, develops at the site of the infections. Generalization with septic forms can also occur rarely.

Clinical picture: Incubation period of 1-7-14 days. Acute onset of general toxigenic syndrome. Most common is the bubonic form with the development of "botons" - painless, non-growing, non-ulcerated lymph nodes. Eye-bubonic, skin, pulmonary, septic forms are rare.

Diagnosis: based on epidemiological and clinical data. Confirmed with serological tests - agglutination reaction with specific antigen and ELISA immunoassay.

Prognosis: Good, more serious in septic form.

Treatment: hospitalization, bed rest. Etiological treatment with antibiotics - from I order: Streptomycin - 1 g/d, tetracyclines such as: Doxycycline - 2 x 100 mg daily oral or Tetracycline - 3 x 100 mg intramuscularly for 10 days. Alternative preparations: aminoglycosides or chloramphenicol.

Prophylaxis: specific for a vaccine. Precautions for handling animals - use of gloves.

PESTIS

An acute infectious disease belonging to the group of particularly dangerous infections, which have a varied clinical picture.

Etiology: Yersinia pestis.

Epidemiology: The main way to infect humans is by biting the fleas in contact with a sick person. In the pulmonary form of plague, the infection is by air-droplet route. Professional is the disease of hunters, forest workers who are in contact with wildlife, as well as of medical workers serving the sick, especially those from the pulmonary and septic forms.

Pathogenesis: Inflammatory process of the lymph nodes develops at the place of introduction with the formation of pest "pimples". From drop infection - severe haemorrhagic-necrotic alveolitis. There may also be primary or secondary haematogenic dissemination.

Clinical picture: Incubation period ranging from 6 to 10 days. Acute onset with marked toxigenic syndrome. Plague bubones are solid, painful, merging with surrounding tissues, often maturate and ulcerating. Pulmonary plague is a very severe haemorrhagic pneumonia with acute respiratory failure and poor evolution.

Diagnosis: based on epidemiological and clinical data. Confirmed by the isolation of the agent from the bubonic content, blood or sputum.

Prognosis: very serious, especially in pulmonary and septic forms. Treatment: obligatory isolation, strict bed rest, observing all measures for handling particularly dangerous infectious disease: special protective clothing, rubber boots and gloves, mask, safety glasses. Etiological treatment with antibiotics - in combination with Chloramphenicol up to 4 g/day + Streptomycin - 2-3 g/day + Tetracycline - 4 g/day for 10-15 days. Pathogenetic treatment. **Prevention:** specific - with a live antipest vaccine. Apply to risk contingents and in an epidemic situation.

TUBERCULOSIS *

Tuberculosis is an infectious disease that can be professionally dependent under certain conditions.

Etiology: Tuberculosis in humans is caused by two major types of tuberculous bacteria: human (typus humanus) and bovine (typus bovinus) type. As an aerobic bacterium, it is best developed in the lung parenchyma, so the pulmonary form, both in the domestic and in the professional aspect, is the most common. This does not exclude other, extrapulmonary localizations of tuberculosis to be etiologically related to the occupation of the ill.

Epidemiology: two main sources of occupational contamination - tuberculosis patients; tuberculously diseased domestic animals (cattle, pigs, horses, goats, dogs, cats) from which sick cows have the greatest importance (especially for udder tuberculosis). The most common route of infection is the air - droplet (spray) and dust particles containing the bacteria. Less frequent infections are through the digestive system and injured skin (eg in the case of slaughter of sick animals). Threatened contingents for professionally-mandated tuberculosis are: medical and other staff serving tuberculosis patients in

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specialized health care establishments; livestock farmers, milkers, veterinarians, milk controllers, and workers from slaughterhouses contacting tuberculosis-infected animals.

Pathogenesis. After infection, the tuberculous bacteria fall and remain most commonly in the lungs. From there they spread through blood and lymphatic routes - the so-called initial bacteraemia. Two immunological phenomena arise - specific allergy and specific immunity. An expression of the specific allergy is skin hypersensitivity to tuberculin (demonstrated by the Mantoux skin-allergic test with 5U PPD, which is reported at 48-72 hours). Specific immunity is natural when acquired after a tuberculosis infection and acquired when it occurs artificially 4-8 weeks after vaccination with an attenuated or avirulent strain (BCG vaccine).

Clinical picture: Professional tuberculosis occurs as well as domestic, such as primary or secondary tuberculosis, with the most common pulmonary localization (the extra-pulmonary forms of tuberculosis - bone-joint, serous, urogenital, intestinal and hepatolienal, lymphatic, are rare forms).

Clinical manifestations are very varied and depend on form, stage, localization, age, immune system of the organism. The illness may also occur completely asymptomatically (to be detected accidentally by fluorographic or x-ray examination). It rarely develops as an acute infectious disease, and more often as chronic pulmopathy.

Subjective complaints distinguish between two groups of symptoms: intoxication phenomena - subfebrile temperature, adynamia, loss of weight, sweating, cardiovascular lability (tachycardia, hypotonia, etc.), gastrointestinal symptoms, etc.; by the respiratory system - cough, expectoration, haemoptysis, chest pains and soreness, shortness of breath.

The physical finding is also very different. Tuberculosis of the tracheobronchial lymph nodes, limited focal changes, small and deeply situated infiltrates, tuberculoma, and in the small cavernous forms did not reveal deviations with the physical methods. Scarce can be the changes even in a miliary tuberculosis. The percussion finding depends on the stage and expression of pulmonary fibrosis and pleural changes, the size of the infiltrates and the caverns - there are attenuations of varying intensity. Auscultation can cause sharp or weak vesicular breathing, and some areas may also experience a variety of wet wheezing. Over the large peak caverns, the "kroning" fields are narrowed by the tracheal adduction and the peak pleural adhesions.

The functional breathing study is mainly deviated in the chronic fibrosis and fibrous-cavernosal forms - the type of restrictive-obstructive ventilatory failure. There may also be confused gas diffusion and evidence of hypoxemia and hypercapnia in severe forms.

X-ray changes in pulmonary tuberculosis are characterized by marked polymorphism. For example, the primary pulmonary complex revealed homogeneous shadowing associated with pulmonary hilus, which was expanded with unclear outlines; in focal tuberculosis has medium and large shadows, and tuberculoma is presented with a well-rounded solid circular shadow; caseous pneumonia is like massive lobar pneumonia, and miliary tuberculosis is represented by uniformly scattered small-spotted shadows - "starry sky", etc.

Bronchoscopy shows: endobronchial stenosis, fresh granulations, deformations and fistulas on the bronchi (fibrobronoscopy takes material for cytological, microbiological and histological examination).

Clinical-laboratory tests: BSR is differently accelerated; mild to moderate leukocytosis, mild to moderate neutrophilia, lymphopenia with monocytosis and sometimes eosinophilia; in chronic forms - anemic syndrome; in tuberculous meningitis, the liqueur is clear with an increased lymphocyte and albumin content and a reduced sugar and chloride content; in kidney tuberculosis the sediment is poor, the albumin is slightly increased, there is microhematury.

Microbiological study: sputum or other biological secretions and milk from diseased animals with evidence of tuberculous bacteria in direct microscopy after Zyel-Nilsen staining or 40-45 days in Löwenstein-Jensen's solid culture medium talks about unveiling forms of the disease. Rapid cultures are confirmed by the Bac-Tech-Dickenson method, as well as express diagnostic by polymerase chain reaction for rapid bac. tuberculosis proofing. In the absence of an exposition - fibrobronchoscopy with bronchoalveolar lavage.

Immunological and enzyme tests: the blast transformation test (BTT) with tuberculin positivates at over 2.9% blast transformation of the cells; adenosine deaminase (ADA) in the blood in tuberculosis rises above 16-20U.

Diagnosis: Complex, based on epidemiological and clinical data. Bacteriological and radiological studies, supported by skin-allergy and immunological tests and biopsy material, are crucial.

Prognosis and acceptance of a professional character: the prognosis is serious - chronic forms of the disease. The primary incubation period for primary tuberculosis in adults is 6 to 8 weeks; in reactivation (secondary tuberculosis) the changes develop for a different period - up to 6 months. During a longer latency period, the probability of causal

relationship with the occupation decreases (in case of unrecognized recurrences, however, the latency period may be up to 12-18 months). In veterinary or livestock breeding, it is important to isolate bovine tuberculous bacteria.

Treatment: always combined with several tuberculostatic agents. Schedule most commonly used in our country: 600 mg Rifampicin (Tubocin) + 300 mg Isoniazide (Rimicide) + 1 g Streptomycin (after the second month is replaced by Ethambutol 25 mg/kg) In the caseous-productive forms, and pyrazinamide 2 g daily, for 3-6 months. The treatment is prolonged - up to 9 months, and in the extra-lung forms - up to 12-18 months. For the first two months, the treatment was conducted in stationary state, and then continued outpatient - under the supervision of the pneumophysiatric dispensary. All healing regimens combine with hepatoprotectors and vitamins - vit. A, B_6, and so on.

Prevention: specific - BCG vaccine, combating nosocomial infections, personal protective equipment, hygienic lifestyle, healthy nutrition.

II. Viral infectious diseases.

LYSSA (RABIES)

An acute infectious disease with specific CNS involvement.

Etiology: Rhabdovirus.

Epidemiology: source of domestic and wild animals with rabies. One gets infected after a bite or contamination with secretions from sick animals, most often dogs. Occupational risk is rabies for hunters, people who have contacts with dogs, veterinarians, laboratory medical workers (cases of laboratory infections have been described).

Pathogenesis: The causative agent is markedly neurotrophic by inducing polyoencephalitis with a characteristic localization in the CNS.

Clinical picture: Incubation period from several days to one year. Start with prodromal manifestations: hydrophobia, paresthesia in the wound, general restlessness, followed by an excitatory phase with psychomotor agitation and a third-paralytic form with manifestations of bulbar paralysis and lethal outcome.

Diagnosis: based on epidemiological and clinical data. Confirmed by the histological examination of the brain in the deceased.

Prediction: Absolutely bad.

Treatment: Symptomatic only. The administration of hyperimmune anti-rabbit immunoglobulin is attempted, but until now no effect.

Prophylaxis: specific - with a vaccine used in risk contingents. When biting or slobbering from a diseased or suspect animal - administering a vaccine against the disease, by scheme and, if necessary, in combination with immunoglobulin.

VIRUS HEMORRHAGIC FEBRIS

FEBRIS HAEMORRHAGICA CRIMENSIS (FHC)

Acute infectious disease with major haemostasis involvement.

Etiology: Congo - Crimean virus from the Bunyaviridae family.

Epidemiology: the source of the infection is domestic or wild animals, and a carrier on the human being - ticks, mainly of the Hyaloma species. One gets infected after a tick bite as well as in contact with the patient's blood. Occupational risk is the disease for farm workers, livestock farmers, veterinarians and zootechnics, as well as for all who are in contact with animal ticks (upon their removal). The occupational risk is very high for health workers to have contact with the patients - FHC is a particularly dangerous infection in this sense (even hospital-borne epidemy are described). Laboratory infections are also possible.

Pathogenesis: The virus primarily damages haemostasis by several mechanisms: direct vascular damage, indirectly through nervous trophics, and the development of disseminated intravascular coagulopathy.

Clinical picture: Incubation period 2 to 14 days. Acute onset with marked toxigenic and craniopharyngeal syndromes. After 2-3 days - severe hemorrhagic syndrome.

Diagnosis: Based on epidemiological and clinical data, it is confirmed by haemostasis disturbances - thrombopenia and virological studies: isolation of the virus from the blood of sick newborn mice or by serological methods - CFT and ANT.

Prognosis: serious, especially in severe forms and delayed treatment.

Treatment: Compulsory isolation and treatment regimen as particularly dangerous infections. Etiological treatment with hyperimmune human anti-haemorrhagic immunoglobulin - 12-24 cm^3, intramuscular or intravenous. Pathogenetic treatment - haemotransfusions, vascular sealing preparations.

Prophylaxis: specific - with a live vaccine administered to risk contingents. Animal decontamination.

HEMORRHAGIC FEBRIS WITH KIDNEY SYNDROME

Acute infectious disease with predominantly kidney and haemostasis involvement.

Etiology: Hantaan virus from the Bunyaviridae family.

Epidemiology: The reservoir of the virus is the mousies rodent. One gets infected with food con-

taminated by secretions and excretions of rodents. Professional is the disease for forest workers, agricultural workers, campers, brigaders, soldiers, construction workers.

Pathogenesis: After primary viraemia, a process of disseminated coagulopathy with a major involvement of the kidney glomeruli develops most often.

Clinical picture: Incubation period of 4 to 12-23 days. Acute onset with toxic-infectious and craniopharyngeal syndromes, moderate hemorrhagic syndrome. After 3-5 days, acute renal failure develops with two phases - anuric and polyuric.

Diagnosis: based on epidemiological and clinical data. Confirmed by the serological test with CFT, recently also by ELISA.

Prognosis: serious, especially in the absence of adequate treatment.

Treatment: compulsory hospitalization. There is no etiological treatment. Try ribavirin as an antiviral. Pathogenetic treatment - hemodialysis at ARF.

Prevention: no specific. Deratization.

HEPATITES VIRALES ACUTAE

Acute infectious diseases with predominant liver involvement. Five types of viral hepatitis caused by hepatitis A, B, C, D, and E are now known. Hepatitis virus G is suspected.

VIRUS HEPATITIS TYPE B

Ethiology: hepatitis virus type B from the Hepadna viridae family.

Epidemiology: Anthroponosis. Source are sick or healthy virus carriers. The infection is by blood or by sexual intercourse, and vertically - by the mother of the newborn. Occupational risk is the disease for medical workers who are in contact with the blood of the sick. Studies have shown that in the different countries of the world, 2 to 15% of the medical practitioners have had an infection (in our country some research shows 5-7%). The occupational risk is particularly high for lab workers, dentists, surgeons, obstetricians and otorhinolaryngologists as well as for staff in infectious departments. The risk is particularly high for nurses and laboratory technicians.

Pathogenesis: After viremia initiation, the virus selectively damages hepatocytes by indirect immunogenetic mechanism and activation of T-hepatocytotoxic cells.

Clinical picture: Incubation period from 40 days to 6 months. After an initial pre-icteric stage with adynamic, dyspeptic and rare arthralgic syndromes, there is icteric syndrome with hepato- and/or splenomegaly and a prolonged convalescence stage.

Complications: At 5% of patients prolonged and inveterated the process and developing chronic hepatitis B. In fulminant development, acute hepatic necrosis with hepatic coma.

Diagnosis: based on epidemiological, clinical and laboratory data - elevated serum transaminases. It is confirmed by the virological serological tests - proof of the antigens of the virus B: Hbs and Hbe, as well as their antibodies: anti-s, anti-e and anti-core IgM class.

Some patients also have co- or superinfection with hepatitis virus D (delta antigen).

Prognosis: serious, especially in fulminant and hepatic coma.

Treatment: compulsory hospitalization, bed rest and dietary regimen. Etiological treatment with interferons, but only in protracted and chronic forms, with insufficient effect. Pathogenetic treatment with hepatoprotective agents.

Prevention: specific - with a hepatitis vaccine (Engerix B, Hevax, Bevax, etc.). It is mandatory for all newborns, risk contingents and all willing (it is mandatory for medical workers).

VIRUS HEPATITIS TYPE C

Etiology: hepatitis C virus from Flavividae family.

Epidemiology: For the time being, the mode of infection by haemotransfusion and hemodialysis procedures has been proven. Occupational risk is the disease for patients undergoing chronic hemodialysis and especially for medical workers in the hemodialysis sectors and departments.

Pathogenesis: After initial viremia selectively most likely a direct hepatocyte lesion with a marked tendency to chronic process and development of chronic hepatitis type C.

Clinical picture: Incubation period of 30 to 90 days. The most common mild course with moderate pre-icterous and icterous stages, but with frequent protracted evolution, recurrence and chronication after the sixth month.

Diagnosis: based on epidemiological and clinical data, confirmation of serological testing and detection of anti-HCV antibodies (seroconversion occurs one to two months after the onset of the disease).

Prognosis: serious given the high risk of chronication.

Treatment: Mandatory hospitalization, bed rest and dietary regimen. Etiological treatment - interferons, in protracted and chronic forms such as: Intron A, Roferon, Heberon et oth. preparations in a dose of 3 or 6 IU, three times a week, for a period of 6-9 months.

Prevention: no specific. Mandatory screening of all donor blood banks for the presence of anti-HCV antibodies.

RUBELLA (EPIDEMIC ROSEOLA)

An acute infectious disease with rash, lymphonodulopaty and often embryotoxic effects.

Etiology: Anthroponosis. The infection is by air-droplet route. Contangion index is about 40%. Occupational disease has the sickness for worked in childcare facilities: children's homes, gardens, hospitals, nurses, doctors.

Pathogenesis: After initial viraemia, lymphotropy, dermatropy, and frequent damage to the fetus during pregnancy.

Clinical picture: Incubation period of 11 to 23 days. Moderately pronounced catarrhal manifestations, occipital and cervical, sometimes generalized lymphonodulopathy, macular rash. The inborn rubella develops with polymorphic malformative syndrome, hepatosplenomegaly, haemorrhages.

Diagnosis: based on epidemiological and clinical data, confirmed by serological tests: CFT, ANT and RDF.

Prognosis: good.

Treatment: There is no etiological.

Prophylaxis: specific with a live antiviral vaccine that is used in the trivaxin - morbilli + mumps + rubella (obligatory in the immunization calendar for children).

SYNDROME OF ACQUIRED IMMUNITY FAILURE - AIDS

This is the terminal phase in the evolution of infection caused by the human immunodeficiency virus - HIV, from the Retroviridae family.

Epidemiology: Anthroponosis is the source of ill and healthy virus carriers. Infection occurs by sexual intercourse, blood and blood products transfusions, in contaminated instruments, cosmetic and other procedures, as well as vertically from the mother of the newborn.

Occupational risk is the illness for medical workers - who are in contact with the blood or sex secretions of the sick or infected. According to WHO data, the occupational risk for health workers is very low - by 1993, WHO registered 119 medical workers infected professionally with AIDS. These are mainly lab technicians, orderlies, surgeons and pathoanatomists, nurses. The most common of these infections were when they were pricked with a contaminated needle.

According to the WHO, the risk of occupational infection by FHC, VC type B and AIDS is as follows 100: 30: 1.

Pathogenesis: The virus selectively damages the immune system, the T-helper cells carrying its CD4-specific receptor, resulting in immune deficiency with subsequent opportunistic infections, neoplasms and secondary bacterial infections. In one part of the infected virus it also exhibits primary neurotrophy.

Clinical picture: HIV infection passes through the following phases with a different time interval between them:

- a primary clinical complex with a mononuclear-like picture;
- asymptomatic phase, with or without indicator opportunistic infections;
- generalized lymphonodulopathy;
- terminal phase of AIDS with general signs, opportunistic infections, bacterial infections (mostly tuberculosis) and neoplasms.

Diagnosis: Based on epidemiological and clinical data, the available immune deficiency - a reduction in the absolute number of TCD4-lymphocyte cells below 400 in mm^3, is confirmed by virological studies: HIV p24 antigen detection, anti-HIV antibodies by ELISA, and confirmed by a Western blot immunoblotting test.

Prognosis: Bad for those entering the final phase of AIDS.

Treatment: antiviral therapy with azidotimidine (Retrovir, Zidovudine) at a dose of 600 mg daily, orally for a period of 6-9 months and more. In combination with other antivirals: dideoxycytidine or dideoxyinosine, lamivudine, protease inhibitors. Treatment of opportunistic and bacterial infections.

Prevention: no specific. Safe sex, mandatory screening of all blood products. For the protection of medical personnel: work with gloves, carry out blood handling with vacuum tainers.

In HIV pricking or contamination with blood, the WHO recommends immediate initiation of azidotimidine prevention at 1200 mg daily for at least four weeks. It is even better with triotherapy: with AZT + lamivudine + one protease inhibitor.

III. Rickettsia and Chlamydia infectious diseases. ORNITHOSIS/PSITTACOSIS.

Acute infectious disease with predominantly respiratory system involvement.

Etiology: Chlamydia psittaci.

Epidemiology: source of domestic and wild birds. One is infected with direct contact with sick birds, most commonly on an aerosol route. There is a professional disease for poultry and workers in poultry farms, zoos, veterinary surgeons and zoo technicians.

Pathogenesis: the front door is the respiratory system where an inflammatory process develops in the alveoli. Rarely generalization is also possible.

Clinical picture: Incubation period of 3 to 30 days. Acute onset with common toxigenic syndrome, pneumonia and APD. There may also be some organ damage: hepatomegaly, serous meningitis, septic form.

Diagnosis: based on epidemiological and clinical data and confirmed by serological tests - CFT with group antigen. A dermal allergic ornithine test may also be used.

Prognosis: Good, more suspicious in septic form.

Treatment: Etiological treatment with antibiotics - from I order - tetracyclines, such as Doxycycline - 2 x 100 mg daily, oral or Tetracycline - 3 x 100 mg intramuscularly for 10 days. Alternate preparations: erythromycin or chloramphenicol.

Prevention: no specific. Struggle the birds epizoonotic.

Q - FEVER

Acute infectious disease with polymorphic clinical picture but with predominantly respiratory involvement.

Etiology: Rickettsia BURNETI.

Epidemiology: source is domestic animals and birds. Carrier is a tick, but there are several mechanisms of transmission of the infection: aerogenic, tick bite, almentary and others. Occupational risk is the disease for agricultural workers, livestock breeders (especially cowman), working in slaughterhouses, meat-packing companies, haylofts and farms. There are single laboratory infections.

Pathogenesis: After an initial phase of development in the monocyte macrophage system and dissemination, there is a predominantly pulmonary, rare and other organ localization.

Clinical picture: Incubation period of 3 to 12 days. Acute onset with toxo-infectious and craniopharyngeal syndromes, followed by pneumonia, hepatosplenomegaly, lymphonodalopathy. Endocarditis, serous meningitis and others are no-rarely developing.

Diagnosis: based on epidemiological and clinical data and confirmed by serological methods - CFT and immunofluorescence.

Prognosis: good. More serious in septic form and endocarditis.

Treatment: etiological treatment with antibiotics - from I order tetracyclines (see ornithosis). Alternative preparations - chloramphenicol or ciprofloxacin - 2 x 400 mg/day.

Prophylaxis: Corpuscular vaccine-specific; vaccine application for risk contingent.

MARSELIENSIS FEVER.

Acute infectious disease with a characteristic rash, a primary affection at the site of the induction of the causative agent.

Etiology: Rickettsia CONORI.

Epidemiology: the source is dogs, rarely, other domestic and wild animals. Carrier is tick - mainly Rhipicephalus sanguineus, but and other species. One gets infected mainly after a tick bite, but and when is crushed. Occupational risk is the disease for shepherds, goatherds, farm workers and forest workers.

Pathogenesis: Endotoxin reaction - "black spot" - "tache noire" is developing at the site of the depot. Generalization followed by systemic vasculitis.

Clinical picture: Incubation period of 3 to 15-20 days. Acute onset with toxo-infectious and craniopharyngeal syndromes, maculopapular rash on the body, including palms and feet, hepatosplenomegaly and "black spot".

Diagnosis: based on epidemiological and clinical data, confirmed by serological tests with indirect immunofluorescence and CFT with specific antigen.

Prognosis: good.

Treatment: etiologically with antibiotics - from I order - tetracyclines (see ornithosis). Alternative preparations: chloramphenicol or erythromycin.

Prevention: no specific. Desacarisation of dogs and animals.

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7.2 PROFESSIONAL PARASITIC DISEASES

ECHINOCOCCOSIS

Human echinococcosis is a chronic helminthic disease resulting from the development of the larval stage of canine tape worm (echinococcal cysts) in various organs and tissues of the organism.

Etiology: Echinococcus granulosus.

Sexually mature form is a small tapeworm With a size of 2.7 to 5.4 mm and parasitized in the small intestine of the final host the dog.It has a head with four suckers and twin corona of hooks, neck and 3-4 members, the latter being mature and containing 400 to 800 eggs.The eggs have a spherical shape containing six-hooked embryo (oncosphere), remain viable in the environment for up to 10-12 months.

The larval stage develops in the intermediate host, and is a cyst full of fluid, ranging in size from bean to large ball and more. The wall of the cyst has two envelopes: external - chitin and inner (germ). From the latter, proliferating capsules are formed in which the scolexes that float in the cyst fluid and form the so-called hydated sand develop. In addition to fertile (containing scolelex), sterile (acephalic) cysts without scolexes also appear **(Figure 1)**.

Epidemiology: Echinococcosis is a synantropic-endemic oral zooantroponosis with natural outbreaks (endemic outbreaks occur in areas with pasture livestock). The main source for man is the domestic dog and very rarely the wolves, the jackals and the wild dog Dingo. Intermediate hosts are the man and many grazing animals (sheep, goats, buffaloes, pigs, horses, camels, elephants, etc.). The mechanism of human infection occurs most often in direct contact with a dog or indirectly - consumption of poorly washed fruits, vegetables, etc.; sheep milking, shearing and wave processing; in the collection of dog and other animal skins; when drinking water from open waters and others. This makes the echinococcosis occupational disease for certain risk groups of the population - stock-breeders, farm workers, leather collectors, breeding and training dogs, etc.

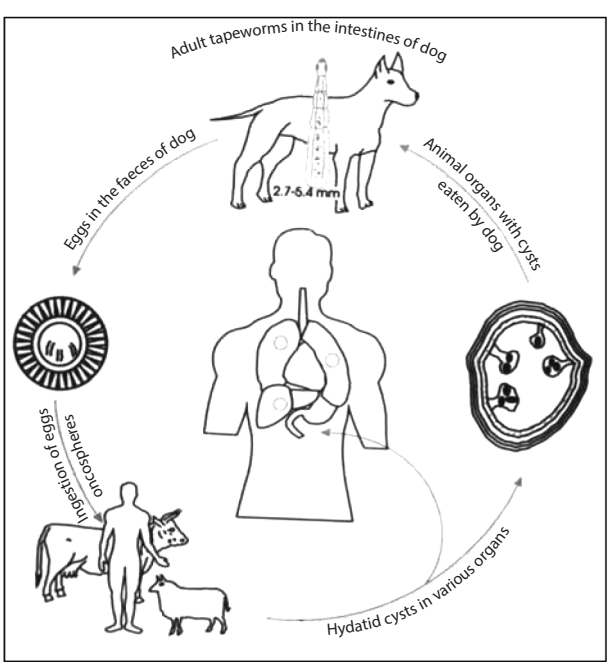

Fig. 1. Biological cycle of Echinococcus granulosus

Pathogenesis: Mechanotrauma and toxo-allergenic effects. Echinococcal cyst rupture - severe anaphylactic shock and scolexes dissemination - multiple echinococcosis.

Clinical picture: The incubation period is from several months to ten years or more. Symptoms of the disease are dependent on organ localization. Most commonly, the cysts are localized in the liver (50-60%), the lungs (35-38%), the brain (3-5%), and then spleen, kidney, peritoneum, bones, muscles, and others.

Regardless of the organ localization, the disease has three stages: 1. Latent - until the first symptoms appear. 2. Stage of subjective complaints - most common weight and support in the right subcostal or epigastric, headache, dizziness, itching, urticaria, dry cough, blood spit (up to 30% of cases of pulmonary echinococcus), easy tiredness. 3. Stage of objective changes - in case of localization in the right part of the liver, the pain syndrome is as in cholecystitis, and in the left part - occurs with nausea, weight in the epigastrium; in the cerebellum cyst - headaches, paresis, paralysis, stagnant papillae, etc.

Laboratory parameters have long been normal - sometimes minor eosinophilia, hyperproteinemia, dysproteinemia, interferon inhibition, etc. have been observed. Functional liver tests change only with large liver cysts. Serological echinococcosis reactions are positive, but in some cases they may also be negative (large old cysts with calcium deposits or immunodeficiency). The prognosis is unfavorable in relation to frequent recurrences (over 21%) and lethality of 1 to 15%.

Diagnosis and differential diagnosis: Complex on the basis of epidemiological data, history and results of laboratory and apparatus research. Basic methods in diagnostics are: parasitological (morphological and immunological); clinical; biological; paraclinic and apparatus (abdominal ultrasonography, CT of the liver and other organs, lung radiography, etc.). The most accurate information is obtained from CT, taking into account the density of the Echinococcus cyst in Hunsfield units. For pulmonary echinococcus, the symptom of Escudero-Nemanov (change in the shape of the cyst when breathing) is characteristic of X-ray.

Early diagnosis - by mass prophylactic studies of risk groups of the population: fluorography, echography and serology: RIHA, RIF, ELISA, etc.

Differential diagnosis - with hemangiomas, polycystic disease, dyssembryogenic or biliary cysts, amoeba abscess, liver cirrhosis, tumors and other focal changes.

Treatment: Radical treatment is surgical. Conservative chemotherapy is only applicable to patients with serious contraindications for surgical intervention (not always with good healing effect). The preparations Vermox (Mebendazol), Zentel (Albendazol), Praziquantel (Biltricid) are used.

Operated patients are dispensary - up to 5 years.

Prevention: Personal prophylaxis requires that contact with dogs and strict adherence to personal hygiene be avoided. Public prophylaxis includes decontamination of dogs - with Dronsit (4-fold dehelminthisation during the year); burial of animal carcasses; veterinary-sanitary control of meat production and others.

ANCYLOSTOMIDOSES

Ankylostimidodes are helminthoses that are chronic with allergic manifestations, dyspeptic and anemic syndrome.

Etiology: "Ankylostomidoses" involves two parasitoses - Ankylotomiasis (Ancylostoma duodenale) and Necatoriasis (Necator americanus), which have a similar clinical picture. Causes are small nematodes ranging in size from 7 to 13 mm. Their body is cylindrical, with a well-developed oral capsule with cutting chitin teeth and platelets. Female parasites emit an average of 9,000 to 30,000 eggs. Ankylostomas live up to 5 years, and neckators up to 15 years. They feed on blood and fragments of the intestinal mucosa (daily one parasite absorbs 0.02-0.2 ml blood). Sexually mature helminths inhabit the duodenum and other parts of the small intestine. Eggs of the parasite fall into the outside environment in an immature form through the stools. At a temperature of 14-37°C, moisture and oxygen from them hatch

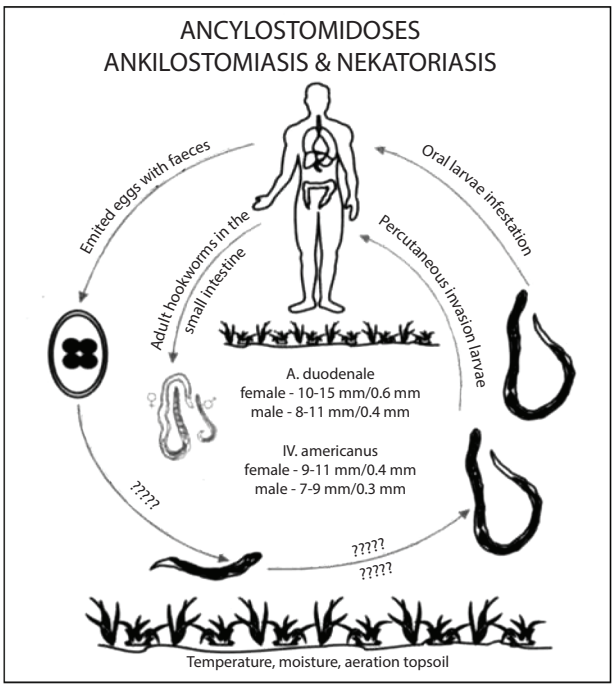

Fig. 2. Biological cycle of A. duodenale and N. americanus

a rhabdiform larva, which after 7-10 days becomes infestive (filamentary). In contact with skin and mucous membranes they penetrate the body and through the blood reach the lungs, migrate through the alveoli and fall into the digestive tract where they develop to adult helminths **(Figure 2)**.

Epidemiology: Ankylostomidoses are natural endemic percutaneous and oral anthropogeohelmintoses. Epidemiological incubation is 35-40 days. They are spread in the tropics and subtropics. In the temperate zone they are rarely encountered - in mines, wet and warm places. The source of the invasion is the parasitic man - through the stools.

Pathogenesis: Mechanotrauma, sensitization, malabsorption syndrome, anemic syndrome - iron deficiency anemia.

Clinical picture: Three stages are distinguished:

1. Stage of percutaneous invasion - erythema, itching, swelling, papules, vesicles and pustules (7-10 days).

2. Stage of pulmonary migration - spastic cough, shortness of breath, haemoptoea, headache, subfebrility, Lyofler syndrome *, eosinophilia, leukocytosis (7-14 days).

3. Stage of intestinal localization (acute and chronic period, mild, moderate and severe form) - nausea, vomiting, epigastric pain, diarrhea, dehydration, easy fatigue, anemic syndrome, occult bleeding in the faeces, hepatomegaly, limb edema, ascites, cardiac decompensation with pulmonary edema.

* Lyofler syndrome - fast-passing eosinophilic infiltrates in the lungs.

Diagnosis and differential diagnosis: Diagnosis is certainly assured by (faecal helminthoovoscopy); Berman** helmintholaroscopy and the cultivation and differentiation of larvae on Harada and Mori***.

Differential diagnosis is made with ascaridosis, strongyiloidosis, anemic syndrome with other aetiology, avitaminosis, heart and kidney disease, etc.

Treatment: For etiologic therapy, preparations: Vermox, Laevamizol (Decaris), Zentel, Flubendazole and Combantrin (Pyrantel, Pamoat) are used.

Prevention: Personal prophylaxis - prevents skin contact with soil, grass and water in endemic areas. Public - search and treatment of patients with subsequent dispensarysation; mass dehelminthization in endemic areas; protection of the environment from faecal contamination; hygienisation of working conditions in underground sites.

TOXOPLASMOSIS

Toxoplasmosis is a protozoonosis that occurs most often asymptomatically (rarely as a disease with a pronounced clinical picture).

Etiology: Toxoplasma gondii. It is a bonded intracellular parasite. It occurs in three forms: a vegetative (trophozoite) of 4-7 μm in size, actively penetrating the cells of the body (toxoplasmic cells are called pseudocysts and are found in the acute stage of the disease); form of cist - the so-called true cysts that have their own envelope and are a conglomerate of thousands of toxoplasms; oocyst form - proved in 1970 and sexual reproduction in the epithelial cells of the intestinal tract of a domestic cat and some wild cat species that emit oocysts with their feces **(Figure 3)**.

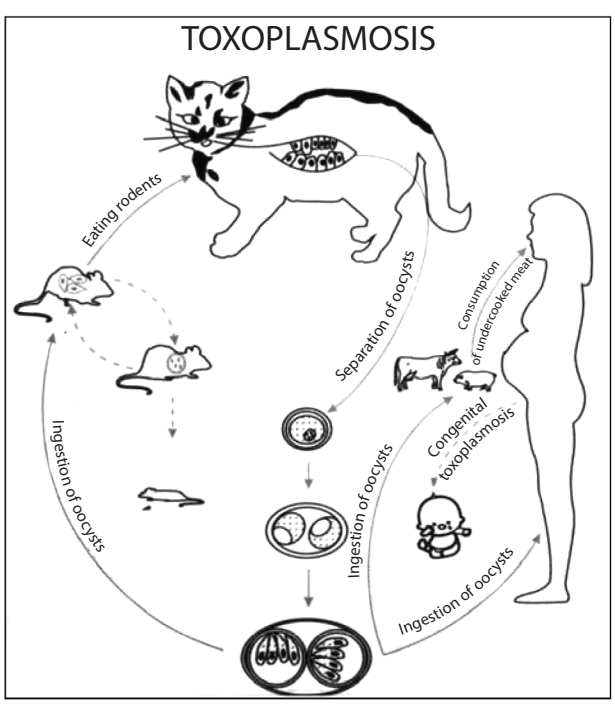

Fig. 3. The biological cycle of Toxoplasma gondii

** Berman - a method for isolating and microscoping larvae from parasites.
*** Harada and Mori - method of cultivation and differentiation of larvae from gut parasites

Epidemiology: Toxoplasmosis is a cosmopolitan, contact-home zooanthroponosis. The source of infection is most often the animal - very rarely a person. The infection is carried out in an alimentary - through the consumption of meat products from animals containing cysts, fruit and vegetables contaminated with oocysts and contaminated hands. It is also possible transplacental infestation of the fetus.

Pathogenesis: Mechanotrauma and toxo-allergenic effects. Not every infiltration of the parasite leads to disease (in nature pathogenic strains are rare, but apathogenic toxoplasmic strains are always immunogenic).

Clinical picture: There are three options:

1. Asymptomatic leakage - only positive serological reactions.

2. Congenital toxoplasmosis - hydrocephalus, calcifications in the brain, chorioretinitis, iridocyclitis, uveitis, micro-ophthalmia, optic nerve atrophy, delayed neuropsychological development.

Acquired toxoplasmosis - lymphadenitis, chronic subfebrility, ocular manifestations (chorioretinitis, uveitis, haemorrhage), brain disorders, myocarditis, interstitial pneumonia, exanthema, etc.

Diagnosis and differential diagnosis: At clinical data diagnosis can hardly be diagnosed. Study material is taken - blood serum, lymph node biopsy, albumin, impress of placenta, brain, umbilical cord, amniotic fluid. The main methods of diagnosis are serologic reactions - RIHA, RIF, ELISA, etc. It is important to have a two-fold serological test in 2-3 weeks - the dynamics of the titres. The presence of elevated IgM antibodies is an acute process. Morphological methods - 3 passages by inoculation of white mice over 5 days and preparations from biological materials stained by Romanovski-Gimza. Differential diagnosis is made with infectious mononucleosis, malignant neoplasms, cytomegalia, rubella, brucellosis and others.

Treatment: Etiological treatment is conducted with: Rovamycin, Tindurin, Sulfalen, Biseptol (Trimesol), Clindamycim, Calcium Foliante. Rovamicin is assigned 2 tabs. 4 times a day for 10-14 days. Tindurin is prescribed on 1 tablet 2 times a day for 5 days. Sulfalen is given 2 g daily in three doses for 5 days.

Prevention: Strict personal hygiene, avoiding the consumption of insufficiently heat treated meat and milk, washing fruits and vegetables, avoiding contact with cats. It is recommended that pregnant women be examined at the beginning of pregnancy to determine their immune status to toxoplasmosis.

Tab. 1. PROFESSIONAL INFECTIONS (by Choen R., 1990)

Diseases	Factor	Target organ	Professional source	Risk professions	Preventive measures
Bacterial Anthrax	Bacillus anthracis	skin, lung systematica-lly	powder (spores in wool, goat hair, fur)	weavers, livestock workers veterinarians, farm workers	immunization
Brucellosis	Brucela abortus, B.suis, B.canis,B.meli - tensis	systematica-lly	blood, urine vaginal secretions secretions and tissues of cattle, pigs, sheep, deer	workers in slaughterhouses and canning plants, manufacturers of lifesaving products, veterinarians, hunters	personal hygiene, serological identification of infected animals
Erizipeloid	Erysipelothrix rhusiopathiae (insidiosa)	skin	fish, meat, molluscs, poultry	fishermen, veterinarians, workers employed by meat and poultry processing	personal hygiene, gloves
Leptospirosis	Leptospira interrogans	liver, brain, kidney systematica-lly	urine or tissue from domestic or wild animals or excreta of rodents, contaminated water	agricultural workers, farmers, workers in slaughterhouses, veterinarians, fishermen, miners working on sewers	personal hygiene, boots, gloves, immunization of animals identification of contaminated waters, prophylaxis with doxycycline
Plague	Yersinia pestis	lung, systematica-lly	fleas from infected rats, wild dogs, martens	hunters trappers	immunization
Tetanus	Clostridium tetani	nervous system	soil prick with dirty sharps	construction workers, gardeners farmers	immunization
Tuberculosis	Mycobacterium tuberculosis	lung, systematica-lly	infected subjects, primates	medical workers, technicians, caregivers of primates	skin test, followed by prophylaxis for Sero-positive
Tularemia	Francisella tularensis	ulcerated papule, systematica-lly	blood, tissue, secretions or bite of an infected animal or arthropod	hunters, loggers, farmers, veterinarians	personal hygiene, gloves, immunization, control insects
Fungal Candidiasis	Candida albicans	skin	frequent skin injuries in a moist environment	washers containers, working in poultry farms and canning factories	skin protection, protection from moisture
Koksidioi-mycosis	Coccidioides immitis	lung, meninges	soil (spores) in arid areas	farmers archaeologists, builders	control dust (where possible)
Dermato-phytosis	Microsporum Trichophyton Epidermophy - ton	skin	animals, hot humid environment	breeders, shepherds, athletes	personal hygiene, protection from moisture
Histoplasmosis	Histoplasma capsulatum	lung, systematica-lly	soil contamination with feces of birds	farmers workers in canning plants	dust control, rehabilitation of the environment, spraying formaldehyde contaminated surfaces
Helminth and protozoan Echinococcosis	Echinocous granulosus E.multilocularis	CNS, lung, liver	feces of infected dogs, foxes and others from this group	stock-breeders, veterinarians, shepherds	personal hygiene
Ankilosto-midoses	Ancylostoma duodenale, Ne - cator america - nus	small intestine	larvae in human feces passing through intact skin	farmers walking barefoot, sanitation workers, parks and gardens	building latrines, the use of boots, shoes and gloves

Toxoplasmosis	Toxoplasma gondii	reticulo endothelial system, eyes	cat feces	technicians, veterinarians, breeders of cats	personal hygiene
Rickettsial and chlamydial Ornithosis	Chlamidia psittaci	lung systematically	disposal of secretions from infected poultry (parrots, pigeons, etc.).	breeders, sellers in shops for pets, zoo officials	detection and treatment of infected birds
Q fever	Coxiella burnetii	systematically liver, lung, brain	placental tissue, bird secretions, excretions from infected animals (sheep, wild animals, goats, etc.).	technicians, workers in slaughterhouses, farmers, stockbreeders	personal hygiene, immunization
Viral Encephalitis	Arbovirus	CNS	laboratory viral cultures infected arthropods	working in virology laboratories	immunization, insecticides, using organic secure methods
Hepatitis B	Hepatitis B virus	liver	accidental introduction of infected human blood or blood products	maxillofacial surgeons, dentists, dialysis workers, clinical laboratory technicians, nurses	personal hygiene, immunization
Rabies	Rabies virus	CNS	wild animals (foxes, bats, polecat), rarely domestic animals	technicians, veterinarians, hunters, people growing wild or unidentified animals	immunisation in contact or immunisation of some animal kinds (dogs, cats)
Rubella	Rubeolla virus	fetus systemat.	infected people	health workers	immunization
AIDS	HIV	Immune system	bodily secretions of infected people	health workers	prophylaxis by universal preventive means for skin and slimes

Seroepidemiological screening among toxoplasmosis risk groups - stock-breeders, zootechnics, veterinarians, laboratory workers, meat - and slaughterhouses workers, etc.

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8 OCCUPATIONAL ALLERGIC DISEASES

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8.1 INTRODUCTION. CLASSIFICATION

The sensitization of the organism to the numerous environmental factors, manifesting itself as latent and clinically manifest, is one of the complex forms of interaction between man and nature. An average of 10% of the world's population is affected by allergic diseases - they occupy an important place in the general morbidity structure of industrially developed countries. Accordingly, the number of professionally-determined allergic diseases, whose relative share in total allergic morbidity varies from 3 to 10%, and in the case of registered occupational diseases - from 10 to 30%.

Professional allergies are associated with specific etiological factors from the work environment. Their number is constantly increasing and today there are few jobs that do not pose a risk of allergies.

A professional allergen is a substance that is found in the human working environment and leads directly or indirectly to sensitization. The sensitization ability of allergens is different - strong allergens are thought to have high affinity for human proteins and therefore lead to frequent and severe allergies to workers. Substances with weaker immunogenicity sensitize only individuals - so-called atopics[1].

It is believed that in atopics there could be no MAC for allergens, however, in most non-atopic patients there is a dose-response relationship. That is why MAC adopted for this substances to be determined by their so-called "sensitizing threshold" - this concentration, which produces an allergic reaction in an experiment. In connection with this, they are divided into classes:

Class I - substances with an allergy threshold lower than 1 mg/m^3. Their MACs should be 10 times lower than the threshold;

Class II - substances with an allergy threshold of 1 to 10 mg/m^3. Their MAC. Is 5 times lower;

Class III substances with an allergy threshold above 10 mg/m^3. In their case, MAC Is 3 times lower.

Classification. Allergens are plant, animal and chemical factors, and in contact with the body are: inhaled, contact (undamaged skin), oral and penetrating through injured skin and mucous membranes **(Figure 1)**.

Risk factors. The action of professional allergens can be facilitated e.g. from a number of exogenous other work environment factors - physical, chemical, biological, which can lead to a change in general or local reactivity, or cause anatomophysiological changes that potentiate the action of allergens - ex. mechanical and toxic irritants and mucosal infections of the respiratory system, eyes and skin. In addition to exogenous, endogenous risk factors (so-called "allergic diathesis") are important: age, gender, family predisposition, endocrine and nervous system conditions.

Occurrence of a professionally allergic disease is a result of a complex, two-sided process between external and internal risk factors. In occupational pathology, great importance is attributed to external factors (occupational allergens and irritants), as unintentional hereditary workers, who are subjected to prolonged periods of action by strong industrial allergens, may develop mass allergic reactions, and backbred hereditary persons may remain healthy under favorable working and living conditions.

Pathogenesis. Allergen, as a genetically foreign biological irritant of the immune system, causes the formation of specific allergic antibodies or sensitized cells (lymphocytes) that are produced in the human immune system. All cells have a unified origin - from the polypotential bone marrow strain cell. During embryonic development, some of them under the influence of the thymus differentiate into small lymphocytes - T-lymphocytes, which are the effectors of cellular immunity, i.e. of delayed hypersensitivity (IV type). Its expression is eczema, eczema-like dermati-

[1] The atopy is characterized by the following main features: enhanced synthesis of reactive antibodies due to a genetic defect associated with decreased T-suppressor Ly; a positive skin reaction (papules and erythema) to the corresponding allergen; family predisposition for allergic manifestations.

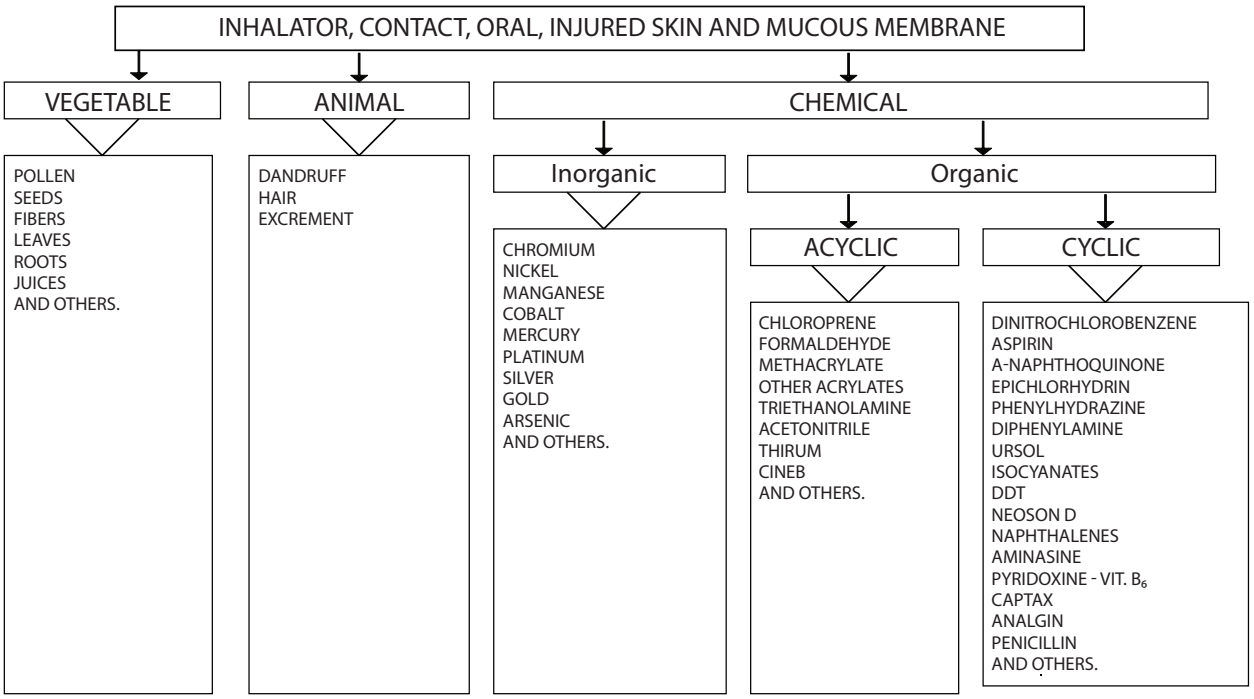

Fig. 1. Classification of professional allergens

tis, some forms of bronchial asthma, and the chronic form of hypersensitive pneumonitis (HSP). Other thymus cells, regardless of thymus, differentiate into B-cells in bone marrow, probably in lymphatic-intestinal structures. Activated by allergens, they are transformed into plasmatic cells that synthesize human immunoglobulins A, D, E, G and M. Of these, E, G and M are important for humoral, antibody-mediated immunity, i.e. for fast - type I, and immunocomplex - III type, allergic reactions. The rapid (type I) allergic reaction is mediated by antibodies - reagins, with anaphylactic reactions, urticaria, Quinche allergic edema, allergic rhinitis, bronchial asthma, allergic conjunctivitis and atopic eczema. Expression of the immunocomplex (III type) reaction, mediated by antibodies agglutinins and precipitins are: some forms of allergic rhinitis, bronchial asthma and acute form of hypersensitive pneumonitis. Because of its complex antigenic mosaic, most professional allergens can cause both a humoral and a cell-type sensitization.

Occupational allergic diseases can have various organ manifestations but most often engage the eyes, the skin and the respiratory system, which is related to the functions of these organs and their anatomical and physiological features - the skin, for example, Is the barrier of the human organism to the environment and in the workplace it is exposed to continuous irritants and allergens. The same type of effect applies to the respiratory system and eyes.

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8.2 ALLERGIC DISEASES OF THE RESPIRATORY SYSTEM

The type of respiratory allergy is often determined by the place of deposition of allergens (nose, larynx, trachea, bronchi and alveoli). The significance of the size of the inhaled particles and the intactness of the lining is essential. For example, in the silk industry in the silk-worm, where the dust is more roughly dispersed and retained in the upper respiratory tract, allergic diseases of the upper respiratory tract predominate. In the production of silk itself, where the allergen is inhaled with steam deeply to bronchioli, bronchial asthma is more common. The number of substances that cause direct or indirect respiratory allergy in the professional environment is progressively growing. According to their mechanism of action, they are divided into three groups:

1 group – allergenic substances leading to sensitization in I, III, and possibly IV immune-allergic type: medicaments, bioproducts, enzymes, epiallergens, pollens, molds, bacteria, etc. .

II group – irritants, which have only an irritant effect on the lining and its receptors: quartz, asbestos, carborundum powder, ammonia, acid vapors, bases, etc.

III group of substances with complex action (allergic, irritant and histaminoliberal) on the respiratory mucosa: most organic powders, salts of heavy metals, organic solvents, some pesticides, paints, lacquers, piperazine, plastics and others. Along with the reflex (vagal) mechanism, some of them also have a histaminoliberal mechanism - some organic powders, ammonium persulfate, snake and bee poisons, some medications such as amphetamine, D-tubocurarine, galamine, papaverine, morphine, atropine, codeine, strychnine, ACTH, synacten, vitamins A, B, C, D, K, dextran, peptones and others. The substances of III g represent the greatest risk of allergies due to the mutual potentiation of the bronchospastic effects (allergic and non-allergic).

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8.2.1 ALLERGIC DISEASES OF THE UPPER RESPIRATORY TRACT

Allergic diseases of the upper respiratory tract (URT) occupy a leading position in the structure of allergic diseases, as the mucosal epithelium of URT is one of the first barriers to allergen contact with the body.

Etiopathogenesis. URTs occupational allergic diseases are related to the individual's residence in the work environment. Occupational allergens penetrate the body by inhalation route in the form of simple or complex compounds such as formaldehyde, epichlorhydrin, furan, isocyanates, aromatic nitrobenzene compounds, pyridazinone, chromium, nickel, cobalt, platinum, beryllium, polymers, organic powders, medicaments, molds, bioproducts and others. Their sensitizing effects may be due to a previous mechanical damage to the lining or to inflammatory changes. The illness occurs in different times from the start of work.

URTs occupational allergic diseases include several nosological units - allergic rhinitis, rhinosinusitis, pharyngitis, laryngitis and tracheitis.

Allergic rhinitis occurs most often and occurs in two forms - acute and chronic. A classic example of allergic rhinitis is **pollenosis** (allergen - tree, grass and flower pollen) that is related to the work environment only for a limited number of professions (agronomists, herbalists, greenhouses workers). The immunological response to the pollenosis is of type I hypersensitivity. Symptoms include: itching in the nose, throat irritation, olfactory disorder, watery secretion from the nose, running early or late (10-12 hours). Common signs of weakness, headache, difficulty breathing, and seasonal dependence are also observed. The rhinoscopic examination shows a pale or livid colored swollen mucosa. Serum IgE is elevated. Eosinophils are increased in the blood and nasal secretions.

In occupational **allergic rhinitis**, complaints are related to the individual's residence in the work environment and resolve in the elimination of contact with allergens at work.

The complaints of the patient are similar to

those of pollenosis: episodes of sudden obstruction, sneezing, nasal secretion, olfactory changes, difficulty in breathing, headaches, which, however, are not seasonally dependent and do not take place acutely. Rhenoscopy reveals: mucosal hyperplasia, narrow nasal passages, secondary infections and lesions, pale mucous membrane.

Allergic rhinosinusitis often occurs in allergic nasal disease but is not always registered because of the prevalence of allergic rhinitis. When combined, the name "allergic rhinosinusopathy" is also seen, which is also observed in two forms - acute and chronic. Sometimes the mucosal polyposis develops. The polyps are pale and pink, with a soft consistency and influenced to appropriate therapy. They further increase nasal obstruction, create conditions for additional banal inflammation, and this is a serious prerequisite for the occurrence of concomitant bronchial asthma and other allergic diseases. The objective study establishes edema, hyperplasia and polyposal degeneration of the lining, mainly bilaterally in the maxillary and ethmoidal sinuses.

Allergic pharyngitis occurs in two forms - acute and chronic. The pharyngeal mucosa is involved in the body's general reaction in contact with production allergens. The focal process in the swallow (tonsils, tonsilar epithelial rest, lymphoid ring) plays an additional role for the sensitization and auto-sensitization of the organism.

Acute edematous pharyngitis (swelling of Quinke) is characterized by acute leakage, diffuse edema of uvula, swallow, tongue and entry of the throat, oral cavity, affecting parts from the face. Breathing, itching, general reaction of the body occur.

Allergic laryngitis can develop alone or in combination with allergic pharyngitis and rhinitis. It occurs in two forms: acute (diffuse or restricted allergic laryngitis) and chronic (laryngopathy, catarrhal, hyperplastic, pollenosic allergic laryngitis). This disease often affects the following professions: textiles, millers, bakers, chemists. Additional factors such as voice overtension, cold air, psycho-emotional tensions that are characteristic of vocalists and teachers lead to illnesses that are neither inflammatory nor functional, and in their etiology, the presence of dust from the scene, library dust, cosmetic materials. Leading complaint is dysphonia, swallowing, and in severe seizures - inspiratory dyspnoea.

Allergic tracheitis rarely occurs as a single disease. It is most often combined with allergic laryngitis and pharyngitis in the form of allergic laryngotracheitis or pharyngolaryngotracheitis. The leading symptom is persistent paroxysmal coughing, which is due to the presence of highly reflexogenic zones in the trachea.

Diagnosis. Of relevance to the diagnosis of occupational allergic diseases of the UAP is the focused profphatological history with a study of the professional route and the factors of the working environment. It is necessary to clarify the relationship of the patient's complaints to his/her place of residence in the work environment - exposure and elimination tests, and clinical, laboratory and other studies:

- detailed ORL review;
- allergenic tests:
  - skin allergy testing;
  - determination of total Jg E in serum;
  - specifically Jg E in the serum;
- endoscopic ORL examination;
- cytological examination of nose material;
- bacteriological examination of the material of the nasopharynx;
- X-ray examination of sinuses;
- examination of mucociliary function of the nasal mucosa (nasal-mucociliary clearance, electron microscopy);
- status of the UAP function (nasal inspiratory air flow, anterior and posterior rhinomanometry);
- olfactometry;
- blood tests: BSR, differential count, Jg E, antineutrophil cytoplasmic antibodies.

Treatment. An obligatory condition for the treatment of occupational allergic rhinitis is the elimination of the ill worker from the harmful working environment. Local treatment is by nasal spray - beclomethasone preparations or cromoglycates. Oral therapy includes antihistamines, calcium preparations, Vit. C, specific desensitization with the causative agent - allergen, immunotherapy. Synthetic cortisons are not recommended in everyday practice, except for extreme situations. Especially favorable for the outcome of the disease is the use of physiotherapy and climatic treatment. In some cases, surgery is also involved.

Working capacity for UAPs occupational illness depends on the localization of the process, the duration of the disease, the clinical signs, the nature of the work activity (permanent or periodic contact with occupational allergens), the presence of combined diseases (UAP and skin manifestations, UAP and pulmonary localization of allergic process, etc.). In case of a tightening and absence of general allergic reactions in dynamic follow-up (endonasal allergy testing), the worker continues to work in the profession but is tested once a year and hyposensibilization therapy is performed. In case of initial disease, it is treated as a professional and temporary work is recommended for another job. In the case of a significant spread of the allergic process in the UAP, long-term removal from the given profession (dynamic monitoring and therapy twice a year) is determined.

In the cases of poly-allergy and marked clinical signs, as well as during a long period of illness, rational labour and working capacity discussions in WEMC are carried out. Combined with bronchospasm and allergic diseases of the skin and lungs, the issue of limited and permanent disability is discussed.

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8.2.2 PROFESSIONAL BRONCHIAL ASTHMA

B. Ramacini (1700) describes an increased incidence of bronchial asthma amongst flourman and millers - he is considered the first researcher of this occupational disease. It is also known that in the nineteenth century frequent cases of asthma were reported among a number of professions: pharmacists, furriers, bakeries, etc.

It is currently reported that in the US, 5% of the population suffers from bronchial asthma, and 2% is professionally determined. In Germany, professional asthma accounted for 12% of all reported occupational illnesses, while in Bulgaria for 1991 - 17.3% (retrospective analysis for a 10-year period shows its uneven increase). The main distinction of professional asthma from other forms of bronchial asthma is that it is associated with a certain etiological factor from the work environment. The number of these factors has steadily grown - more than 300 causative agents are registered in more than 100 risky occupations that can cause bronchial asthma.

In occupational asthma, substances (powders, vapors, and gases) provoke bronchospasm through a variety of mechanisms:

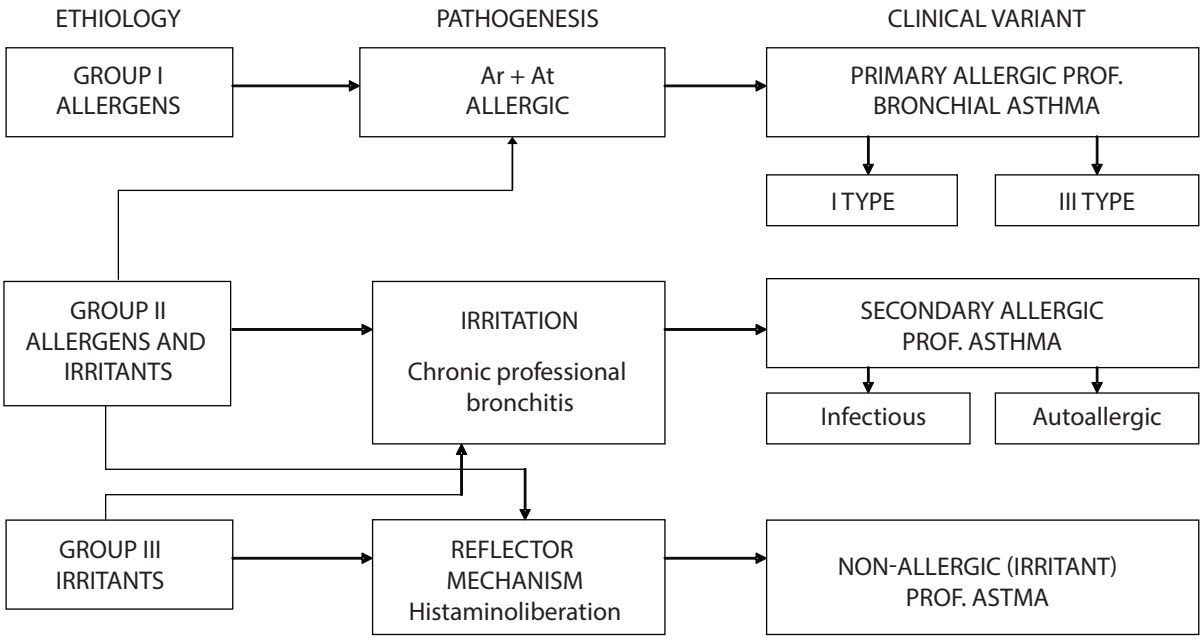
- **Allergic** as a result of the reaction between Ar and At, through complete antigens and so-called haptenes with subsequent release of mediator's substances responsible for clinical symptomatology.
- **Inflammatory**, by inflammation of the lining, which increases the sensitivity of bronchial receptors. It is known that inflammatory responses can also be initiated by non-immunological activation by cellular and humoral effector mechanisms **(Figure 1)**.

Substances such as lectins (concavalin A, phytohemagglutinins), bacterial polysaccharides, viruses, trypsin, papain, silica and asbestos, activate non-immunologically lymphocytes, macrophages, mast cells, basophils and even the complement system. Concavalin A and phytohemagglutinins selectively stimulate T-lymphocytes, while polysaccharides stimulate B-lymphocytes and macrophages. Non-immune activation of effector cells and mechanisms can also be induced by other agents. Fungal products, for example, activate by non-immune way T-cells and macrophages, leading to interstitial fibrosis. These agents stimulate lymphocytes to release lymphokines, and basophils and mast cells - mediators, as well as macrophages - interleukin 1. Silicon dioxide and asbestos directly stimulate macrophages to release interleukin 1, which in turn stimulates fibroblast growth, collagen synthesis, and T- and B- cells.

- **Histaminoliberated**, via endogenous non-immunological histamine release.
- **Blockade of β-adrenergic bronchial receptors** and uptake of α-adrenergic receptors, with subsequent tendency to bronchospasm.

Most substances in the working environment act on a complex pathogenic mechanism. For example, isocyanates released in the manufacture of plastics, paints, lacquers and adhesives have in their chemical structure reactive NCO groups which, in contact with the bronchial mucosa, can functionally block β-adrenergic receptors with subsequent imbalance and the effect of α-adrenergic bronchial receptors. In addition, the isocyanate molecule has a high affinity for reacting with the amino groups of the proteins, whereby the isocyanate-hapten becomes a com-

Fig. 1. Classification of occupational asthma



plete antigen, i.e. non-specific activators, together with specific antigens, play an important role in immune-allergic reactions.

There is nothing specific about the **clinical picture** of professional asthma - specificity is related to its etiology and therefore, in most cases, paroxysmal dyspnea with noisy whistling breathing is exposingly dependent on the work environment. They occur more often at work, but are not uncommon when bronchial spasm occurs later and seizures can occur after leaving work and at night. Using functional and immunological methods, it has been shown that substances with chemical and organic-powder nature can cause two types of rapid reactions - those that are blocked by intal and others that are not blocked by it. Two forms of late bronchospastic reactions have also been studied - one begins after 1-2 hours and disappears in the fourth to sixth hour, and the others are held for hours or appear at night. Biphasic reactions have also been observed - a rapid, introducing, passing, followed by a second, late reaction. Most professional factors, as well as bacteria and fungi, can cause both rapid and delayed reactions, and sometimes biphasic.

The main pathogenetic mechanisms for the occurrence of bronchial asthma determine its different forms:

- Immunological, **Allergic asthma**;
- Non-immunological, causing so-called **Irritative (non-allergic, syndrome) asthma**;
- Combined, leading to the so-called **Mixed asthma**.

A common sign of all forms of asthma is the increased sensitivity of the bronchi to a number of non-specific or specific stimuli - i.e. bronchial hyperreactivity.

Allergic form in professionally determined asthma may occur suddenly in visible health, i.e., primary, or to form against a background of another chronic occupational pulmonary disease with frequent infections (pneumoconiosis, chronic occupational bronchitis, etc.) as a complication thereof, i.e. secondary.

The primary-allergic form occurs both in atopic and non-atopic individuals. In the first, it occurs in the rapid type of hypersensitivity with the formation of reagin antibodies and the formation of bronchoconstriction at 15-30 minutes from contact with the allergen. In noatopic, it develops semi-delayed with the addition of complement and agglutinin and precipitin antibodies, where bronchoconstriction occurs after 4-6 hours. Diagnostic skin and inhaled provocative tests are positive at 30 min or 4-6 hours, respectively.

The secondary-allergic form is most often infective-dependent and rarely occurs with auto-allergic mechanisms. Clinically, this form is severe with frequent attacks that are difficult to treat. Labour readjustment is ineffective, unlike primary-allergic asthma.

Non-allergic professional bronchial asthma (called irritant in Anglo-Saxon literature, and in French-syndrome) is a common form of occupational asthma, especially in the chemical industry. It does not happen with allergic symptoms, but the bronchospastic crises are dependent on the working environment and therefore the change of work has a good healing effect.

Criteria for Diagnosis of professional bronchial asthma **(Table 1)**.

The occupational hygienic criterion, through professional history, production characteristics of the

Tab. 1.

Criteria for allergic professional asthma	Criteria for non-allergic professional asthma
1. Labour-hygiene criterion. 2. Epidemiological criterion. 3. Exposure period. 4. Nonspecific increased bronchial reactivity 5. Positive exposure test. 6. Positive elimination test. 7. Specifically increased bronchial reactivity to prof. allergens. 8. Detection of specific antibodies to professional allergens by skin-allergy tests or by more sophisticated immunological methods.	1. Labour-hygiene criterion. 2. Epidemiological criterion. 3. Exposure period. 4. Nonspecific increased bronchial reactivity. 5. Positive exposure test. 6. Positive elimination test.

enterprise and sanitary inquiry from the medical services, occupational safety or NII, proves the presence, type and amount of the professional risk.

With the epidemiological criterion and the so-called collective test is looking for a possible increase in bronchospastic manifestations in a group of workers in the profession.

In the so-called exposure period it is established whether the disease does not precede work in the profession.

The first three criteria, the proving of non-specific increased bronchial reactivity through mediators, β-blockers and others, pharmacodynamic substances, as well as the exposition dependence of seizures through the so-called exposure and elimination tests, are equally valid for both primary forms of occupational asthma.

In so-called allergic professional asthma outweighs the diagnostic value of skin allergy tests, bronchoconstriction tests with allergens and, in some cases, immunological studies (RAST, immunoprecipitation, haemagglutination, etc.). The data from these tests are only relevant when they correlate one-way with the history and clinical course. On the other hand, negative skin-allergy tests can not categorically reject the professional nature of asthma. This is especially true for chemically-induced bronchial asthma.

Treatment

The wide variety of etiological factors and complicated pathogenetic mechanisms in occupational asthma require an individual approach when formulating the healing plan **(Figure 2)**.

It is particularly important to have targeted and adequate **etiological** treatment. Labour readjustment here is equally effective for both primary-allergic and non-allergic professional asthma.

In the etiological aspect of allergic asthma, it is also possible to rely on specific hyposensitization with the professional allergen if it is organic-dust etiology. Infectious-dependent secondary-allergic bronchial asthma also includes treatment with polyvalent bacterial preparations such as respivax or bronchovaxom, ribomunil and others.

The main task of **pathogenetic** treatment is to deal with bronchial obstruction. Included in the treatment plan are agents for suppressing the release of mediators from mast cells and their blocking, as well as those for controlling bronchospasm, edema and discrimination - direct and indirect sympathicomimetics, parasympathicolytics, methylxanthines and corticosteroids.

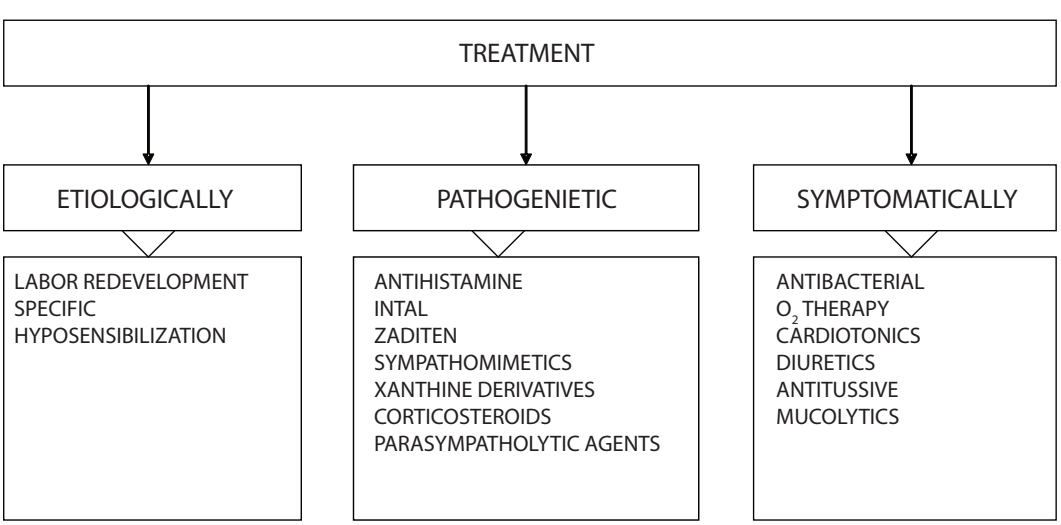


Fig. 2. Treatment of occupational asthma

Symptomatic treatment is used for complications such as insert infection, respiratory and cardiac insufficiency. Treatment requires special attention in the presence of concomitant diseases such as arterial hypertension, IHD, ulcerative disease, diabetes: in the case of cardiovascular diseases, the preparations of Atrivent and Hamona are recommended for inhalation; in coronary disease cortisone preparations are administered, mainly inhaled and only in vital signs - parenterally; diabetes also avoids cortisone treatment, but cortisone-dependent asthma regularly monitors blood glucose with readiness to correct anti-diabetic treatment.

Labour expertise. Temporarily disabled are patients with mild and initial forms of asthma, after which they have to be labour-readjustment without harm, but with equal qualification. Where this is not possible or when the ability to work is not reimbursed for 12 months, permanent disability (invalidity) is determined and determines the percentage of lost working capacity.

Prevention. Modern technical prophylaxis, by technological and ventilation improvements (automation, sealing) reduces the risk of allergies. Professional recruitment is an essential tool of medical prophylaxis. The importance of periodic medical examinations of risk contingents is very important - to detect the initial forms of bronchial asthma.

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M. Demirova, Y. Simeonov, M. Stoynovska, D. Baltadjieva

8.2.3 HYPERSENSITIVE PNEUMONITIS

Hypersensitive pneumonitis (HSP) is an allergic inflammation that engages the most peripheral airway structures - the alveolar wall and the interstitial. Depending on the source and its causes, there are different names - J. Pepis groups the syndromes in the disease unit "exogenous allergic alveolitis"; in the United States his synonym is "hypersensitive pneumonitis". Organic powders and the thermophilic mushrooms contained therein are the major sources of the disease. Similar lesions are also caused by some enzymes (e.g., proteolytic produced by Bacillus subtilis); antibiotics (for example, basitracin in forage mixtures) and chemical compounds (e.g., desmoduron used in plastic production). To date, more than 20 species of HSP have been known, with their number growing constantly **(Table 1)**.

Pathogenesis. It is currently assumed that the main mechanism of development of hypersensitive alveolitis is immune-allergic, which is also aided by non-immune activation. It is believed that in the

Tab. 1. Etiological factors, sources of HSP

Synonym of the HSP	Sources	Etiological factors
		I. Bacteria
Farmer lung	Mouldy hay	Micropolispora faeni
Bagasosa	Mouldy bagass (sugar cane)	Termoactinomyces sacchari
Mushroom - grower's lung	Compost	Thermophilic actinomycetes
Disease of detergent workers	Washing powder	Proteolytic enzymes of Bacillus subtilis
		II. Fungi
Pulmonary aspergillosis	Spores on aspergillus	Aspergilus fumigatus
Suberosis	Cork powder	Penicillinum frequentans
Sequoiasis	Wood powder from sequoia	Aureobasidium
Maple bark disease	Maple bark powder	Criptosoma corticale
Disease of the carpenters	Wood dust	Alternaria
Bookwarmer's disease	Paper dust	Alternaria
Cheese-maker's disease	Production of cheese	Penicillinum casei
Brewer's disease	Malts	Aspergilus clavatus, niger, fumigatus, nidulans
Wine-maker's disease	Grapes	Botritus cinerea
Weaver disease	Textile powder	Cephalosporium trochoderma
Disease of fur-dressers	Leather powder	Pleurotus florida
Disease of millers	Flour powder	Aspergilus flavus
Newguinea disease	Mouldy papua roofs	Streptomyces olivaceus
		III. Animal
Bird's lung	Dust and excrement	Bird proteins
Disease of breeders	Fish meal	Fish protein
Experimenters disease	Urine from rats and experimental animals	Animal proteins
		IV. Medications and chemical substances
Lung of metals	Aerosols and metal compounds - Al, Cr, Co, Ni, Zn etc.	Metal - protein complex
Isocyanate lung	Toluendiisocyanate	Isocyanate haptene
Lung of pituitary powder	Animal pituitary powder	Proteins of cattle and pigs

acute forms of forehead is the third type of immuno-allergic reaction in which the specific precipitating antibodies bind to the antigen, forming immune complexes with complement activation. For chronic pulmonary fibrosis, stage leading is the allergic reaction of type IV. Sensitized T-lymphocyte cells then bind to the antigen, releasing the lymphokines responsible for local tissue inflammation and necrosis. The immune-allergic mechanism in hypersensitive pneumonitis is of paramount importance, but it is also aided by an additional non-immune mechanism. Disclosed in the cell wall of various bacteria and fungi lipopolysaccharides directly activate an alternative complementary pathway, resulting in the release of anaphylatoxins - chemotaxis for phagocytes. Lipopolysaccharides stimulate lymphocytes to release lymphokines, and lymphocytotoxins and macrophage activators (responsible for allergic inflammation).

During biopsy, clusters of neutrophils, monocytes, lymphocytes, plasmocytes, histiocytes, macrophages, and giant cells have been observed, with granulomas devoid of caseous necrosis unlike tuberculomas.

Clinical symptoms are a combination of bronchopulmonary cough with scarce expectoration, inhaled or mixed type dyspnoea and flu-like syndrome - fever, sweating, headache, myalgias with arthralgias. An objective study is the so-called "silent" dyspnoea, i.e. breathless with cyanosis without noisy whistling breathing. In auscultation, creaking or small wet wheezes are heard in the lungs. For diagnosis is essential the radiography of the lungs. In the acute stage there are infiltrative-pneumonic shadows of different density and size, located bilaterally and symmetrically in the middle and lower pulmonary fields **(Figures 1 and 2)**. In the chronic stage, there are X-ray data for diffuse pulmonary fibrosis and emphysema **(Figure 3)**. Functional breathing patterns indicate pulmonary restriction and disturbed gas diffusion - reduced diffusion capacity, and in heavier, and especially chronic, hypoxia in blood-gas analysis. In contrast to bronchial allergies, in hypersensitive pneumonitis no bronchial hyper-

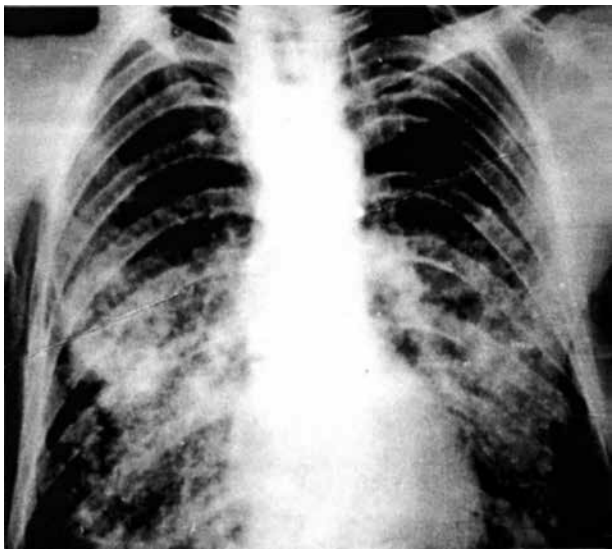

Fig. 1. Acute stage of HSP - infiltrative-pneumonic shadows in stock-breeder (Thermoactinomyces)

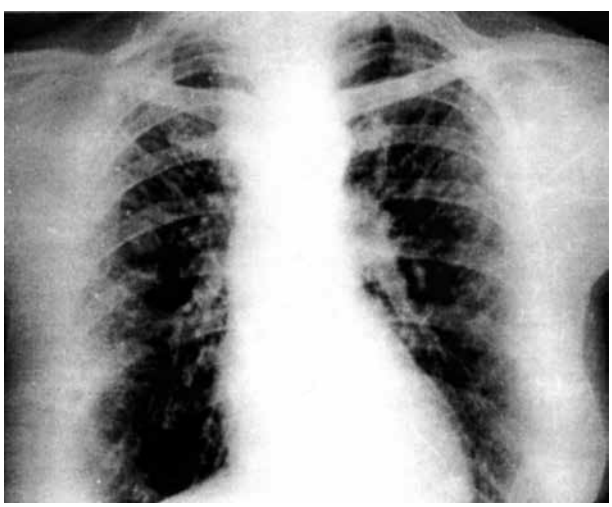

Fig. 2. The same case after 20 days of treatment with corticosteroids

reactivity was found - the broncho-provocative test with methacholine was negative.

Laboratory values is uncharacteristic and inconsistent, but may have leukocytosis with neutrophilia.

Important are the results of immune-allergenic tests with the suspected allergen: positive skin allergy tests to the professional allergen at 6 hours, positive immunoprecipitation tests (by Uchterloni, electroimmunodiffusion and other immunoprecipitation tests).

Also important for the diagnosis are bronchoalveolar lavage (BAL) data for a significant increase in IgG and a real increase in lymphocytes (predominantly T cells) and precipitating antibodies.

Diagnostic criteria. For the timely and correct diagnosis of HSP, two conditions are essential:

- to have a professional medical history;
- use a clinical minimum of laboratory and instrumental examinations **(Table 2)**.

The main diagnostic criteria are:

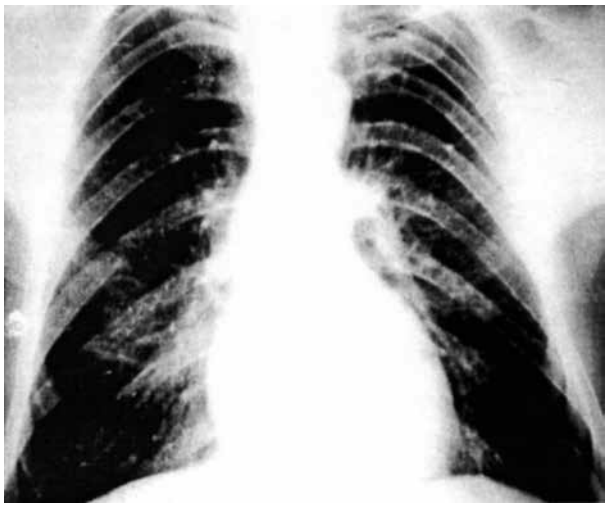

Fig. 3. Chronic Stage of HSP - diffuse pulmonary fibrosis in detergent worker (Bacillus Subtilis)

- **Clinical:** start of complaints 6 hours after professional contact with organic powders, detergents and other chemical substances;
- **Functional:** Restrictive or mixed type ventilator failure, reduced diffusion capacity, hypoxemia, normal bronchial reactivity;
- **X-ray:** data on infiltrative shadows in the middle pulmonary fields (acute stage) and diffuse pulmonary fibrosis (at the chronic stage);
- **Immuno-allergenic:** positive skin tests for the pro-allergen at 6 hours and evidence of specific immuno precipitin antibodies by Uchterloni or electroimmunodiffusion.

Treatment. From the etiological point of view, it is best to immediately remove the patient from the provoking environment. Only effective is the medication treatment of allergic inflammation with corticosteroids at a dose of 40-60 mg daily. Dehydrocortisone with gradual dose reduction for a total duration of treatment of 3-4 weeks - for prophylactic use, cortisone therapy should be combined with a broad spectrum antibiotic. Symptomatic therapy is conducted in the acute stage when there is respiratory failure and in the chronic stage with definitive pulmonary fibrosis leading to respiratory and heart failure.

An antibacterial therapy is added to the inflammatory process in chronic forms in the treatment plan.

Allergic alveolitis concomitant diseases require careful judgment - in hypertension and IBS, cortisone treatment is conducted under enhanced and controlled hypotensive and coronarodilatory therapy; in ulcer artery disease, corticosteroids are administered by parenteral and inhalation routes only, with daily Hb control, occult blood stools and abdominal status; in diabetes, oral treatment is combined with aerosol cortisone treatment in blood glucose control.

Tab. 2.

Symptoms	Laboratory indices
Shortness of breath, cough with scarce expectoration; Fever, sweating, headache, myalgia, arthralgia.	I. Routines: ESR, PKK, KGA, CRA II. Specific: HPP at 6h and 24h to professional allergens; Immunological tests to demonstrate specific precipitates.
Clinical features: Dyspnoea, predominantly of the inspiratory type with cyanosis, no noisy breathing; You can hear creaking or small wet wheezes in the lungs and the middle fields.	
Instrumental methods: Ro-graphy and scintigraphy of the lungs, FID, diffusion test, bronchoconstriction tests, exposure tests (in rare cases), BAL, fibrobronoscopy with biopsy	

Labour expertise. Hypersensitivity pneumonitis is a disease with serious consequences. In order to avoid chronic, with definitive pulmonary fibrosis, stage of the disease, it is necessary to perform an immediate labour readjustment through WEMC. At dequalification determines a percentage of loss of working capacity.

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8.3 PROFESSIONAL ALLERGIC CONJUNCTIVITIS

The eye has specific anatomical and physiological barriers that make it unique in its immune response - the eye has the ability to perform all types of allergic reactions. The conjunctiva is very often a place of these reactions because it is easily contacted for various exogenous allergens. The normal conjunctiva is a natural barrier that prevents the penetration of exogenous substances. It has a huge number of cells involved in inflammatory reaction-lymphocytes, plasmocytes, neutrophils. Ig from all 5 classes were found in the subepithelial tissues, and in the prelimbic zone Ig - producing plasmocytes. The leak film contains immunoglobulins - A and G, complement, lactoferins, lysozyme and they form the first protective barrier to the pathway of exogenous antigens. The proximity of the conjunctiva to the eyelid skin and the connection of the lacrimal pathways to the nasal mucosa is a prerequisite for the emergence of combined dermato - and rhino-conjunctival forms.

Occupational risk exists in many professions (endangered occupations): workers in the chemical industry, cement production, agriculture, pesticide production, pharmaceutical and cosmetic industry, textile industry, food, shoe and leather industry, medical personnel, etc.

Pathogenesis. In patients sensitive to one or more allergens, a local type I-type hypersensitivity reaction followed by a response to the ocular muco-

sa. The immunological response begins with a specific binding of an antigen with an IgE antibody on the membrane surface of the conjunctiva mast cells, defined as "tryptase-containing" and "tryptase + chymase-containing". "Pre-formed" mediators (histamine, chemotactic factor, cytokines) and "newly synthesized" mediators / prostaglandins - PgD2, leukotrienes - LTC4, LTD4, LTE4 are released. The presence of mediators of inflammation results in acute conjunctival symptoms, as well as secondary accumulation of neutrophils, Eo, Ba. These cells are prolonged by the inflammatory processes ending with chronic inflammatory association of non-specific hyperreactivity. The release of biologically active substances leads to conjunctival hyperemia, increased vascular permeability, hypersecretion of the mucosal glands, edema of the eyelids and conjunctivitis.

Clinical picture. Acute conjunctivitis is seen in a single encounter with the allergen. Subjective complaints are: itching, burning, irritation, dryness or tearing in the eyes, photophobia. The objective symptoms are: diffuse conjunctival hyperemia, bulbous and tarsal conjunctival haemorrhage, watery-mucous lucid secretion, edema of eyelid skin, eyelash rib hyperaemia, papillary hypertrophy, follicular reaction, blepharoconjunctivitis, keratoconjunctivitis.

Chronic form occurs as an IV type reaction. In this form there are less objective symptoms than subjective complaints.

Pathogenetically, T-cell mediated allergic reactions occurring with inflammatory infiltrative changes in ocular tissues. These include:

- Contact dermatitis and eyelid eczema
- Blepharitis
- Contact allergic conjunctivitis and keratoconjunctivitis (concomitant contact dermatitis).

Diagnostic methods. Biomicroscopy; skin-allergic tests (skin-prick test, intradermal test, epicutaneous test); immunological diagnosis: determination of IgE specific IgE immune-enzyme method "ELISA", UniCAP-test system, determination of total Ig-E, determination of inflammatory factors - eosinophilic cationic protein; cytological study of curettage material from the tarsal and fornix conjunctiva for the presence of Eo, Ba, Ne, Pl; elimination and exposure test; microbiological examination of conjunctival secretion.

Diagnostic expertise is associated with difficulties arising from the fact that there are no limit concentrations or safe concentrations of allergens in the working environment. For workers with a genetic predisposition to allergic diseases, contact with professional allergens in the work environment only unlocks the disease.

Criteria for etiological diagnosis are:

- Occupational risk for allergy;
- Collective epidemiological test;
- SAT with professional allergens;
- Elimination and exposure test.

Treatment. The first determining factor in the treatment of allergic eye diseases is the establishment of an allergen and the discontinuation of contact with it.

The means of immunopharmacology are specific and non-specific hyposensibilization, cytostatics, immunostimulators, steroids and non-steroidal anti-inflammatory agents, antihistamines.

Medicinal anti-allergic agents are for general and topical use. Anti-allergic preparations for topical use are:

Prophylaxis. Medical prophylaxis includes preliminary and periodic medical examinations of workers. For some professions, a thorough allergic history and research is needed to "treat atopic individuals". Early diagnosis is necessary for timely rehabilitation of patients. Recently, so-called "lubricants", as a preventive agent for conjuctiva contacting by environmentally harmful agents.

MEDICINAL	DOSAGE MODE
LIVOSTIN	2 DROPS MORNING
BICROMAT	4 x 1 DROP
VISINE	2 x 1 DROP
SPERSALLERG	3 x 1 DROP
CUSICROM	3 x 1 DROP
EMADINE	2 DROPS MORNING
ALERGODIL	2 x 1 DROP
ALERGOFTAL	2 x 1 DROP
CELESTON “S”	3 x 1 DROP
MAXIDEx	3 x 1 DROP
ULTRACORTENOL	3 x 1 DROP

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9 PROFESSIONAL MALIGNANT NEOPLASMS

D. Todorov

9.1 ONCOLOGICAL DISEASES - BASIC SITUATIONS

Oncological diseases (malignant neoplasms) are considered to be a composite term involving a large *heterogeneous group of over 200 different nosological units*, previously called "localizations". Individual oncological units have common characteristics, but they differ significantly. Thus, breast, lung, prostate, testicular, melanoma, ovarian, and so on cancers, are considered as separate nosological units rather than localizations of a disease. Some of them, according to modern perceptions, are also formed by several different nosological units - small cell and non-small cell lung cancer, seminomal and non-seminomal testicular tumors, etc. (especially great is the heterogeneity of the group of lymphomas, brain tumors and other oncological units). They differ significantly in their clinical picture, treatment options, prognosis, traceability and rehabilitation, and probably in their specific etiology and pathogenesis at the molecular level, hence the prophylactic approach.

Oncological diseases is generally characterized by impaired control mechanisms of cell proliferation, differentiation and apoptosis, heterogeneity of tumor cell populations, early dissemination and micrometastases, invasive growth, and others common characteristics.

Diseases have recently been identified as cell signaling pathways caused by activation of multiple oncogenes or loss of tumor suppressor genes.

One of the main functions of oncogenes is to encode proteins, components of intracellular signaling pathways, activated by growth factors. Therefore, overexpression of certain oncogenes or their permanent activation by mutation, leads to a continuous delivery of growth signals to the cell. The combination of a number of oncogene-activated signaling pathways may result in the transformation of the normal cell into a cancer cell. On this most recent theoretical basis, new approaches to the prevention, diagnosis and treatment of cancer are now being sought.

In the more advanced countries of the EU, *more than 50% of all cancer patients survive for more than five years and more than 60% in US (with a high percentage and radically cured patients)*, by early diagnosis and modern complex treatment and tracking, which is balanced and individualized for each patient. Progress here is also evident from the fact that the five-year survival in 1900 was only 5%, and since then it has grown slowly but steadily.

Epidemiology. According to WHO statistics, the world's cancer patients are currently over 15 million in total. Most of them are in developing countries. Every year about 10 million new patients are found and around 6 million per year die. According to leading American biostatistics, over 20 million new cancer patients are expected to be found in the world by 2020, and 10 million of them will die.

Oncological diseases in most developed countries is now second of socially significant diseases after cardiovascular diseases. However, scientifically-based projections indicate that, *around 2020, and even earlier, oncological diseases will become the most important disease in Europe*. This fact is explained both by the real positive results of the prolonged, well financed and organized at the highest state level cardiovascular prophylaxis in the developed countries, as well as the successes of their modern treatment. According to the latest data for the United States, there is probably now a social equalization between cardiovascular and oncological diseases.

Globally, taking into account developing countries, about one-third of all cancers are now being prevented by oncoprophylaxis, 1/3 are radically cured, and 1/3 are kept palliative in good quality of life.

However, for individual oncological units in the developed countries, overall mortality and morbidity still increase, albeit with different rates for individual nosological units, for different countries and regions of the world. Worldwide, the highest incidence of stomach cancer (although declining in developed countries and also Bulgaria in recent years), followed by lung cancer (although some decline in the United States, England and other developed European

countries, but not and in our country) and colon cancer. There are also trends for a predominant increase in prostate cancer in men and breast cancer and uteri cervical cancer in women. Taking into account the clinically unidentified microcarcinomas, prostate cancer is probably the most common tumor in men, and perhaps the most common tumor in humans at all (excluding skin cancer). *These onco-epidemiology data serve as a solid scientific basis for the organization of prophylaxis in developed countries*.

Every year around 27 000 new cancer patients are found in Bulgaria and about 17 000 die, with a total number of cancer patients in the country already exceeding 200 000. There has been a constant increase in the overall morbidity and mortality rate since the 1950s. Particularly unfavorable is the relatively late detection, at an advanced stage, of a significant percentage of cancer patients in our country. It leads to poor therapeutic results and a significant cost of treatment, which does not correspond to the capabilities of the developed countries of the European Union, US and Japan. The reasons for this are numerous - especially the lack of a scientifically and financially-based modern cancer program in our country, involving the whole society (and not just doctors-oncologists) at the highest priority national level, such as programs that have long been successfully implemented in all developed countries. Unsystemic and inadequate is the oncology training and qualification at all levels - the whole civil society, as well as the medical oncological and non-oncological staff.

Bulgaria also finds the well-known *"rejuvenation" of cancer mortality* observed in developed countries (but still the major percentage of cancer patients are in the "third" age). Thus, in people between 20 and 39 years of age, oncoletality has increased by about 40% in the last 15-20 years. In women between 15 and 59 years of age, oncological diseases accounts for 1/3 of the causes of death, while another 1/3 of cardiovascular diseases and 1/3 of all other diseases, traumas and road accidents.

Etiology and pathogenesis. As a *genetic disease of somatic cells*, the cancer is characterized by impaired regulation of cell proliferation and differentiation, unlimited invasive growth and metastasis, possibly caused by disorders in gene regulation and transmission of cellular signals between cells and/or within the individual cell. Increasingly importance in this respect has recently been given to the suppression of programmed cell death (*apoptosis*) by various molecular mechanisms.

It is assumed that the majority of *malignant tumors have clonal origin*. One or more of the dividing each day about 350 billion normal cells (about 1/3 of all cells in the body) is transformed into a tumor cell by natural mutation or physical, chemical or biological induction. As a result of the proliferation of this cell (becoming practically immortal, unlike normal cells), a new cell cluster is formed. It may result from: a malignant tumor and the clinical picture of oncological disease; a capsular benign tumor may be formed or the transformed tumor cells may remain in a "sleeping" state for a long time - they may also later develop a tumor or at least lack clinical manifestations throughout the human life. However, it is extremely rare that a clinically proven and histologically proven tumor undergoes a "spontaneous" reversal of development, with "spontaneous" regression and self-healing of the diseased, currently poorly understood mechanisms - for example, in melanoma patients, in chorioncarcinoma, kidney cancer.

The formation of a malignant tumor (oncogenesis, carcinogenesis) is a complex, prolonged and multifactorial process. Oncogenesis passes through three main stages:

- *The initiation stage* - lasts from minutes to hours and days;
- *A promotion stage* - lasts from a few weeks to several years. It has accumulated genetic changes in somatic cells, with activation of protooncogens and / or inactivation or loss of antioncogenes;
- *Progression stage* - progressive changes are likely to be reversible and, in principle, the possibility of reversing the tumor cell is normal. This view has an extraordinary theoretical and also practical relevance to oncology, but it is not yet conclusively proven in humans. So far, such **differentiation** of tumor cells to corresponding normal cells has been found primarily in tumor cell lines "in vitro" and in some leukaemias. It is the basis of vanguard *therapy with differentiating agents*, for example retinoids that are still of limited clinical use, and other potential differentiating antitumour drugs are in different clinical trial stages.

The malignant tumor may have a *long latency period that is clinically asymptomatic*. It then goes through *several clinical stages - local, loco-regional, recurrent and/or distant metastases*, and it is possible to develop new tumors.

Chemical, physical and biological carcinogens can play the role of initiators of carcinogenesis.

Polycyclic aromatic hydrocarbons, nitrosamines, aromatic amines, amino azo derivatives, various aflatoxins, alcohol, alkylating agents, androgens, oestrogens, arsenic, asbestos, phenacetin, and so forth are already well studied in *chemical carcinogens*. The results of such studies have been regularly published

for many years in the IARC Newsletters of the Lyon Agency for Research on Cancer (IARC).

Physical carcinogens may be ionizing or non-ionizing radiation of different wavelengths. Many of them are still only suspected but not proven and are undergoing intensive research.

Biological carcinogens (oncogenic viruses) that have been proven to be such are hepatitis B virus for liver cancer, multiple human papilloma viruses such as type 16 and others. For uteri cervical cancer, HTLV-1 for endemic T-cell leukemia in adults, Epstein-Barr virus in Burkitt's lymphoma and many others viruses - so far more suspected than proven. Other substances can act as *promoters* that are not oncogenic in themselves, but can potentiate the effect of subcarcinogenic doses of true carcinogens. Promoters may be croton oil and phorbol esters therein, saccharin, bile acids, fatty acids, diterpenic esters, phenobarbital, surfactants, and many others chemical agents. Certain carcinogens require mandatory metabolic activation in the body, most often in the liver (less frequently in other tissues) to exert their carcinogenicity. Therefore, there may be large interindividual differences in their carcinogenic effect, depending on the individual metabolic competence of the body - many studies show that it is predominantly genetically determined.

Molecular carcinogenesis, protooncogens, oncogenes and tumor suppressor genes. In normal cells, there are so-called *protooncogens* or cell oncogenes that are very conservative in evolution, and are likely to play a role in regulating normal cell proliferation and differentiation. So far more than 40 protooncogens have been identified in various retroviruses - RNA tumor viruses (many others are undergoing intensive research and demonstration). Under the influence of the described carcinogenic factors, by mutation, chromosomal rearrangement or gene amplification, these inactive protooncogens can be converted into active oncogenes. **Oncogenes** encode the synthesis of various protein products, including *growth factors* from the epidermal, thrombocyte, insulin, fibroblast, hepatocyte, and other families growth factors as well as *growth factor receptors* involved in the regulation of cell proliferation and differentiation, and hence in the growth of tumors. *Much of the oncogenes act by encoding the synthesis of membrane tyrosine-specific protein kinases* - in this way they probably influence the molecular processes of cell signal transmission, which is now the subject of particularly intensive experimental studies.

The protooncogens are classified into five groups according to the functions of their protein products:

- protooncogens for growth factors;
- protooncogens for growth factor receptors;
- protooncogens for cell signal transmitters (transmitters);
- propionogens for transcription factors;
- protooncogens for regulators of apoptosis.

In addition to the described molecular mechanism of carcinogenesis - the activation of protooncogens to active oncogenes, there is also a second major mechanism - suppression of the so-called tumor-suppressor genes (antioncogens), most commonly by point mutations. Between the tumor suppressor genes, p53 is the central site of the antioncogen. It is a nuclear protein with a molecular weight of 53 kd, located on the short arm of chromosome 17. It is called a "guardian of the genome", which causes programmed cell death or arrest of the cell cycle. In many cases of oncological diseases, the p53 antioncogen has been found to have a mutation that has lost its function. These p53 mutations were most commonly observed in adult oncological patients and significantly less frequently in childhood tumors. It has been found that some mutant forms of p53 may activate cells to unrestricted divisions and cause immortalization of the cells.

Other recent anticogens have been found to be RET (tyrosine kinase receptor), WT (a transcription factor) that has been mutated in the Wilms tumor in children, the p16 gene in melanomas, gliomas, bladder cancer and leukemias, NF1 and NF2 in neurofibromatosis, BRCA1 and BRCA2 in breast cancer of women, and others -now "candidate antioncogens".

More than 100 oncogenes and more than 40 anti-oncogenes have been discovered - the description of new ones and the clarification of their functions continues. The enormous difficulties in clarifying the molecular mechanisms of oncogenesis, however, come from the fact that only 6-8 oncogenic diseases, and most of these rares, are caused by monogenic defects. *The prevailing number of over 200 tumor diseases is characterized by a polygene (multiple) nature of the defects*.

The research of oncogenes and anti-oncogenes, the identification of changes in them and the modern molecular methods for their quantification reveal in principle new possibilities for real *molecular prophylaxis, diagnosis and monitoring of oncological diseases*, as well as a new approach to their complex treatment.

Biology of human tumors - general data. Using conventional clinical diagnostic methods, it is now possible to detect tumors with a diameter of at least 0.5-1 cm in which there are about 1 billion tumor cells then the tumor has already undergone 30 consecutive doubling. Numerous studies on tumor biology have shown that distant hematogenic or lymphogenic micrometastases have already been

diagnosed at 50-60% of the oncologists at this time of diagnosis. They can be detected with labeled monoclonal antibodies and the highly sensitive and specific polymerase chain reaction. This basic fact of tumor biology explains why, in such patients, radical therapies with local action (surgical and radiotherapy) are doomed to failure due to the development of recurrence and metastasis, most often with fatal outcome. These data on the early micrometastasis and the so-called *residual disease* (a small number of residual tumor cells after removal of the main tumor mass) have become the theoretical basis for the creation of modern therapeutic approaches for *adjuvant (complementary, prophylactic) therapy as well as for neo-adjuvant therapy*. Adjuvant therapy is administered after radical surgery or radiotherapy (very rarely) and *neo-adjuvant (primary)* therapy before other radical treatments.

Biological characteristics of the individual tumor. *The kinetics of tumor growth* is essential for the prognosis of oncological disease and the prediction of its sensitivity to drug treatment. In tumors with a small "growth fraction", a high "doubling time" and slow growth, such as some nodular forms of non-Hodgkin's lymphomas, so-called aggressive drug treatment, is no-recommended, whereas in other patients with the same diagnosis, but of aggressive clinical course is necessary and more aggressive combined drug treatment. The "growth fraction" or "proliferative pool" of the tumor is formed by the active fission tumor cells, that are in one of the cell cycle phases (G1-pre-syntetic, S-synthetic, G2-postsynthetic and M-mitotic). Tumor and normal cells that are out of the cell cycle, so-called "sleeping cells" or G0 phase cells are generally considered to be mildly sensitive to drug treatment and irradiation. For a variety of reasons, they may re-enter a cycle and thus relapse the oncological disease, but they are usually re-sensitive to treatment (if they have not developed resistance to it). High sensitivity to antitumor drugs generally exhibits malignant tumors with a large "growth fraction" and rapid growth such as Burkitt's lymphoma, uterine chorionepithelioma, some leukemias, lymphosarcomas, and others. Tumors with small "growth fraction" and slow growth are usually more resistant to drug treatment - primary or acquired resistance (eg. stomach cancer, rectum and colon, pancreas, prostate, etc., so-called "solid" tumors). Normal bone marrow cells have a large "growth fraction" and are highly susceptible to antitumor drugs. Therefore, myelosuppression is one of their most common and severe side effects. In fast-growing tumors, the optimal therapeutic effect is achieved by intensive intermittent drug treatment at short intervals, where damaged bone marrow cells recover faster than tumor cells. Conversely, in slowly growing "solid" tumors, due to the same features of tumor biology, an optimal therapeutic effect is achieved by longer courses of treatment with greater intervals between them.

The greater the tumor mass, the smaller the "growth cell fraction", and the lower the sensitivity of the tumor to drug treatment and vice versa. Therefore, in large, advanced tumors, first, the tumor mass should be reduced to the minimum possible by surgical or radiological treatment and then drug treatment is used to destroy the remaining relatively few viable tumor cells. It is now well known that only one or several viable tumor cells remaining after treatment are sufficient to induce a relapse of disease. The so-called adjuvant drug treatment (destroying a relatively small number of residual but viable tumor cells) has been administered to the so-called *residual disease* following radical operative or lesser-radiation treatment. Tumor biology data and experimental therapy indicate, according to most researchers, that *immunotherapy* (treatment with modulators of biological response) *administered alone can only be effective in very few viable tumor cells* (below 100,000 cells or less than 0.1 cm diameter of the tumor), which is also theoretically expected to be effective in a residual disease. Conversely, in large, advanced "solid" tumors or leukemias where the tumor mass is very high, immunotherapy is not only ineffective but can potentiate tumor growth, possibly due to the formation of blocking antibodies, and other reasons that have not yet been clarified.

One of the *mathematical models of the dynamics of tumor growth* is expressed by the *Gompertz sinusoidal curve*. It is characterized by an early phase of slow proliferation, followed by a second phase of rapid logarithmic growth and a third phase of a new delay in proliferation, until reaching a plateau in growth close to the death of the patient. From the cytokinetic point of view, in order to achieve complete destruction of the tumor cells, it is necessary to administer the antitumor drug repeatedly at certain intervals. So far such cell kill has only been achieved in experimental conditions in rapidly growing ascites transplanted tumors in mice, such as lymphoid leukosis L1210, a model of Skipper-Schabel-Wilcox. According to their hypothesis about the logarithmic destruction of tumor cells, this is a first order process in which a constant portion of the tumor cells is destroyed at each subsequent application of the antitumor drug. Thus, in 2-log arithmical destruction of the cells, 99% of them are destroyed, in case of 5-log. destruction, 99.999% of them are killed, and so on. An antitumor effect may occur theoretically in 2 to 5-log. tumor cell destruction by the antitu-

mor drug (monotherapy) or a combination of drugs - most commonly used in clinical oncology as multiple protocols for combination drug therapy. Another cytokinetic model that is closer to human "solid" tumors is that of Norton-Simon.

Heterogeneity of tumor cell populations. Although tumors are likely to have a clonal origin because of the large genetic plasticity and instability of tumor cells, mutations often occur, some of which are inherited and thus several different cell clones are developed in a malignant tumor. Thus, 2 to 4 cell clones are most commonly found in human breast tumors, and more than 20 different cell clones are found in the multiple glioma of the brain, which can be explained by extremely complex brain histogenetics. Some of these cell clones are sensitive to antitumor drugs and/or radiation therapy and are rapidly destroyed, but others are naturally resistant or acquired resistance, and therefore remain and proliferate. Cell clones are also different in their ability to induce angiogenesis, invasion and metastasis. Heterogeneity of tumors also justifies some of the benefits of intensive and high-dose drug treatment, as well as multi-drug combinations with different molecular mechanisms of antitumor activity, to monotherapy (treatment with only one anti-cancer drug that is used relatively less frequently). Heterogeneity of tumor cell populations; drug resistance, especially multiple drug resistance to a large number of MDRs; as well as early micrometastasis, are one of the main reasons for the failure of drug treatment for cancer.

Invasive growth. Metastasis. Angiogenesis. *Invasion and metastasis are complex multistage processes (so-called metastatic cascade)* that sequentially involve the following *relationships between the tumor and the oncopatient's organism*:

1. *Tumor initiation:* during the initiation the carcinogenic effect occurs, activation or depression of oncogenes and rearrangement of chromosomes.

2. *Promotion and progression:* characterized by karyotype, genetic and epigenetic instability, gene amplification of promotional and growth-associated genes, mutation or loss of suppressor gene products.

3. *Uncontrolled proliferation:* includes autocrine growth factors or their receptors as well as receptors for most hormones, for example, for estrogens.

4. *Angiogenesis:* Multiple factors of angiogenesis are activated, incl. known growth factors.

5. *Invasion* of the local tissues, blood vessels and lymph vessels - with the participation of serum chemotractants; autocrine factors of motility; attachment receptors; degrading enzymes; loss of expression of proteinase inhibitors.

6. *Circulating tumor cell suppression and extravasation* - homotypic and heterotypic aggregation of tumor cells occurs through the following steps:

a) adherence to the endothelium - is accomplished by interacting the tumor cell with fibrin, platelets and hemostatic factors, adhesion to receptors of the PGD type;

b) endothelial retraction - involving platelets as well as tumor cell factors;

c) adhesion to the basal membrane - receptors for laminin, thrombospondin and collagen type IV;

d) dissolution of the basal membrane - metalloproteinases, serine proteinases, heparinase, cathepsin;

e) movement - autocrine motility factors and chemotaxis factors have been identified.

7. *Formation of colonies at a secondary site* - receptors for local tissue growth factors; angiogenesis factors; mutation or loss of metastatic suppressor genes.

8. *Avoidance of tumor cells from the protection of the organism and the emergence of resistance to therapy* - resistance to destruction by the macrophages of the organism; to natural killer cells and activated T-cells; unsuccessful expression or blocking of tumor-specific antigens; amplification of drug resistance genes.

Neovascularization is a prerequisite for the local expansion of tumor colonies out of the area, limited by the diffusion of oxygen and nutrients.

Angiogenesis is needed at the beginning and end of the metastatic cascade. Because of the disruptions in the basal membrane, the tumor vessels are permeable, the tumor cells easily pass through their walls and enter the circulation in large numbers - several million tumor cells per day. Studies have found a positive correlation between the number of tumor microvessels in a biopsy material and the patient's fate in prostate cancer, melanoma, breast and ovarian cancer.

The individual stages of the metastatic cascade - angiogenesis, adhesion, proteolysis, motility and proliferation, can serve as new guidelines for the prevention and treatment of oncological diseases. Thus, the antiangiogenic effect of alpha-interferon - suppresses gene expression for angiogenic growth factor, leads to regression of life-threatening hemangiomas in children. Clinically, anti-angiogenic agents, such as the known teratogen thalidomide, also TNP-170, marimastat, angiostatin, and others, are tested. Intensive experimental studies are also devoted to influencing the signaling pathways of the metastatic process - calcium homeostasis, kinase activity, ras-oncogene activation, specific antisense nucleotides, selective cyclooxygenase-2 (COX-2) inhibitors, etc.

Prevention. The origin and evolution of oncolog-

ical diseases is determined by complex interactions between numerous *environmental factors* (carcinogens, cocarcinhogens, anticancerogens, etc.) and *factors of the patient's own organism* (immune system, hormonal status, cell populations, individual metabolic competence, state of mechanisms recovering DNA, genetic predisposition, etc.).

About 80-90% of the disease is caused by environmental factors, most of which are well-known and relate to the lifestyle of modern man. According to the WHO data, the most important part is the nutrition (diet) followed by smoking. They account for about 60-70% of oncooletality together. Relative role is played by reproductive and sexual behaviour of people, alcoholism, occupational risk factors, environmental pollution, excessive use of certain drugs and other risk factors. About 5% of oncooletality is related to professional factors (the classic first description in this area refers to the high incidence of scrotum cancer in chimney sweep, done in 1775 by Percivall Pott in England). For founders, miners, workers in the rubber and textile industry, and shipbuilding, printing, leather, painting, asbestos workers, and others occupations, a higher frequency of individual onco-nosological units is registered.

Based on these onco-epidemiological data, advanced ***primary cancer prevention*** programs have been developed to reduce or eliminate carcinogenic factors. After more than 30 years of activity on a well-organized and powerfully funded *anti-smoking program,* involving the entire society and the President of the United States, there has already been a reduction in lung cancer (a similar reduction has been seen in England and elsewhere developed EU countries).

A relatively new direction in primary prophylaxis of oncological diseases is so-called *pathogenetic prophylaxis*. Hygiene prevention is primarily intended to reduce the risk of contact with various types of carcinogenic agents and known risk factors. It is expanded and supplemented by pathogenetic prophylaxis, which aims to inhibit carcinogenesis in all its stages, with the help of specific diets and chemical agents with anticancer activity. Pathogenetic prophylaxis is targeted at healthy people from the general population or at higher risk populations - e.g. precancer patients, smokers or patients undergoing specific anti-tumor drug therapy (alkylating cytostatics and some other antitumor drugs as well as ionizing radiation are known to have a strong carcinogenic effect and are capable of inducing chemically induced secondary tumors at 2- 10% of cancer patients - most often leukemias and lymphomas, especially in long-term survivors). It is suggested that by *priming prophylaxis and mainly pathogenetic prophylaxis, it is possible to halve the incidence of oncological diseses*.

Secondary prophylaxis of oncologi diseases includes their early diagnosis, emphasizing the particularly important role of detecting precancerous diseases. *Screening programs* have proven to be effective primarily in uterine cervical cancer, where Papanicolau vaginal smears screening has led to a reduction in mortality from the very prevalence of oncological diseases in women by about 70% over the past 40 years. In another, so far limited part of the cancer, screening studies have a controversial location according to the value-effectiveness indicator and are recommended primarily for some relevant risk groups of the population.

Recently, the *genetic predisposition* has been intensively investigated by the body's factors. Over 100 monogenic diseases have an increased risk of developing cancer. In some of the monogenic diseases, the carcinogenic risk is increased by more than 10,000 times, for example in familial retinoblastoma, familial colon polyposis, and oth. Xeroderma pigmentosum describes a defect in DNA repair and hence increased carcinogenic risk. In developed countries, special genetic counseling works on particular oncological diseases, especially when patients are involved in clinical trials. However, their widespread routine implementation in practice is, for the time being, considered insufficiently scientifically and prematurely. Thus, for breast cancer (the most common tumor in women and the most common cause of death among women in the European Union), it is now recommended that genetic counseling should not be done on a large-scale, but only within clinical trials.*

At now known "copy" of human genome (50 terabytes of information), made by the US Government's Human Genome International Project and in parallel and independently by the privately-owned American company Celera Genomics, as well as by the latest methodological achievements of molecular biology for simultaneous measurement of the expression of several tens of thousands of genes *(DNA microarray technology)*, by *so-called oncogenomics and proteomics*, expected a new break in molecular oncology and pharmacogenomics (targeted generation of drugs directed to specific genes) is expected as a basis for future prophylaxis, diagnosis and treatment of oncological diseases.

The latest WHO cancer priority ladder are: tobacco control, infection control, cancer treatment program, effective pain control, early detection

* Genetic research can pose a potential threat to women's personal freedom and safety, which is why legislative changes are needed to protect women from these dangers.

strategy, national cancer registry, healthy nutrition program, updated permanently guidelines, nursing training, national oncology specializing network, clinical trial units, technological renewal of the region, clinical research program, fundamental research program, program for international assistance.

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9.2 MALIGNANT NEOPLASMS OF THE RESPIRATORY SYSTEM

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9.2.1 PULMONARY CANCER

The interest in the **etiology** of respiratory tumors has increased especially after a number of epidemiological studies that indicate that lung cancer is the most common cause of death from malignancies - an average of 25% 35% for men and 15% for women), and among those with occupational hazards - from 3% to 17%.

In 1987, the IARC issued a list of chemical substances and industrial products for which there is evidence of carcinogenicity in humans. According to him, carcinogens for lung tumors are: arsenic and its compounds; asbestos, chloromethyl ethers, 6-valent compounds of chromium, asphalt, coal, nickel and its compounds (oxides and sulphides), coke gases, radon and thorium, vinyl chloride. Additional registers also discuss substances that are likely to be lung carcinogens but with insufficient evidence of this: acrylonitrile, benzidine, beryllium and cadmium with their compounds, hydrazine, chlorophenothane, dibromochloropropane, chloromethan, epichlorhydrin, ethylene dibromide, dimethylaniline, tetrachlordibenzodioxin, etc.

The following are reported as risky professions and factors for the occurrence of malignant lung tumors:

- aluminum production, coal mining and coke gas, with probable carcinogens polycyclic aromatic hydrocarbons;
- production of iron and steel with probable carcinogens polycyclic aromatic hydrocarbons and metal aerosols;
- operations related to the refining of nickel, likely to cause nickel, nickel oxide and nickel sulphide;
- rubber production with possible carcinogenic aromatic amino compounds;
- production of uranium (thoron), iron and non-ferrous metals with probable agents radon and thoron
- production and production of asbestos and asbestos products.
- using arsenic preparations in fruit growing and in the production of copper ore

Histology. Four major histological types of lung carcinomas are:

- squamous cell (epidermoid);
- adenocarcinoma;
- giant cell;
- small cell (type of oat grain).

Squamous carcinomas more often have a central localization associated with hyalous lymphadenopathy. Adenocarcinomas are usually presented as peripheral nodules with involvement of the pleura and the chest wall. Giant cell carcinoma has a large peripheral localized formation with added pneumonitis. In small cell carcinoma, a central lesion with athelectasis is present, and hyalous and mediastinal lymphadenopathy is also common. No histological type is pathological for professional injury - i.e. there is no characteristically dominant histological type in occupational lung tumors.

There is nothing strictly specific about the **clinical picture** of occupational lung tumors. Their clinical picture, as and unprofessional malignant tumors, are defined by the following major syndromes: :

- bronchial-pulmonary cough, haemoptysis, dyspnoea, pneumonia, etc.;
- intoxication: weight loss, anorexia, asthenia, subfebrility, sweating, anemia;
- from squeezing due to tumor growth and invasion of adjacent structures or metastasis: tracheal obstruction, esophageal compression, "vena cava superior syndrome", paralysis of n. recurrens, n. vagus with dysphonia, etc.;
- paraneoplastic syndrome.

In addition to the clinical symptoms of diagnosis, some additional research methods are also essential:

- X-ray methods provide the best opportunity for early diagnosis. The X-ray finding depends on the histological type of the tumor; of its location (central or peripheral); from the stage and the presence of metastasis in the regioneal lymph nodes or in the lungs.
- Cytological examination of phlegm in 60% of cases helps diagnose.
- Invasive methods allow the most accurate di-

agnosis as well as the operability of the malignant tumor. Approximately 65% of cases of lung carcinoma are visualized with flexibel fibrobronchoscopy, but an accurate diagnosis is made by biopsy (including brush) - 90% of the cases. In some cases the diagnosis is beneficial for the transthoracal aspiration biopsy under the X-ray control. In difficult cases, an explorative thoracotomy can also be performed.

- Conventional and computed tomography, as well as a radioisotope assay, is made when the patient is unsuitable for invasive screening. A radioisotope assay with ^{67}Ga-citrate seeks selective accumulation of the isotope in tumor tissue.
- Of the routine blood and biochemical examinations, the BSR, the red and white blood counts, the cytological and biochemical examination after pleurocentesis.

Diagnostic criteria for the professional nature of lung neoplasms. For a more accurate etiological diagnosis, the following criteria are recommended for this type of illness:

- Labour-hygiene, which has to address the issue of occupational risk, i.e. for the presence of carcinogens in the working environment.
- an exposure criterion that corresponds to the assumed exposure duration and the latency period **(Table 1)**.

Organ	Carcinogen	Average, minimum and maximum latency period
Lung	Asbestos	18 (15-21)
	Chromates	15 (5-47)
	Nickel	22 (6-30)
	Ionizing rays	25 (7-50)
	Carbon vapors	16 (9-23)

At low exposure and particularly short latency period, there is little probability of professional tumor etiology. Usually, small exposure prolongs the latency period.

- Epidemiological test (collective test), which is becoming increasingly important especially in the discovery of new occupational carcinogens.
- Localization should be consistent with the prospective experience and literary data. Asbestos carcinoma, for example, tends to multiple locations in the lungs basis, where is also asbestos fibrosis.

Difficulties arise in the combination of professional and non-professional carcinogens. For the same critical organ, the following could be observed:

- if both can cause the tumor, it must be recognized as professional;
- if only the sum of the two injuries can cause the tumor, it is also considered to be professional because the occupational risk is implicated and without it the disease develops;
- if the impact of occupational carcinogens is of minor importance, the adoption of an occupational disease is not justified.

Types of professional pulmonary neoplasms.

A. Pulmonary neoplasms of physical factors and dust. In this group are tumors caused by ionizing radiation, mineral powders - mainly needles and isometric, and some organic powders.

Professional lung tumors of ionizing radiation. Occupational risk exists in:

- miners from the extraction of rare metals. It is known that modern uranium mines are characterized by low dustiness (near to the MAC) under constant emission of α-particles;
- working on the shredding and further processing of uranium ore;
- labtechnicians of radioisotope laboratories;
- working in nuclear power plants;
- radiation risk, albeit less significant, in some mines with increased radioactive background: for non-ferrous metals (eg copper - Burgas copper mines), iron, fluorite extraction, some tunnels, hiding places, quarries and opencast mines (Zeolite).

Radiation risk is due to uranium and thorium that emit the radioactive gases radon and thoron. They act with α-particles that penetrate to a depth of no more than 50 μm, i.e. around the basal membrane of the bronchi.

The clinical picture does not differ from that of another aetiology of cancer. Signs of radiation sickness (adynamia, asthenia, anemia, leucopenia) can be observed, but may be absent. Radiation is thought to increase the relative share of undifferentiated small cell tumors and reduce the relative share of squamous tumors.

In previous silicosis, sudden changes in the general state, the accelerated BSR, rapid changes in the X-ray image, lead to a diagnosis of lung carcinoma. The diagnosis is refined by cytomorphological examination of the phlegm, bronchobiopsic methods and computed tomography.

Professional pulmonary neoplasms of needles dust. The carcinogenic effect of mineral needles is mainly related not to their chemical but to their physical qualities. They have their own mechanism of tumor induction and their carcinogenic effect is related to certain properties:

- the needles should be long and thin - with a length of more than 5-10 μm and a diameter less than 0.5 μm, that is, the ratio of these two metrics is 10: 1 - 15: 1;
- needles should have a pronounced bio-resistance, i.e. to be able to persist in the tissues of the lungs for decades.

Asbestos. The carcinogenic effect of asbestos has been discovered and studied at the earliest. All types of asbestos fibers are considered to be carcinogenic, but crocidolite (blue asbestos) is more dangerous than chrysotile (white asbestos).

Asbestos neoplasms (bronchopulmonary cancer and pleural mesothelioma) are generally associated with pulmonary or pleural asbestosis (fibrosis).

Epidemiological studies by *Doll* and *Breslaw* scientifically demonstrated the causal relationship between asbestosis and lung cancer. Also noteworthy is the Selikoff study - tracked 17,800 asbestos workers for the 1967-1976 season and found 486 deaths instead of the 105-106 predicted.

Pulmonary cancer is the most important asbestosis-related disease that causes 20% of all deaths in asbestos-producing groups. Studies in Bulgaria show that in professional asbestos-exposed patients the incidence of lung cancer is 3 times higher than that of the standardized by sex and age general population.

The synergistic carcinogenic effect of smoking with asbestos dust has been proven. Asbestos particles are believed to absorb benzopyrene from cigarette smoke. Also, here are some additional carcinogens, such as natural mineral oils adsorbed by the anthophyllite and amosite asbestos, as well as chromium and nickel content in chrysotile and crocidolite and iron in amosite and crocidolite.

Pulmonary asbestos cancer develops after a long latency period against the background of diffuse pulmonary fibrosis - asbestosis. Lack of asbestosis reduces the likelihood of asbestos-induced lung cancer. The localization of the tumor is predominantly in the lungs basis, where fibrosis is most pronounced. Histologically, the tumor does not show differences from unprofessional tumors. Diagnosis is supported by biopsy material taken by fibrobronchoscopy. Finding asbestos bodies in phlegm or biopsy material supports massive contact with asbestos.

Mesothelioma of the pleura is a relatively rare tumor - its spontaneous frequency is 1 case per million inhabitants per year *(J. Stumphius)*. The relationship between this tumor and asbestos has been proven by *J.Warner* et al. in epidemiological observations of workers from asbestos mines in South Africa.

All types of asbestos can cause mesothelioma, but especially crocidolite, whose properties make it accessible to the pleura (the more rough fibers are retained in bronchiols and cause usually pulmonary asbestosis or lung cancer respectively). For the carcinogenic effect, only the dose is not critical and therefore there is no relationship between dose and biological response - mesothelioma may develop even after short exposure, but over a long latency period. Unlike asbestos lung cancer in mesothelioma, smoking does not significantly affect its incidence.

Mesothelioma develops from the mesothelial cells of the serous shells, most often from the pleura to the asbestos plaques. It is a thick mass with a thickness of 0.5-1.0 cm and an ivory color that progresses along the pleural layers and interlobar slits. A large mass is created with a rubbery consistency that presses, "crushes" the lung and can grow to the pericardium, chest wall, diaphragm, mediastinal lymph nodes. Metastases in other organs are extremely rare. There are 4 types of histology: tubulo-papillary, sarcomatous, undifferentiated and polygonal, and sometimes mixed and transient forms resembling sarcoma or carcinoma.

Clinical picture - breathlessness that progressively increases. Often there is weight and pain in one chest, which is initially of a grate character, but later becomes strong and constant. General signs of intoxication - adynamia, weight reduction, night sweats, as well as persistent and rapid gathering of pleural hematic fluid are also characteristic. The content of hyaluronic acid in fluid has increased. Although it is not pathognomonic for mesothelioma (also occurs in asbestos plaques and is associated with the accumulation of hyaline in them), it is an indication of asbestos origin of the tumor. Diagnostic value of X-ray and computed tomography is great **(Figure 1)**.

Treatment here is symptomatic only, as the tumor is resistant to radiation and to cytostatics. The outcome is unfavorable - cachexia, severe respiratory and heart failure, bacterial pneumonia.

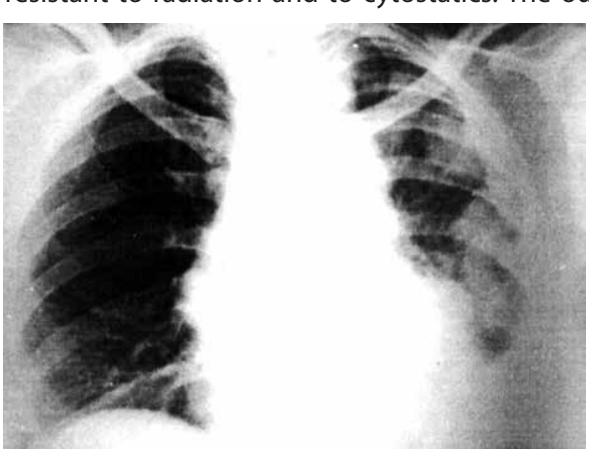

Fig. 1. Mesothelioma of pleura

At present, there is evidence that, besides asbestos and other needles and fibrous minerals, they have a carcinogenic effect. Pulmonary neoplasms have been

described in Turkey from needleloid zeolite material, **erionite**. In the Bulgarian zeolite deposits it is not found, but mainly **clinoptilolite** and the needle-like **mordenite**, which experimentally have less carcinogenic activity. For needles mineral **actololite**, the carcinogenicity was confirmed experimentally.

The carcinogenic risk of synthetic asbestos substitutes - mineral and glass wool - is also discussed. Most authors, though with some caution, believe that there may be an increased risk of lung neoplasms.

Professional pulmonary neoplasms of isometric mineral powders. The carcinogenic action of mineral isometric powders has been the subject of many controversies (in the past prevailed the view that there was no statistically significant increase in pulmonary neoplasms in pneumoconiosis patients).

The most attention is paid to **quartz**, because epidemiological data on the increased risk of lung cancer in exposed to crystalline silica: minerals from iron mines, iron workers, casting cleaners, sandblasters, even ceramics workers, have been highlighted in recent years. The official statistics of Sweden and Canada showed increased lung cancer mortality in silicosis patients. There is also experimental evidence that quartz may be a direct acting carcinogen or co-carcinogen. Three hypotheses are discussed:

- the direct carcinogenic action of quartz;
- silicosis as a basis for developing the so-called "scars" carcinoma;
- quartz as an absorber of polycyclic aromatic hydrocarbons originating from the working environment or from smoking.

Other isometric materials such as hematite, lemonite, grunerite, sandstone, kaolin, dolomite, vermiculite, etc. are also the subject of this study.

Professional pulmonary neoplasms of organic powders. There has been an increased incidence of lung cancer and pleural mesothelioma in sugar cane workers. It is shown here in the powder of biogenic silicon fibers, which are considered a promoter, and as the initiator of tumor growth - polyaromatic hydrocarbons.

The connection between the fine wood dust from tropical and subtropical trees with upper respiratory tract cancer has also been proven.

B. Pulmonary neoplasms of chemical factors

Chloromethyl ethers are one of the most important chemical compounds with carcinogenic risk for pulmonary neoplasms. *W. Weiss* in 1982 described a small epidemic of small cell tumors of the lungs and larynx in young non-smokers. Experimental animals inhaled with chloromethyl ethers showed metaplasia and bronchial epithelium atypia.

Arsenic is associated with convincing epidemiological data with the increase of lung cancer in the production of copper ore, as well as among viticulturists using arsenic sprays. In 163 deceased viticulturists in the Reinach region, 66% of the autopsy confirmed lung cancer.

Nickel (metal) and some of its salts - nickel sulphide (Ni_2S_3) and nickel oxide (NiO), were recognized in 1988 as professional carcinogens. The issue of nickel carbonyl is controversial. It is currently assumed that the risk of lung carcinoma develops only in the nickel refining (purification). **Manganese** in an experiment also showed carcinogenic activity like nickel.

Chromium and especially its 6-valent compounds, have a pronounced carcinogenic effect. Chromotic cancer predominates small cell tumors. There are no specific, clinical or X-ray traits for the professional nature of the tumors.

Polycyclic aromatic hydrocarbons with the most well-known representative **benzopyrene,** are commonly accepted lung carcinogens. An increased incidence of lung cancer has been reported in workers at gas generators, in coke chemistry plants, and in iron production, in asphalt bases, with diesel engine testers. Workers on the electrolysis of aluminum can also be affected by the content of the coal tar in the anode mass. The high tumor burden of rubber workers some authors associate with the use of soot.

Due to the greater aggressiveness of chemical carcinogens, the lag period for lung cancer is shorter - on average 15-17 years.

Treatment. Resection is the most reliable method for the treatment of non-small cell carcinomas. Small chemotherapy or irradiation is used in small cell carcinomas that are contraindicated for surgery.

Survival depends on the stage of the disease and on the histological type of the tumor. The prognosis of squamous tumors is more favorable. Small cell carcinomas have the worst prognosis due to early and extensive metastases.

Prevention. The main purpose of the technical prophylaxis is to eliminate the possibility of exposure to carcinogens by adequate ventilation, use of respirators or other protective devices, as well as by technological improvements for the inclusion of non-cancerous substitutes.

For **medical** prophylaxis, the importance of early x-ray (fluorographic) diagnosis, cytological examination of phlegm in risk groups, and active legal and health-enhancing work on smoking cessation is great.

Labour expertise. Patients with occupational pulmonary neoplasms are 100% disabled and therefore retire from WEMC.

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M. Melamed

9.2.2 LARYNX, NASAL AND LARYNGEAL CAVITIES CANCER

I. Malignant neoplasms of nose and sinus cavities represent 0.5-2% of all malignancies and 6% of malignant neoplasms of the upper respiratory tract. Carcinomas are most common between the ages of 50-70 years. Sarcomas predominate at younger age or in children. Men are more likely to suffer than women - a ratio of 2: 1. The most common malignant tumors of the nose and sinus cavities are carcinomas. Sarcos are observed 10 times less frequently. In terms of histology, malignant tumors are distributed as follows: carcinoma - 86.5%, sarcoma - 12%, other malignant tumors/melanomas, plasmacytomas, etc.) - 1.5%. Of the sarcomas most often occur reticulosarcomas, less often fibrosarcomas, lymphosarcomas, angiosarcomas and sometimes chondrosarcomas, Ewing's sarcoma, Paget's disease, etc.

Etiology. Causes of nasal and cavity carcinomas are known to have pre-existing illnesses - inflammation, tuberculosis, syphilis, scars, and injuries. In the sarcomas, there is sometimes a history of trauma long before the neoplasm appears. Chronic inflammatory processes cause metaplasia of the cylindrical epithelium in the flat and create favorable terrain for the occurrence of malignant tumors.

Morphology. Depending on the histological structure of the carcinomas, there are three main groups - spinocellular, glandular and undifferentiated (anaplastic).

Extremely undifferentiated carcinomas show strongly marked atypia of epithelial cells (even monstrous cell richness) and deletion of the nesting structure. This in some cases makes it difficult to diagnose them correctly and they are even defined as sarcomas.

Flat cell carcinomas are characterized by strong growth and bone involvement in about 1/3 of the patients.

Nasal neuroblastoma - esthesioneuroblastoma (which is relatively rare), originates from the neuroleptic olfactory membrane. Its histological structure is characterized by a certain gestation. The cells are slightly larger than lymphocytes, with scarce cytoplasm and oval or circular hyperchrome nuclei.

From lymphoreticular tumors that can begin to develop in the lining of the sinuses, lymphosarcomas are the most common. However, in severely expressed lymphatic hyperplasia, differentiation of these changes of lymphosarcoma is sometimes difficult, and multiple studies are required.

Plasmacytoma is also a rare lymphoreticular tumor in this area. Its differentiation from a chronic inflammatory process with a wealth of plasma cells

in the infiltration is difficult, and only the availability of other elements in the inflammatory process can help correct diagnosis.

Sarcomas from other connective tissues tumors are also rarely seen in the nose and sinus area. Most often they have a histological structure of fibrosarcoma, hydrosarcoma and mixosarcoma. Characterized by local destructive growth and recurrence.

Malignant melanoma, which also rarely develops in the lining of the nose and nasal cavities, does not cause any difficulty in diagnosing when it is pigmented. In the absence of a pigment, its histological similarity to extremely undifferentiated carcinoma or fibrosarcoma may make it difficult to accurately determine it. Malignant tumors of the nasal and laryngeal cavities provide metastasis in the regional lymph nodes, the submaxillary and the deep cervical nodes. Distant metastases are rare.

Clinical picture. The development of symptoms in malignant tumors of the nose and sinus cavities is related to the localization, spread and histological appearance of the tumor. By localization, some authors distribute the malignant neoplasms of the nasal and sinus cavities in the following way: the upper jaw and the sinuses - 38%, the maxiloethmoid angle - 23%, the nose 22%, the ethomoid bone - 12%, the frontal and the sphenoid cavity - 3%, in the nasal septum - 2%.

In order to properly record the topographic distribution of malignant tumors, a number of schemes have been developed, the Sebileau scheme being the most appropriate. According to her, the malignant tumors in the facial-cranial bones are distributed in lower, middle and upper floors (infra-, meso-, suprarstructural tumors). To the lower floor are the tumors in the alveolar growth, the hard palate and the lower part of the maxillary sinus. The middle floor includes the tumors in the walls, including the orbital and upper-jaw cavity. Upstairs are tumors in the ethmoidal labyrinth, the frontal and sphenoidal sinuses. Limit localization examines the tumors that are detected in the maxilo-ethmoidal area. In a separate group are the primary tumors in the nasal cavities that secondary growth to the sinuses.

The first and the earliest symptoms (both carcinoma and sarcoma) are a unilateral disturbance in nasal breathing due to more extensive exudation and blood supply disorder. There are hyperemia and swelling of the lining of the nose, which cause interference with breathing, smell and headache.

To the sneeze-purulent exudation is added an admixture of blood. In the sarcomas there are frequent and heavy nasal bleeding.

Upon engagement of the upper-back inner segment of the maxillary sinus, the tear duct is pressed, ophthalmic events are determined - exophthaly impaired eye movement, persistent headache, olfactory disorders. Upon lateral growth, swelling of the outer face of the face occurs, and the lateral wall of the swallow can also engage, after back speaded infiltrate. When compressing or infiltration of n. Infraorbital, neuralgia and paraesthesia in the area of the nerve or upper back teeth pain due to tuber maxillar involvement. The initial forms of malignant tumors of the ethmoid, frontal and sphenoid sinus develop in the clinical picture of persistent sinusitis that does not respond to anti-inflammatory treatment. Typical symptoms occur when the tumor passes the sinus boundary - the breakthrough of the hard palate, the incidence of the orbit, the spread of the tumor to the septum, the tuber maxillar growth, and the facial area are signs of an advanced malignant process.

Nutrition disorders, severe frequent bleeding, breathing difficulties, malnutrition and cachexia are also the consequences of an advanced neoplastic process.

Local metastases occur in the area below the lower jaw, along the neck, distant metastases are in the lungs and the liver, and, moreover, the bones and other organs.

Diagnosis. It is difficult in the initial forms when the tumor is small, hidden in the sinus and the symptoms are insignificant. From history, one-sided progressive nose obstruction, sneezing-purulent and sometimes bloody exudate, decreased sense of smell, epistaxis, persist headache, sometimes eye manifestations.

In anterior rhinoscopy, the carcinomas are seen as an uneven, grainy, easily bleeding formation. Unlike carcinomas, sarcomas are tumors with a smooth surface and equal edges. The posterior rhinoscopy provides a view of the nasopharynx and the choanae. X-ray examination plays an important role in determining the exact distribution and destruction of bone barriers.

Biopsy is, in any case, mandatory. In tumors in the cavities a puncture biopsy or a possible trepanation is performed.

Differential diagnosis. It occurs with acute and chronic inflammatory processes, granulomas and benign neoplasms, lues gum, osteomyelitis, polyposis, dental diseases, essential neurinomas and others.

Treatment. For the treatment of malignant tumors of the nose and the sinuses, surgical, radiation, chemotherapeutic methods and combinations thereof are used.

Surgical treatment is a method of choice, but due to small spatial possibilities, it is not always possible to provide for radical treatment - combined treatment - radiotherapy and chemotherapy (in some cases radiotherapy can be pre-operative).

The endonasal surgical procedure is used in biopsy and in small restricted tumors at the front of the septum and the lower extremity.

To remove carcinoma of the maxillary sinus, total or partial resection of the maxilla is performed. Full resection of Dieffenbad is required for middle-class carcinoma. In tumors on the lower floor resection of alveolar growth, hard palate and part of the walls of the sinus are performed. At maxiloethmoidal localization, the Maure or Zange paranasal access is applied. The same method is used for tumors in the nasal cavities or only for the ethmoid bone. When the carcinoma grows in the roof of the ethmoid bone or the back wall of the anterior sinus, Preising access is used. For operative treatment of fossa pterigopalatina, the Mozer method is used with resection of the anterior, lateral and posterior wall of the maxillary sinus. When engaging the skull base, it is necessary to open a dura mater and an additional plate.

Prevention. The primary focus should be on removing carcinogenic noxiousness in aniline, tar, chromium, nickel and oth. productions. Chronic purulent inflammation, especially with mucosal changes - hypertrophy, metaplasia, should be treated. Smoking has a role to play in the changes in the lining of the nose and the paranasal cavities, so it should be limited.

II. Malignant neoplasms of the larynx

Of all malignant tumors in the larynx, 99% are of epithelial origin. Connective tissue tumors - sarcomas, endotheliomas, malignant lymphomas, etc. are rare and are observed in 0.5-1% of patients.

It is reported that the incidence of cancer of the larynx is 1-3% of all malignant neoplasms. The incidence of laryngeal cancer varies from different country: for countries in Europe, it ranges from 0.54 to 8 per 100,000 population. In Bulgaria, the morbidity is from 3.6 to 4.8 per 100 000 population. Men suffer about 25 times more often than women. The most common age of involvement is 40 to 60 years, as in recent years there is a tendency to shift the morbidity to younger age.

Etiology. There are a number of carcinogenic factors, the prolonged effects of which can cause cancer in the larynx - e.g. benzpyrenic components from internal combustion engine fuels, production gases and powders. Over 65% of patients with larynx cancer have occupations associated with inhalation disorders (asphalt, painters, miners, petrochemical, tobacco, etc.).

From NIO studies, 83.5% of patients with larynx cancer are smokers and only 16.5% are non-smokers. Physiological effects of laryngeal cancer also include ionizing rays. Recently, the number of radiogenic cancer patients has risen significantly - from 4 to 9% of secondary cancer from all larynx cancer patients. A certain role is played by changes in larynx - scarring, lues, tuberculosis, and all mucosal processes that are accompanied by hyperplasia and epithelial proliferation. Chronic laryngitis is not precancerous, but it is a predisposing cause. It is essential that larynx precanceroses - keratoses and papillomas - which can lead to cancer (morphological studies show that 20.6% of the carcinomas originate from papilloma and in 22.6% of the patients the larynx cancer is combined with papilloma).

Morphology. At the beginning of the process, there are pronounced seals or plaques, later massive infiltrates, and in advanced development the carcinoma is presented in three well-defined forms - papillary, modular and ulcerative (the ulceration is in most cases secondary). Histologically largest is the number of carcinomas with a structure of spinocellular carcinoma of varying degrees of differentiation - highly differentiated (highest percentage), moderately and poorly differentiated. Histologically they are connected to the flat epithelium covering the true vocal cords or to a metaplastic cylindrical epithelium. In the spinocellular carcinoma group there are two varieties - verrucous and fusiform-cell carcinoma.

Verrucose carcinoma, which is modular or papillary, with a smooth or slightly rough surface, histologically has a papillary device but proliferates the epithelium inwards. Grows relatively slowly and later metastasizes.

Fusiform carcinoma (with synonyms-carcinoma with sarcoma-like stroma, pleomorphic carcinoma, carcinosarcoma, pseudosarcoma) is a relatively rare tumor.

Glandular carcinomas are very rare. They are characterized by infiltrative growth, mainly in the course of nerves.

Carcinomas of the larynx, mainly those with the location of true vocal cords, remain for a long time localized at the site of their appearance.

Laryngeal sarcomas are extremely rare - the number of fibrosarcomas is greatest, but angiosarcomas and chondrosarcomas are also seen.

The lymphoreticular malignant blastomas are also very rare - they may be lymphosarcomas and reticulosarcomas. Melanoma is localized extremely rarely in the larynx and is more pronounced pleomorphism than skin melanoma.

Clinical picture. A division of the larynx of the super glottis, glottis and subglottis region is assumed.

The two-thirds of the larynx are most often covered by the malignant process, the posterior third of the larynx is extremely rarely affected. There are several forms of growth of larynx cancer: monofocal

- from a restricted mucosal site; wall-paper form - primary, on a wide basis; multifocal - simultaneously in several places (multiple outbreaks gradually merge and form a broad-based tumor).

Laryngeal cancer, regardless of its location, develops in two basic forms - infiltrative and exophytic. The most common is the ulcer-infiltrative form, often exophytic-ulcer. The color of the tumor depends on the maturity of the epithelium - the horned epithelium is white, and in the case of the no-horned is dark-brown or greyish-red. The ulceration of the tumor indicates the development of the process and occurs more in the infiltrative forms. Regarding localization and macroscopic appearance, our observations show that infiltrative-ulcer forms are more often found in supra-glottis, and exophytic forms - in glottis (predominantly infiltrative forms are found in the subglottis). In more advanced stages it is difficult to determine the patterns of development due to the inclusion of different parts of the larynx and the occurrence of inflammatory changes.

The early symptoms of malignant tumors of the larynx depend on the localization, size and distribution of the primary tumor. The initial manifestations of malignant tumors of the supraglottide are due to interference with the swallowing function, in glottis - disturbances in the phonation, and in the subglottis - breathing disturbances. With the progression of the disease and the passage of the tumor from one to the other anatomical area proceedings of the swallow, the phonation and the breathing are combined. In very initial forms the symptoms are hardly spotted - mild discomfort in swallowing and feeding, stickness, feeling of a foreign body, changes in the timbre of the voice, annoying cough and breathing disturbances.

Dysphonia is the first and earliest symptom of true vocal cords cancer. At first, dysphonia may be transient but gradually becomes permanent.

Dysphagia is the first symptom of the cancer of the supraglottis and can be expressed with mild irritation, a sense of "foreign body" sticking, painfulness when swallowed.

Dyspnea is the rarest initial symptom (it is characteristic of exophocal tumors that develop in the subglottis).

When the tumor progresses, tumor diffluence, additional inflammatory process, exteriorization, the occurrence of close and distant metastases, intoxication of the body occur. Very common complications such as bronchopneumonia, bleeding from the disintegrated tumor, inability to ingest, difficulty breathing to asphyxia and severe carcinomatous cachexia appear. In a way of growth, laryngeal cancer develops slowly - sometimes for 1-2 years (such growth has a glottic localization - in the case of the supra-glottis, the process develops more quickly).

The mature forms of cancer metastasize less frequently and later than the immature. According to epidemiological studies, the superglottis cancer produces lymphatic metastasis in 40-50%, glottis cancer - 0.4%, the subglottis - from 15 to 45%.

Diagnosis. It is based on medical history data, objective finding, X-ray and biopsy. An anamnesis should also pay attention to the fewest complaints of the patients on the part of the phonation and swallowing - throat irritation, feeling of a foreign body, a slightly hoarse voice or a change in timbre lasting more than 20 days should give rise to doubt about malignant tumor.

An extrinsic view at more advanced stages may reveal laryngeal enlargement or a broken neck configuration. The palpation is done with a bent head, systematically examining the waistline, the area of the Adam's apple, the cricoid cartilage, the anterior and posterior edges of the sternocleidomastoid muscles and the supraclavicular fossae.

The internal view gives the most rich objective data on the larynx status. It is performed through indirect laryngoscopy, direct laryngoscopy and laryngoscopy through an optical laryngoscope and a microscope.

Biopsy is mandatory in all cases (sometimes a thyroidtomy with a subsequent biopsy). Cytological examination of sputum and larynx smears helps diagnose.

Differential diagnosis. It is caused by tuberculous lesions of the larynx, tertiary gummy infiltrates, laryngoceles in the area of the ventricular folds, infectious granulomas, fibromas and polyps, chronic laryngitis, keratoses (pachydermia and leukoplakia), Reinke edema, papilomas, foreign bodies in the larynx.

Treatment. The main treatment methods used are surgical treatment, radiotherapy, chemotherapy, hormone therapy and their combination. Radical healing methods are surgical, ray and their combination. Initially, the elective method of treatment of laryngeal carcinomas is operative.

In single limited variations, an operative treatment is made taking into account the age of the patient. Up to 50 years of age, hordectomy is preferred due to the likelihood of a longer life expectancy. In older patients radiotherapy is preferred because of a low chance of secondary radiogenic cancer.

At supraglottis locations T1 - central and peripheral epiglottis carcinoma, better results are achieved with surgical treatment. In the glottisal T1 localization in the anterior third of the true vocal cord (exophytic differentiated spinocellular carcinoma), the same durable results (90%) are achieved both by chordectomy and radiotherapy.

Primary-stage subglottis cancer is successfully treat-

ed with hemylarringectomy or subtotal laryngectomy.

The operative treatment of T3 along with the radical removal of the tumor is associated with new functional methods for the recovery of the voice and the airway. In this direction, the following operational methods have been implemented:

1. The operation of Serafini, which is a reconstructive methodology after the horizontal resection of the lower floors of the larynx.

2. Subtotal laryngectomy - with some modification of the Ogura, Myodonski methods and real possibilities for restoration of function.

3. Supracricoid functional laryngectomy - a new methodology, (M. Melamed).

4. Operative methods for voice production - the operation of Stafieri, Amatzu.

Prevention. Lung cancer prevention activities should be aimed at ending and eliminating carcinogenic harm in the household, labour and the environment and active treatment of precancerous diseases.

An important role in the overall prevention is the fight against smoking and alcohol use. Also treatment of predisposing moments (chronic rhinitis, sinusitis, laryngitis, etc.) and active treatment with dispensary monitoring of precanceroses.

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9.3 BLADDER CANCER

The disease is the second most frequent among the tumors of the urogenital system. The ratio of male/female bladder cancer patients is 3: 1, with men being the fifth place among all tumors in men.

More than 700 newly discovered cases are registered annually in our country, accounting for 3.6% of all human tumors - also since 1970, the disease has been steadily rising and this trend has increased since 1993. The highest incidence of newly discovered tumors are seen in the highly industrialized countries - the United States, England, Wales, Denmark, etc., and the lowest in the Asian countries. An exception is the epidemiological spread of bladder tumors in Taiwan, which is attributable to the high content of arsenic in drinking water.

Possible cause of the increased incidence in industrialized countries is the higher risk of exposure to industrial carcinogens. It is stated (Matanoski G.M., and Ellciot E.A.) that occupational injuries are the cause of cancer in 15% to 35% in men and 1% to 6% in women.

Etiology. In 1895, the Swiss urologist Rehn first described a high incidence of bladder tumors among aniline dyes. Subsequently, numerous epidemiological and experimental studies confirm the increased risk of developing bladder tumors in the chemical industry.

It has been found that urologic carcinogenesis is caused by aromatic amines: 2-naphthylamine, benzidine, 4-aminobiphenyl, dichlorobenzidine, orthodianisidine, orthotolidine.

To the workers with the highest risk for tumor development are 5 industrial areas: chemical and dyeing, rubber, printing, petroleum processing and leather-dressing. Less risk is experienced by plastic, dyeing, hairdressers, working with organic chemicals and those in laboratories.

There is a certain correlation between the duration of exposure to the occupational environment, the younger starting age of workers and the risk of developing a tumor disease.

Along with aromatic amines, phenacetine, chlornaphazine and cyclophosphamide also come into the group of carcinogenic factors. The increased incidence of bladder tumors in smokers (50% in the male and 31% in the female) is attributable to the excretion of alpha- and beta- naphthylamine in the urine of smokers. Factors that determine the development of the tumor of the urothelium include infections, various instrumental procedures and calculus, causing mechanical trauma.

Pathogenesis. Neoplastic changes in the ureterium result from the so-called "multistep phenomenon". The multiple nature of the transformation process involves the activation of oncogenes as a result of the prolonged action of various carcinogenic factors and the activation or loss of cancer suppressor genes. Tumor suppressor genes regulate cell proliferation and their loss or inactivation results in tumor development and progression. Aromatic amines with a proven carcinogenic effect come into contact with the body through the skin, the gastrointestinal tract and the inhalation route. As a result of high kidney concentration, the urinary bladder is the body exposed to longer duration of carcinogenicity. Significant in this regard is the increased percentage of localization of tumors in the trigonum area.

A major barrier to the metabolism of aromatic amines in carcinogens is the liver where they either inactivate or become urothelial carcinogens. The inactivation of aromatic amines is accomplished with the enzyme system of N-acetyltransferase. Conjugated N-hydroxy metabolites can be deconjugated by beta-glucuronidase in urine at optimal pH and release active carcinogens interactioned with urothelialcell. In the presence of acid urine, N-hydroxylamines damage cellular DNA, which is a major prerequisite for the occurrence of carcinogenesis.

Morphology. Bladder tumors in 98% of cases are present in epithelial tumors, of which approximately 90% are transient-cellular. Less common are adenocarcinomas (2% of cases), squamous cell carcinoma (6-8%), non-differentiated small cell carcinoma-like pulmonar carcinoma (less than 2%) and mixed tumors or a combination of transient cellular, glandular squamous and undifferentiated carcinoma (in 4-6% of cases). Rare non-epithelial tumors include: phaeochromocytoma, choriocarcinoma, lymphoma and various mesenchymal tumors.

Most commonly, transient cellular tumors are presented as papillary exophytic formations and, less often, as solid or ulcerative. Carcinoma "insitu" is characterized as an anaplastic epithelium whose apparent manifestations can often be mild edema, elevation and redness of the bladder mucosa. The average period in which carcinoma "in situ" goes into invasive carcinoma is 26-33 months.

Based on the degree of cellular atypia, nuclear abnormality and the number of mitoses, transient cellular tumors are graded ("grading"). There is a clear correlation between tumor grading and the incidence of tumor invasion, progression and recurrence. Tumors with a high degree of differentiation (Grade I) are characterized by 10 years of survival in 98% of cases, while survival in undifferentiated (Grade II-III) reaches only 35%.

Clinical picture. In the clinical manifestation of the disease, hematuria dominates - in 85-90% of cases, most often painless and macroscopic. In a few patients, it may be accompanied by manifestations of dysuria.

In advanced cases, the manifestations of the disease include: edema of the limbs in venous and lymphatic obstructions, bone pain in bone metastases and abdominal pain in retroperitoneal metastases or ureteral obstruction.

Urine testing in most cases (except for macroscopic haematuria) indicates the presence of erythrocytes. Concomitant infection can also cause pyuria.

In profusion and prolonged haematuria, blood testing may indicate the presence of anemic syndrome. In cases of urethral obstruction, azotemia may be expected.

Urine cytology is important both at the beginning of the diagnostic process and in the course of post-treatment follow-up. Flow cytometry is diagnosed in 80% of cases. Investigation of blood grouping agents (AHVs) in the presence of tumors demonstrates the lack of antigenicity that correlates with the tumor stage, extent and frequency of progression.

Intravenous urography may indicate a defect in filling and unilateral or bilateral ureteral obstruction. There are similar abdominal ultrasound capabilities. Intracavitary ultrasound can provide useful information about the depth of the process in the muscle wall. Computed tomography (CT) and nuclear magnetic resonance imaging (MRI) inform about the extent of muscle invasion and the presence of enlarged lymph nodes in the pelvis.

Pulmonary and bone metastases are diagnosed respectively with lung Ro-graphy and bone scintigraphy.

The cystoscopy and biopsy confirm the final diagnosis. Biopsy through transurethral resection enables the extent of tumor bladder invasion to be determined.

Prognosis. The prognosis of superficial bladder tumors with the use of treatment and prophylaxis is very good - with a 5-year survival in more than 90% of cases.

The prognosis for invasive tumors is determined by the presence or absence of a metastatic disease. The five-year survival rate of invasive tumor patients is 40-50%. In the presence of lymphatic metastasis, the five-year survival after cystectomy is 15-20% (in patients with micrometastases in lymph nodes - 35-40%). In case of surrounding organs, the 5-year survival after cystectomy is below 10%.

A determining factor in the **treatment** of bladder tumors is the stage of the disease. The usual treatment of superficial tumors is transurethral resection (TUR) with subsequent intravesical BCG vaccine or cytostatics (Thiotepa, Mitomycin, Epirubicin) prophylaxis for relapses.

In situ carcinoma treatment includes transurethral resection and intravesical BCG vaccine. In more advanced cases and those not affected by BCG vaccine therapy, it may be necessary to perform radical surgical treatment - cystectomy.

Partial resection can be performed in 6-8% of cases with small invasive tumors with a possibility of removal in a healthy tissues and non-metastatic process.

Radical cystectomy, with or without preoperative radiotherapy, gives serious chances for long-term survival of patients without lymphatic metastases. Over the last decade, combining different cytostatics lead to increased results in metastatic cases. The use of M-VAC chemotherapeutic mixtures (Methotrexate, Vinblastine, Cisplatin), CIS CA (Cisplatin, Adriamycin and Cyclophosphamide) and CMV (Cisplatinum, Methotrexate and Vinblastine) results in an objective affect in 50-70% of the cases.

Prevention. The main thing in preventing professional bladder tumors is to take measures restricting the exposure of workers to carcinogens. These measures include the use of highly developed industrial technologies with maximum safety, the use of protective clothing and regular control of the working environment.

Higher risk productions use screening for workers - control studies for urine cytology, flow cytometry and blood grouping (AVG) can contribute to early detection of the disease.

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9.4 MYELOPROLIFERATIVE DISEASES

Myeloproliferative diseases (MPD) are malignancies that originate from malignantly transformed bone marrow stem cells. Due to the varying passing of malignant progression in the clinical development of the disease, there are differences. Two major groups are identified - **leukemias** (originating from myeloid stem cells, i.e. myeloproliferative diseases (MPDs) with acute, subacute or chronic course - **Table 1**) and **lymphomas** (arising from malignant transformation of lymphoid stem cells - bone marrow or peripheral lymphatic organs).

Etiology. MPDs are one of the diseases in oncology with the clearest etiological noxae, pathogenesis and predisposing factors. In leukemogenesis **external factors** found in the environment have been proven: radiation, chemical mutagens, drugs (alkylating cytostatics), mycotic factors in agricultural production.

Ionizing radiation is the earliest established leukemogenic factor and the most common. The first observations were on radiologists from 1930 to 1950. Radiologists who work with sources for healing purposes have 9 times more neoplasms. The emergence of MPD is after about 5 years of professional exposure, with the peak being between the 5th and the 9th year, but also described after 35-40 years. Among Japan's atomic bombing survivors, there is a slow increase in chronic MPDs and a dose and epicenter distance dependence. Workers in uranium mines and accidents in nuclear reactors as well as after radiotherapy of bones (for example Bekhterev's disease) have a definite increase in CMPD. At present, despite improved radiation protection, there is a risk of MPDs for patients with other neoplasia who are receiving radiation therapy, especially in conjunction with alkylating cytostatics. In all these cases genotoxicity is due to free radicals. The higher risk should be noted at lower doses, but given for a long time.

Chemical agents are a large group of leukemogenic factors, the most dangerous of which is benzene and its derivatives. Long-term contact with them in the chemical, drug and manufacturing industries leads to six times the incidence of MPDs, and exposure for more than 5 years - the risk increases 21 times. Benzene is believed to block proliferative processes in the bone marrow (diagnosis of so-called benzene myelopathy) by colchicine-like action on the polymerization of tubular components in the tubular apparatus for the mitosis process. Mutagenic action is also found for a number of agricultural products - DDT, lindane, hexachlorocyclohexane and others.

The complicated leukemogenic process is facilitated by the involvement of **internal factors**, most commonly presented as chromosomal diseases: Down's syndrome (trisomy 21), Klinefelter syndrome and Fanconi syndrome. There is a higher incidence of MPD in patients with congenital immune deficiency (Louis-Barr syndrome - teleangiectasis ataxia, Chediak-Higashi syndrome, Wiscott-Aldrich's IgM deficiency syndrome).

Pathogenesis and classification. Although somewhat conditional, the emergence of MPD occurs in three phases (with varying duration over the years), with only the end (third) phase being clinically recognizable and conducting treatment.

In the first phase (tumor initiation), molecular changes in the genetic apparatus of target cells in the bone marrow (stem myelopoietic cells - "CF2L-GEMM" - polypotential) develop. Changes in certain genes (oncogene activation), alteration in RNA transcription, suppression and mutation of regulatory genes (p53, p14, p15, INK gene RB, etc.) have been established (including in experimental lines), changes (mutations) in receptor configurations and activation of enzymes (caspases) with apoptotic disorders - e.g. acceleration in myelodysplasia or delay in myeloleukaemia of programmed cell death. A particular site for tumor cell development has disturbances in the synthesis of protein molecules from controling check-point genes such as the CHN 2 gene.

The discovery of changes in the first phase of leukemogenesis began in 1960, when the Nowell and Hungerford translocation between 9 and 22 chromosomes. It has been established that two oncogenes - BCR and ABL - are fused, and a new RNA is created from which at least three types of proteins with tyrosinekinase enzyme activity are created. Enzymatic stimulation of the granulocyte clone proliferation, it expands and myeloleucosis develops. Such mechanisms operate in acute leukemias, myelodysplasias and other MPDs.

In **the second phase of leukemogenesis (promoter)** mutated cells form a colony (clone) and overcome the immune barrier. Branch expansion is stimulated by growth factors (cytokines), hormones, interleukins and lymphocyte blocking factors. An-

Table 1. CLASSIFICATION OF MIEOPLIPERATIVE DISEASES *

Type MPD	Symbol/s	Frequency
I. Acute myeloblastic leukemias	**AML / AML**	
- Undifferentiated	AML-Mo / AUL	5%
- AML without maturation	AML M1	15%
- AML with maturation	AML M2	30%
- Promyelocytic	AML M3 / APL	10%
- Myelomonoblastic	AML M4 / AMML	20%
- Monoblastic	AML M5 / AMoL	15%
- Erythroleukemia	AML M6 / AEL	3%
- Megakaryoblastic	AML M7 / AmegL	2%
II. Subacute MPD / primary myelodysplasias	**SMPD; PMDs**	
- Refractory anemia increasing	I type / RA / RA	20-30%
- RA with pathological sideroblasts (sideroblastic)	II type / RA / RA	5%
- PA with myeloblast increasing	III type / RAW / RAEB	30%
- Chronic myelomonocytic leukemia	IV type / CMMML / CMML	30%
- RAM in transformation	V type / RAWM / RAEB	25%
III. Chronic myeloproliferative diseases (CMPD)		
- Chronic Myeloleukemia (leucosis)	CML / CML	
(chronic granulocytic leukemia)	HLG / CGL	
- Essential thrombocythemia	ET / ET	
(megakaryocytic leukemia)	CmegL	
- True polycythaemia	IP / PV	
- Di Guglielmo syndrome	DiG	
(chronic erythroleukemia)	HEL	
- Myelofibrosis (osteomyelofibrosis)	MF / OMF / MF	

* In addition to the FAB (French-American-English) classification, there is a newer version of the WHO since 1997. Including cytogenetic and phenotypic data - very detailed and practically complicated.

titumor immunity (both cellular and blocking antibodies) is an essential precondition for the duration of this phase, respectively for rare cases of "self-healing". Tumor clones in the promoter phase only in recent years can be identified by the so-called tumor markers (molecules of tumor origin that can be identified and monitored in blood serum). This allows for very early screening prophylaxis of tumors.

During **the third phase (tumor progression)**, cell mass increases to a degree recognized by imaging and invasive methods. It is generally believed that this is possible in 10^8 tumor volume and quality medical diagnostics. This may highlight the difficulty of unassigned increases in undifferentiated cells in the bone marrow (at 10^8 cells in MDL in the myelogram accounts for about 4% myeloblasts). However, there is no difficulty in an advanced process - 10^9 cells are up to 10% myeloblasts, and at values above 20% the tumor volume is 10^9-10^{10} cells, i.e. the weight of the tumor mass is already above 10 g. During this phase are also the non-random chromosomal abnormalities in bone marrow and peripheral blood cells. For example, in AMLs there are t (8; 21) and t (15; 17) and many others, for MDS there are deletions usually 5; 7; 8; 11; 20; chromosomes, in HMPD is the typical Philadelphia chromosome - t (9; 22).

Knowledge of the quantitative side (leakage phase) of leukemic processes is the mainstay of modern quantitative hematology and is a guideline for:

- planning and conducting the initial (inductive) antileukemic (cytoreductive) treatment by polychemotherapy and/or bone marrow transplantation;
- evaluation of the effectiveness of the treatment - resp. ineffectiveness due to drug resistance (timely change of treatment scheme);
- establishing recovered hematopoiesis and minimal leukemia cell count, i.e. a state of remission ("minimal residual disease"), which should be maintained for years and is the basis for cure;
- establishing timely recurrence of the disease re-conducting intensive cytoreductive therapy;
- in screening studies of endangered healthy subjects for early detection of dysplastic changes and assessment of leukemogenic risk.

Epidemiology. In Bulgaria, in 1996, the incidence of leukemia was 11.5 per 100,000 people - 14.1 for men and 8.9 for women (the most incomplete information on subacute MPD - myelodysplasia, but MDS at least 8 times more than acute leukemias). There are age, racial, geographic and professional characteristics and deviations from the average data. Despite these deviations, statistics show a very stable morbidity rate during the 1981-1996 period.

Clinical picture. The three types of MPD show both general clinical manifestations and differences in acute, subacute or chronic course. The basis of the symptoms is the rate of clonogenic malignant cell proliferation with the whole cascade of humoral effects, the reduction of normal hematopoiesis with consequences of decreased bone marrow population function and associated complications, and the phenomena of suppressed immune responses. Here are the so-called "Major symptoms," predominantly for acute leukemias and so-called " "Small

symptoms" that occur in all MPD, especially at the beginning, and are more common in subacute and chronic MPD.

Pathogenesis interferes with and generates certain complaints of tumor populations that initiate narrowing of normal hematopoiesis and cytokine production, tumor necrosis factors, blocking factors, and creating a humoral imbalance. Of these complex intracellular relationships above a certain threshold (determined by tumor volume), the uncommon but very important **common symptoms** of MPD include weakness, weakness and decreased physical activity, loss of appetite, decreased body mass, night sweats, bonealgies, abdominal pain of enlarged visceral organs. Humoral imbalance and cytokine production above the needs are most often manifested in the so-called **"Paraneoplastic syndrome** - subfebrilitis, skin changes, adynamia, weakness. There is also a **specific symptom** - bone pain with periostal and widespread character; skin infiltrations (leukemias) throughout the body; skin and mucous bleeding similar in time, buco-pharyngeal and gingival syndromes of necrotic nature; spleen infarcts (such as abdominal dramas); obstructive or haemolytic jaundice, initial anemic organ disorders - CNS, eye scotomas, cardiac symptoms, respiratory failure; renal and endocrine complaints due to leukemic infiltrates; complaints of hyperleukocytosis - hyperviscosity syndrome of the CNS, eyes, heart. Often the initial manifestation is from hemorrhagic or inflammatory organ processes. Symptoms and frequencies of the various MPD are summarized in **Table 2**.

In the assessment of **laboratory data**, some factors, which are also the basis for standard diagnostics adopted in the developed countries' practice **(Table 3)**, should be considered:

- they are mandatory both at the beginning of the diagnostic process and in the differential diagnosis And the final decision must be optimal in volume and include classical cytological, cytochemical, immuno-phenotypic, cytogenetic and molecular-biology tests;
- some of the indicators (quantitative) are very variable and should be carried out frequently;
- interpretation requires specific haematological preparation:
- adequate treatment is associated with standardized laboratory control of cytoreduction, requiring further studies on the efficacy of the therapy (cytology, oncogenes by PCR

Tab. Symptoms in acute, subacute and chronic MPD

Type of MPD / Syndrome	Frequency	Pathogenesis
I. ACUTE LEUKEMIAS		
Astheno-adynamic	95 %	Anemia + toxicose + cytokines
Anemic	92 %	Induced erythropoiesis
Hemorrhagic	80 %	Thrombocytopenia + hypocoagulation
Feverin	75 %	Infections + paraneoplastic expression
Infections	45 %	Granulocytopenia + Immunodepression
Neurological	25 %	Leukemic infiltrates + anemia
Osteoalgic	10 %	Periostal infiltrates
Splenomegaly	70 %	Leukemic proliferation
Hepatomegaly	55 %	Leuk. Infiltrates + toxicosis
II. MYELODYSPLASIAS		
Anemic-asthenic	80-90 %	Impaired erythropoiesis
Adynamia	70 %	Analytic aggression
Infections	25-40 %	Dysgranulocytopoesis, -penia
Hemorrhagic	20 %	Distrombocytogenesis
Hepatosplenomegaly	5 %	Secondary MDP's complications
III. CHRONIC MPD (myeloleucosis, polycythaemia, thrombocythemia, myelofibrosis or osteomyelofibrosis, Di Gullielmo syndrome - chronic erythroleukemia)		
Weight (abdominal pain)	95 %	Organomegaly-splenomegaly and heart attacks
Feveriness	60 %	Paraneoplastic syndrome / infections
Anemic	48 %	Pressed erythropoiesis
Night sweats	35 %	Paraneoplastic syn. + TNF *
Consumative	30 %	TNF + increased exchange
Osteoalgic	10 %	Osteoporosis + infiltrates
Thrombosis	20 %	Hyperthrombocytosis
Hypervsceral (CNS, heart)	20%	Hyperleukocytosis
Splenomegaly	95 %	Leukemic proliferation
Hepatomegaly	75 %	Infiltration + hepatitis
Lymphadenomegaly	10 %	Extramedular haemopoiesis
Skin leukemias	5 %	Leukemic infiltrates + necrosis
Priapism	3 %	Thrombosis + pens infiltrates

* TNF - tumor necrosis factor (cytokine)

Tab. 3. DIFFERENTIAL DIAGNOSTIC CRITERIA FOR MPEs

Parameter	Chronic MPDs *					Benign leukemoid reaction
	CML	PV	ET	MF	DiG	
I. HEMOGRAM:						
– Hemoglobin	–	+++	н	–	–	–/+
– Leukocytes	++/+++	+	+	+/++	+	+/++
– Platelets	+/++	+/++	+++	–/+	–	–/+/N
– DBP**	leu.	leu.	leu./N	N/leu.	N	leu.
– GAP*** – index	zero/–	+	N/+	+++	N	N/+
II.MYELOGRAPHY:						
– Celluritisy	++/+++	+/++	+	„Dry puncture“	++	N/+
– Populations:						
Granulocytes	+++	+	+	–/+	N	+
Erythroblasts	–	+	+	–	+	+
Megakaryocytes	+/–	N/+	++/+++	+/–	–	N
III. ORGANIC EVENTS						
– Splenomegaly	+/++	N/+	N/+	++/+++	N/+	N/+
– Hepatomegaly	N/+	N/+	N	N/++	N/+	N
– Lymphadenopathy	–	–	–	–/+	–	–/+
– Haemorrhages	+	+	+/++	–/+	+	–
– Thrombosis	–/+	–/+	+	–/+	–	–/+

Note: * the names of the chronic MMPs listed in table. 1; ** DBP - Differential Blood Pressure; *** GAP - granulocyte alkaline phosphatase index; Quantities symbols: "-" - Missing / Reducing; "+, +++ +++" - moderate, intermediary, significant increase; "N" - norm.

methods, residual resting molecular molecule probes G_o through so-called FISH methods).

A special place is under the control of the two main processes: tumor (leukemic) proliferation and cytoreductive polychimotherapy. The control is based on quantitative cytological examinations of the bone marrow (every 14 days - the time to generate mature cells from normal stem precursors), peripheral blood (reduction in the percentage of malignant cells) and regenerative processes - quantity and qualities of restoring normal populations (granulocyte, erythroblast, megakaryocyte and immunocompetent cells - lymphocytes and plasmocytes) in myelogram and hemogram. Retaining the malignant population is the first sign of primary or emerging resistance and is a motive for changing or intensifying treatment. In the case of effective treatment and minimization of the malignant population ("remission"), there is a reason to determine the chance of cure, which includes and bone marrow transplantation (the possibilities and access of the patients for this difficult and expensive treatment is not, however, massive and that is why in all countries the main treatment Is poly-chemotherapy). Polyhemic therapy has achieved the so-called "minimal residual disease" i.e. persistence of a minimal number of leukemia cells and restored (regenerated) hematopoiesis with complete recovery of clinical health and working capacity. In about 30-40% of patients, such MPD remissions can be maintained for years.

Treatment. The principles of anti-leukemia therapy are:

- **aggressive initial ("inductive")** therapy for maximal eradication of bone marrow cloning cells and resp. in organs with appropriate protection from complications (infections and haemorrhages);
- the repetition of the inductive courses at the beginning is maximally compressed i.e. **intensive application** of the adopted treatment protocol;
- **combination of several cytostatics** is mandatory and phase-dependent (mitogenic inhibitors) and phase-independent (for interphased cells in "calme" - G_0 phase).
- **substitution therapy and anti-infectious drugs** should be included early in the course of treatment to protect patient from the often fatal complications;
- **psychotherapy** should prepare the patient and his/her relatives for prolonged and difficult anti-tumor therapy.

In acute MPDs (acute myeloleukemias = M_0-M_7 of Table 1), the major drugs are Cytosine-arabinoside (antimetabolite of pyrimidine) acting on the synthesis mitotic phase, anthracycline preparations (Daunorubicin or Zavedos) that intercalate into the DNA and damage it and purine antimetabolite Thiogwanin, Lanvis) acting on the S-phase of fission cells by suppressing the synthesis of purine and hence DNA and RNA synthesis. Promyelocytic leukemia (M_3) required specific treatment with retinoids (ATRA). Once a remission has been achieved, the so-called maintenance treatment for 2 or 3 years with purine antimetabolites - mercaptopurine (purinetol) and methotrexate (a competitor of folic acid in the synthesis of thymidine and purine).

In myelodysplastic syndromes (subacute MPD),

treatment is still ineffective to stop the transformation to leukemia. It starts with substitution therapy (platelet and erythrocyte concentrate against cytopenias) and stimulates therapy with growth factors that affect granulocytopoiesis and thrombocytopoiesis (against anemia - with erythropoietin), i.e. a cocktail of inducers to activate the stem cell compartment. In type I and II MDS, this therapy is basic - sometimes in the type II assisted and chelates (desferal) to release from hemosiderine cumulation. In III, IV, and V types, additional effects are assessed with: anabolic hormones, retinoids (Vesanoid), vit. D_3 (Calcitriol), Vestatin, alpha-interferons (Roferon A, Intron A). In IV and V type with transformation, polychemotherapy is performed as in acute leukemia (M1 or M5). Ineffective chemotherapy is the cause of bone marrow transplantation - with good effect in 40-60% of patients.

Treatment of chronic MPDs is specific for each.

In chronic myelogenous leukemia, primary treatment includes hydroxyurea, alpha-interferon and cytosine-arabinoside, and bone marrow transplantation (made in the chronic phase - early, giving 45% cure for the first 5 years). Excellent results have been reported for Glivec, which blocks adenosine triphosphate receptors and successfully stops the proliferative leukemia process in over 75% of patients.

Essential thrombocythaemia is treated with cytostatics, hydroxyurea, melphalan and successfully with alpha-interferon. Combination with antiplatelet anti-aggregates (aspirin, antistenocardin), and the latest - for example, Anagrelid.

Treatment of true polycythaemia aims at reducing blood viscosity and suppressing the hyperaphinity cell branch to erythropoietin. A combination of systemic bleeding, hydroxyurea, bisulphonates and, in some cases, radioactive phosphorus (^{32}P) is used. Antiaggregants are mandatory.

Chronic erythroleukemia (Di Gugliemo's syndrome) is treated according to the rules of granulocytic leukemia in the chronic phase.

Myelofibrosis (myeloosteosclerosis) is difficult to treat. The healing complex includes: replacement therapy (erythrocyte concentrates); medicamental treatment (hydroxyurea, alpha-interferon against megakaryocytic proliferation, gamma-interferon against overproduction of collagen, testosterone, cortico-hormones) and symptomatic treatment of splenomegaly (surgery or fractional radiotherapy).

The prognosis of these malignancies is pessimistic without treatment and with increasing optimism in modern treatment, including bone marrow or stem cell transplantation (isolated from peripheral blood or umbilical cord and placenta of a newborn).

In acute MPDs without treatment, the disease occurs within 1-2 months. Summarized data show that in polychimiotherapy, up to 2 years experience in 60-80% of patients, but up to 5 years - 30-35%. Young people have a significantly better prognosis - over 80% get remission, while in patients over 70 - 30% they get remission. The average duration of the first remission is 12-25 months, and the follow-up is shortened. Significantly worse is the forecast for so-called secondary leukemias (occurring after anticancer treatment of other neoplasias). This happens after a period of 5-8 years and often has primary drug resistance.

The prognosis of myelodysplasia differs in type I and type II - occurring for years with substitution therapy only, and III, IV and V type - within 12-24 months, there is a transformation into acute leukemia.

Chronic myeloproliferative diseases have been ongoing for years.

Prevention. The main purpose of preventing damage from **ionizing radiation** is to minimize the absorbed dose. Prophylactic events include a wide range of measures: organizational, medical, health-education, architectural and planning, etc. The aim is to ensure that they do not exceed the norms (annual dose limits, annual radionuclide inputs, and annual average allowable concentration) when working with ionizing radiation.

For exposure to **chemical agents** (benzene and its derivatives), prophylactic measures provide for control to assess levels of toxic substances in the air in the working environment and compliance with the MACs. If possible replace toxic substances with less toxic.

Conducting prophylactic medical examinations (preliminary and periodic) and protective professional nutrition - diets A, E, according to the active noxae.

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9.5 MODERN APPROACHES TO CHEMOTHERAPY OF ONCOLOGY

One of the major unresolved problems of clinical drug therapy for tumors (classical chemotherapy), which significantly limits successful treatment, along with early micrometastasis, high heterogeneity and genetic instability is the drug resistance of tumors (natural or acquired). Despite the significant recent success of oncology, more than 90% of the oncology mortality is associated with some form of resistance to treatment. This is largely due to the non-selective genotoxic (common cytotoxic effect on all rapidly proliferating cells in the body) of the majority of existing anti-cancer drugs. With the accumulation of enormous numbers of molecular biology and oncology data over recent decades and the discovery of many new targets in the complex multi-stage regulation of cell proliferation and differentiation, it has been possible to create a fundamentally new approach to the treatment of cancer - targeting drug therapy that has a high degree of selectivity on tumors without genotoxic and general cytotoxic action.

Oncodiseases is generally characterized by impaired control mechanisms of cell proliferation, differentiation and apoptosis, heterogenity and micrometastasis, enhanced angiogenesis, invasive growth, and the like common characteristics.

Global trends are towards a gradual increase in morbidity from most oncological diseases, with a slower increase in mortality and the possibility of turning oncodiseases into chronic, treatable diseases in developed countries.

Contemporary treatment of malignant tumors is complex - it includes the three basic healing methods: surgical, radial and medicinal.

Drug treatment for malignant tumors is ***systemic* and includes cytostatic therapy (chemotherapy in the narrow sense), hormone therapy, target therapy and other types of biological treatment.**

CYTOSTATIC (CHEMO-) THERAPY

Clinical chemotherapy of oncology disorders in the 20th century is dominated by **genotoxic drugs with non-specific cytotoxic action** on all rapidly proliferating cells - normal and tumorous. The first practiced antitumor drug, the action of which is based on this approach, was discovered by accident. The use as a warfare poison mustard gas, used during the First World War, causes suppression of the nervous activity through its interaction with the myelin envelope of the nerve cell and myelotoxic effect. These effects initiate clinical trials as an anticancer agent in patients with lymphomas.

At the beginning of the 20th century, the effect of vitamins was identified as low molecular enzyme cofactors, and in 1946, with the determination of the folic acid structure, light was cast on their role in many diseases. Clinical studies with the folic acid analog aminopterin resulted in significant inhibition of acute leukemia in children.

The relationship between these two compounds with radically different chemical characteristics and biological activity was also found in their ability to damage DNA. For the antitumor agent, mustard gas was found to chemically interact directly with DNA, causing the so-called alkylation of the molecule while the antimetabolite aminopterin disturbs the DNA biosynthesis and destroys it by the so-called antimetabolic action. The massive creation of new cancer drugs that break the integrity of the genetic material of the cell begins and every two years at least one new drug has entered the clinical practice.

Mitosis inhibitors. The colchicine - plant substance, isolated from autumn crocus, is one of the first to be tested on mouse tumor models. It has been found to retain the cells in the mitosis process and to demonstrate a new way of initiating destruction of the genetic material - by disrupting its even distribution in the daughter cells during mitosis. Despite its effectiveness, colchicine exhibits unacceptably high toxicity to be administered as an antitumor drug. Therefore, interest was directed towards so-called Vinca - alkaloids isolated from the plant Vinca rosea. The two alkaloids vincristine and vinblastine, as well as their semi-synthetic derivatives

vinorelbine, vindesine and others, and now play a role in the treatment of a number of oncology disorders. Studies on karyokinetic spindle inhibitors have led to the discovery of a new class of compounds isolated from the yew plant (Taxus baccata). Thus, paclitaxel in its biological action is principally distinguished from vinca alkaloids by enhancing rather than inhibiting the assembly of the microtubules of the karyokinetic spindle. Paclitaxel and docetaxel (its semisynthetic derivative) have a significant place in modern anti-tumor therapy, as one of the most powerful therapeutics.

DNA-interacting drugs. Mustard gas derivatives have provided the basis for the synthesis of a large number of clinically applicable derivatives, such as melphalan, cyclophosphamide and dozens of others. They exhibit their antitumor activity by alkylating the macromolecule of DNA. A great number of natural products such as bleomycin and others also interact chemically with DNA. An important role in the treatment of cancer among the DNA-interacting compounds plays cisplatinum. Platinisation of DNA has become a new way of disrupting its structure and has led to the creation of new platinum derivatives with reduced toxicity and broad antitumor spectrum.

DNA replication inhibitors. After the clarification of the structure of DNA, it was quite natural for the synthesis of analogs of the bases that make up this macromolecule. They were suggested to exhibit antitumor properties by disrupting the replication process. Such analogs are the thymine analog 5-fluorouracil and the purine analogs 6-mercaptopurine, 8-azaguanine and the like. The cytotoxic effect of aminopterin and methotrexate is based on their ability to inhibit the enzyme dihydrofolate reductase, which converts deoxyuridine monophosphate to thymidine monophosphate. The so-called "death in the absence of thymine," which occurs in bacteria that have lost the ability to synthesize DNA in the absence of this base, which also occurs in animal cells. This fact has begun a new class of biologically active substances - antimetabolites, which have already been created on a rational rather than empirical basis. Once all the individual enzymes involved in DNA replication have been identified, it has been found that the successful normal operation of the replicase complex requires a constant influx of triphosphate precursors. Breaking this influx leads to distortion of DNA synthesis, and hence to antitumor action. By disturbing DNA replication, the arabinose nucleosides isolated from the Cryptothethya fungus also act. Further testing of chemical analogs of cytosine-arabinoside has led to the discovery of many other antitumor drugs with improved properties, e.g. gemcitabine, which is currently widely used in many cancers.

The topology of DNA as a target for antitumor agents. Detection of DNA-topoisomerases provides an explanation for the molecular mechanism of the cytotoxic action of many natural products that damage DNA but do not interact chemically with it. Thus, the antibiotic actinomycin D, isolated from streptomycetes, now with limited use in rare oncological diseases, binds to DNA by inserting polycyclinc chromophore between the base pairs of the double spiral. Actinomycin D inhibits the synthesis of RNA, but this does not explain its antitumor activity. The subsequent discovery of both anthracycline derivatives - daunorubicin and doxorubicin, which also bind to DNA by inserting their chromophores, however, shows reduced inhibition of RNA synthesis compared to actinomycin E. Clinical application of daunorubicin Is limited to haematological malignancies, mainly due to unacceptable toxicity, unlike doxorubicin and its derivatives, which have a broad spectrum of action and main therapeutic application at present. The synthetic compound amsacrine, which has limited use in acute leukemia, binds to DNA by insertion of its acridine chromophore but has a negligible effect on RNA synthesis. These drugs act as inhibitors of the enzyme topoisomerase 2, changing its normal function to one that leads to DNA destruction.

The so-called podophyllotoxin analogs derived from root mandrake. Podophyllotoxin, similar to colchicine, binds to tubulin, but some synthetic glycosylated derivatives, called epipodophyllotoxins, show a broader spectrum of antitumor activity at reduced toxicity. Etoposide and related teniposide, useful in a large number of solid and haematological oncological diseases, have reduced ability to bind tubulin, but enhance DNA destruction by modifying the function of topoisomerase 2.

The non-DNA-bound plant product camptothecin specifically modifies the topoisomerase function 1. The water soluble analogues of camptothecin topotecan and irinotecan have greatly expanded their clinical application after topoisomerase 1 has become a new target for antitumor therapy.

Increased selectivity of anti-tumor agents. The selectivity of cytotoxic therapy depends mainly on the characteristics of the tumor mass. The use of microbiological models has shown that the action of acylating drugs is killing cells in exponential dependence - a certain percentage of the cell population is destroyed with each subsequent dose administered. On this basis, using animal models, the cytotoxicity of certain antitumor agents has been optimized, depending on the moment of their application to

the cell cycle, as well as by rational combination of the patterns for their application. This approach has been successfully applied to haematological oncological diseases, which in most cases has a high level of cell division but does not form solid tumors.

Another approach to increasing selectivity focuses on intra-cell transport mechanisms of enzymes and active substances that differ in tumor and normal cells. Many antimetabolites exhibit their selectivity in this mechanism. Topoisomerases are an example of such selectivity in which high cellular activity is associated with increased susceptibility to agents that bind to these enzymes.

In recent years, increasing selectivity has been achieved through the creation of the so-called precursors of cytotoxic agents, that do not have their own cytotoxic activity until they are activated by an enzyme or other agent. Initially activators of these precursors were normal enzymes in the cell, but activating enzymes have recently been investigated by targeting antibodies or by gene therapy.

The most important feature of **cytostatic therapy** is that it primarily damages proliferating cells by various mechanisms and leads to cell death. The sensitivity of a malignant tumor to chemotherapy is determined by the specificity of the cells that make it, namely:

- Cell cycle and doubling time;
- Growth fraction;
- Volume of the tumor (proliferation fraction/ resting cell ratio);
- Sensitivity to cytostatics;
- Heterogeneity of the tumor.

On the other hand, the effectiveness of a particular cytostatic depends on its concentration and time of exposure, as well as on the presence or otherwise of resistance.

Since malignant tumors are comprised of a heterogeneous population of cells of varying sensitivity to the various cytostatics, the most effective cytostatic combinations contain substances with a different mechanism of action and with proven antitumour activity on self-administration. The ideal option is for the cytostatics of the combination to have a synergistic effect. At the same time, overlap of toxic effects should be avoided. Apart from a suitable combination of cytostatics, the therapeutic efficacy of chemotherapy can be increased by administering high doses or by the so-called "compressing" the doses.

Currently, due to the discovery of new antitumour substances with a different mechanism of action, it is possible to treat about 20% of new cases of malignant neoplasms only with systemic therapy.

In practice, cytostatic treatment is generally divided into three types, depending on the goals it pursues:

A. Adjuvant (post-operative) chemotherapy - is applied in cytostatically drugs sensitive tumors following radical surgery to fully reverse the possible micrometastases . Adjuvant treatment is conducted for a certain duration with cytostatics and combinations of these, which have proven to be most effective in multi-center international clinical trials, in certain groups of patients, depending on complex factors derived from the pathomorphologic finding or after additional immunohistochemical and other studies so-called prognostic and predictive factors). For example, in the classical known prognostic factors of mammary carcinoma (involvement of axillary lymph nodes, hormonal sensitivity, tumor size, overexpression of HER2), lately, the success of genetics has also been added - revealing the so-called "genetic signature": 70- or 21- genetic profile characteristic of patients who progress rapidly after adjuvant treatment is complete.

B. Neoadjuvant chemotherapy - is performed before the main therapeutic procedures - surgical treatment or radiotherapy with several main objectives: reduction of tumor volume with a view to its radical removal; treatment of micrometastases in distant organs; provides information on the sensitivity to the cytostatic combination used, to assess the type of postoperative treatment most appropriately. Before starting treatment, it is necessary to study tumor material and to determine the maximum number of prognostic and predictive factors. Neoadjuvant chemotherapy is only susceptible to drug-induced tumors. In hormone-dependent tumors (breast cancer), neoadjuvant hormone therapy could also be performed.

C. Palliative chemotherapy - performed in a metastatic disease. The results of this treatment depend on many factors: tumor sensitivity, general condition of the patient, type and size of the metastasis, side effects of the combination administered. At the stage of metastasis, most tumors are incurable, the primary goal of treatment is to control the symptoms caused by tumor growth, improve quality of life, and increase survival.

WHO group malignant drug-sensitive tumors into five categories:

- **Category 1** - tumors for which there is evidence that the use of drugs alone or in combination (with each other or with other treatments) will result in a cure of normal duration of life or prolonging survival for the majority of patients. Here are some child hematological diseases, trophoblastic tumors, germ cell tumors.
- **Category 2** - tumors where median survival is prolonged after chemotherapy as an adjuvant after local surgical intervention or radiothera-

py in early stages of the disease. These include malignant breast tumors, ovarian carcinoma, tumors of the colon and the right gut.

- **Category 3** - tumors where there is evidence that the use of medication alone or in combination causes a tumor volume reduction of more than 20% in some patients with distant metastases and almost certainly improves the quality of life in all patients treated. This group refers to the metastasis stage indicated in category 2, as well as malignant lung tumors, some lymphomas and oth.
- **Category 4** - tumors where local disease control can be improved with chemotherapy before, during or after surgery or radiotherapy - such as malignant breast, ovarian, malignant bladder tumors, and prostate carcinoma.
- **Category 5** - tumors where there are currently no effective drugs. Such are malignant tumors of the kidney, pancreas, primary liver carcinoma and others.

Combination chemotherapy requires knowledge about the possible side effects of treatment. Because the metabolic processes in normal and tumor cells do not differ significantly, as well as due to the lack of selectivity of cytostatics, cytostatic treatment inevitably impacts healthy tissues as well. The most suffering mitotic activity cells-haematopoietic and reproductive. Accurate dosing and tracking of the patient in the critical time for **haematological toxicity** is key to avoiding life-threatening complications such as febrile neutropenia, thrombocytopenia.

Almost all cytostatic combinations are accompanied by **gastrointestinal toxicity**, expressed to varying degrees: nausea and vomiting, diarrheal syndrome, mucositis and stomatitis. Adequate anti-emetic therapy, the training of patients to adhere to an appropriate hygiene regime and timely reporting of symptoms of toxicity have a key role in overcoming gastrointestinal toxicity.

Some of the cytostatics used are characterized by so-called **cumulative cardiotoxicity** (anthracycline antitumor antibiotics). This requires that their application be preceded by a thorough examination of the myocardial function as well as its monitoring during the course of treatment. Cardotoxic are also some modern antitumor agents - the monoclonal antibody Herceptin, some "small molecules" - Lapatinvib and others.

A typical unwanted side effect following platinum salt administration is **nephrotoxicity**. This fact also necessitates a thorough preliminary refinement of the renal function and its follow-up during the course of treatment. Other parenchymal organs could also be impaired when certain cytostatics are administered: **pulmonary toxicity** is observed with Bleomycin, **hepatic**, with some enzymes, and so on.

Hypersensitivity reactions are another life-threatening side-effect phenomenon requiring rapid and adequate intervention. This is the case with taxanes - preparation with an oral or parenteral corticosteroid is required from the day before the infusion of cytostatics. Anaphylactic reactions have been observed with platinum salts, epipodophyllotoxins (etoposide), and others (for cytostatics in the taxane group, as well as for platinum derivatives, **neurotoxicity**, which is dose dependent and usually reversible, is also characteristic).

The most common toxicity that damages **the skin and its appendages** is alopecia. There are currently no effective means of prevention. Usually, alopecia is reversible. Signs of skin sensitivity include hyperkeratosis, hyperpigmentation, photosensitivity. A kind of skin toxicity is so-called palmar-plantar erythrodysaesthesia - painful erythema of the palms and feet, which can be complicated by desquamation, exsulceration and overlapping infection. This toxicity is typical of Capecitabine antimetabolite (Xeloda) and liposomal doxorubicin (Caelyx).

In addition to immediate (observed over time or shortly after treatment with cytostatic drugs), appear and **suspended** to **toxicity** - delayed growth in childhood, late cardiotoxicity (cardiomyopathy and congestive heart failure), deferred renal and pulmonary toxicity, ototoxicity, endocrine and metabolic dysfunction (hypogonadism, hyperlipidemia, etc.), the onset of secondary tumors. The higher incidence of leukemias in patients treated with alkylating agents or topoisomerase II (anthracycline antitumor antibiotics) inhibitors, is known to occur. No other preventative measures to avoid late toxicity are known except for the qualitative and systematic follow-up of the patients treated.

Systemic treatment of fundamental importance in oncology is ***hormone therapy***. Some malignant tumors require hormonal stimulation for their growth - e.g. malignant tumors of the mammary gland and prostate require corresponding action with estrogens or androgens. Capture places of hormones in the cell are called hormonal receptors. In hormone sensitive tumors, blockage of receptors may result in tumor growth arrest or apoptosis. Hormone treatment is associated with much less pronounced adverse side effects than cytostatic treatment, and the effects achieved with this therapy are of longer duration. This treatment often brings additional benefits - eg. in the treatment of breast cancer with hormone therapy (anti-estrogens) reduces the risk of malignant tumor in the contralateral mammary gland, increases bone mineral density, improves

the lipid profile.

TARGET THERAPY

Simultaneously with the development of the described cytotoxic drugs, new alternative approaches have been developed to influence the signals that control cell growth and differentiation. With the rapid development of molecular biology and oncology, dozens of potential targets have been identified for pharmacological impact on the complex regulation of cell growth and differentiation processes. Thus, in 1960-1970, in vitro cell cultures - normal and tumor cell studies have shown that different sets of polypeptide growth factors as well as their receptors are vital for these processes. Many of these are tissue-specific. The basis of their action is the activity of these growth factors on the regulation of DNA replication and cell division.

These targets are considered to meet the following general requirements: to be found in a large proportion of patients with a certain type of cancer, to be causally related to the tumor genesis, to be essential for the tumor cell, and for the normal cell To have little meaning. For most of these targets, specific target-directed anti-tumor drugs of a new type that are non-genotoxic and have no cytotoxic effect on all fast-growing cells in the body have already been developed or are being investigated. These new drugs usually bring tumor cells into a "sleeping" state without being divisive, but their destruction requires the action of anti-cancer drugs from the classical cytotoxic type.

Programmed cell death (apoptosis). One of the most impressive discoveries in the processes of dividing normal and tumor cells was the presence of the so-called a biological clock, that program the cell when it replicates DNA and when it enters a division cycle. A number of known antitumor drugs have been shown to act by enhancing the processes of apoptosis in the tumor along with other molecular mechanisms of action, but these processes will not stop here.

Potential molecules - "targets" are found **inside cellular structures or on the surface of the cell**. Examples of extracellular targets are stromal factors (so-called matrix metalloproteinases) and blood vessels. Targets on the inner or outer side of the cell membrane are membrane cell receptors and intracellular signaling processes.

I. Targets outside the tumor cell

1. Matrix metalloproteinase inhibitors.

It is known that proteolytic changes of the extracellular matrix surrounding tumor cells as well as the basal membrane have a key role in tumor growth processes.

When tumor cells pass the basal membrane and enter the extracellular space, invasive tumor growth occurs. If tumor cells invade newly formed vessels in the extracellular space, there is a tumor spreading - a process known as metastasis. Proteolytic changes of the extracellular matrix are determined by the activity of four classes of endopeptidases: serine, cysteine, aspartyl proteases and matrix metalloproteinases (MMPs). The latter are zinc-dependent endopeptidases capable of destroying almost all components of the extracellular matrix. In some tumors, the expression of MMP is higher than in normal tissues. In particular, the expression of MMP-2, MMP-7 and MMP-9 correlates with invasive tumor growth, the metastatic potential of the tumor and the adverse outcome of the disease. Inhibition of MMP activity is achieved with a number of substances that have proven their effectiveness in preclinical studies, but due to underlined side effects, they have not yet found a wide clinical application. The different phases of clinical trials are Marimastat (I-III phase), Batimastat (I-II phase), Neovastat (I-III phase), and others.

2. Angiogenic Inhibitors.

Important discoveries have laid the groundwork for the rapid development of this field: endothelial cell culture in vitro, the creation of methods for quantification of angiogenesis, the detection of angiogenic stimulants (BFGF - basis fibroblast growt factor; VEGF/VPF-Vascular Endothelial cel Grouth Factor/Vascular Permeability Factor; ang-1-angiopoeitin), the detection of endogenous inhibitors (TSP-1-thrombospondin, INF alpha, angiostatin), the development of anti-angiogenic drugs on a rational basis, the role of oncogenes and tumor suppressor genes in tumor angiogenesis, the possible lack of drug resistence to angiogenesis inhibitors as well as the beneficial integration of angiogenesis inhibitors with standard treatment methods (mainly with classical cytotoxic antitumour drugs).

Angiogenesis is needed at the beginning and end of the metastatic cascade. Due to breaks in the basal membrane, the tumor vessels are permeable, and the tumor cells easily pass through their walls and enter the circulation in large numbers - several million tumor cells per day. A positive correlation was found between the number of tumor microvessels in a biopsy material and the patient's fate in prostate cancer, melanoma, breast cancer and ovarian cancer.

Tumor angiogenesis is a multistage process stimulated by pro-angiogenic factors. VEGF secreted by tumor cells has been most extensively studied. It has many isoforms. Since there is a correlation between VEGF expression, increased angiogenic activity, aggression of the malignant tumor, its metastatic potential, and the adverse outcome of the disease, inhibition of angiogenesis has been perceived as an

important antitumor target for decades. After obtaining the results of large-scale clinical trials, the monoclonal anti-VEGF antibody Bevacizumab (Avastin) is already being clinically proven. Its use in malignant colon tumors leads to a higher percentage of clinical remissions, greater duration of effects achieved and greater survival when adding to chemotherapy line I at this location. This is the first angiogenic inhibitor to achieve better survival in randomized clinical trials.

Another way to inhibit VEGF-induced angiogenesis is blocking tyrosine kinase receptors. There follows an intracellular information cascade of signaling processes leading to suppression of endothelial cell proliferation. Numerous small molecule inhibitors of VEGF receptors for tyrosine kinase activity are found in different phases of clinical trials: PTK 787, SU11248, ZD 6474, and so on.

The large number of angiogenic factors identified, the more important and better-understood are fibroblastic growth factors (FGF family), vascular endothelial growth factor (VEGF), the family of angiopoietins, etc., as well as their receptors, give possibility to discover many new targets for rationally targeted non-genotoxic therapy. Of great theoretical and practical interest are the most advanced molecular studies on the structure of these receptors and in particular on the identification of surfaces involved in receptor recognition **(Figure 1)**.

Fig. 1. Spatial Structure of Ang-2 (Barton W. A., Tzvetkova D., Nikolov D. B., 2005)

An Ang-2 - RBD model showing the location of the side (active) radicals involved in receptor recognition in oval and rod-shaped form. The exact location (spatial orientation) of each of the side radicals in the P-domain (active site)



1. Side (active) radicals
2. P-domain (active site)

II. Intracellular targets and targets on the cell surface

During the 1980s, tyrosine kinase phosphorylation processes were established and their key role in regulating important cellular processes: cell cycle, cell metabolism, cell growth, cell proliferation. The family of the so-called epidermal growth factor receptor (EGFR). It consists of four types of receptors: ErbB1, ErbB2 / HER2, ErbB3, ErbB4. They all have a common structure made up of an extracellular and intracellular (cytoplasmic) part that contains a protein kinase and can be phosphorylated. Attachment of ATP to the cytoplasmic site results in activation of the tyrosine kinase followed by a cascade of cellular processes.

It has been shown that in many epithelial human tumors the expression of EGFR has been increased, which correlates with tumor aggressiveness and adverse outcome of treatment. Inhibition of uncontrolled EGFR-tyrosine kinase activity is a target for a number of antitumor agents, some of which are already in clinical use. These include: monoclonal antibodies Transtuzumab (Herceptin), Cetuximab (Erbitux), reversible tyrosine kinase inhibitors Gefitinib (IRESSA), Erlotinib, Imatinib (Glivec). For monoclonal antibodies, the target is extracellular part of the EGFR has been shown to result in blocking receptor activity. Widespread clinical use in metastatic breast cancer, as well as in adjuvant treatment, finds Transtuzumab (Herceptin). Its use together with cytostatic treatment or alone leads to an increase in the percentage of clinical remissions as well as survival. Similar are the effects of Cetuximab (Erbitux) in metastatic colorectal cancer - alone or in combination with chemotherapy.

Small molecules - tyrosine kinase inhibitors of EGFR are also in advanced phases of clinical trials or already with clinical use. Such medicinal product is Gefitinib (IRESSA) in non-small cell lung carcinoma - alone or in combination with chemotherapy. In a small percentage of the patients (about 10%) who have a specific EGF receptor mutation, the effect is remarkable-disappearance of the symptoms of the disease for a few days and reversal of the tumor.

Chronic myeloleucosis and gastrointestinal stromal tumors are two distinct diseases due to a single genetic defect - the presence of so-called Philadelphia chromosome (t / 9,22) translocation and presence of KIT - protooncogene, respectively. Imatinib mesylate (Glivec) is a tyrosine kinase inhibitor that has resulted in complete clinical remissions in these two incurable diseases **(Figure 2)**.

The individual stages of the metastatic cascade - angiogenesis, adhesion, proteolysis, motility, and proliferation, serve as new targets for the prevention

and treatment of oncology. Thus, the anti-angiogenic action of interferon that suppresses gene expression for angiogenic growth factor leads to regression of hemangiomas in children.

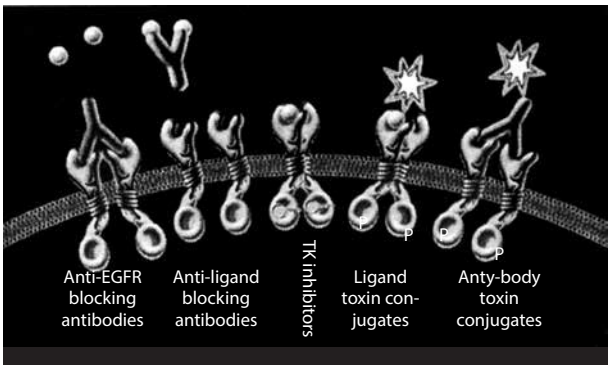
Several antiangiogenic agents such as thalidomide, TNP170, and others that suppress endothelial cell proliferation are clinically tested. Intensive strategies for blocking vascular endothelial growth factor, strategies for inhibition of integrin function, strategies for blocking the degradation and remodeling of the extracellular matrix, etc. are being developed. Intense experimental studies are devoted to influencing the signaling pathways of the metastatic process - calcium homeostasis, kinase activity, ras oncogene activation.

The introduction into clinical practice of target molecular therapy has fundamentally altered the way of thinking about the treatment of malignant tumors. This type of treatment is getting closer to the optimal option - applying a specific treatment for each individual tumor and for each individual patient.

Approaches to target EGFR dysregulation.

1. Antibodies against EGFR - receptor antagonists interfere with ligand binding. They are widely used in practice - Mab Thera, Herceptin.

Fig. 2. Target blocking approaches (Noonberg S. B., Benz C. C-2000)



2. Antibodies against the ligand - at the stage of preclinical testing.

3. Low-molecular substances that inhibit reversibly or irreversibly tyrosine kinase (TK) activity at intracellular level - block all EGFR. Two such drugs are due to be registered.

4. Cytotoxin or cytotoxin ligand conjugates - kill the cell after internalization as an add-on to inhibition of tyrosine kinase. The ability of the monoclonal antibody to transport the cytotoxic agent to the target receptor on the cancer cell is used - after internalizing the receptor, the cytotoxic agent is released inside the cell.

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10 PROFESSIONAL SKIN DISEASES

Skin is a biologically active organ that separates the human body from the surrounding environment. In many professions it is exposed to harmful factors such as chemicals, biological materials, mechanical and physical violence. Occupational diseases of the skin develop in the balance between its resistance and the strength of the factor of the environmental and the professional environment.

There are different views on the classification of occupational diseases of the skin. According to Sezary, professional dermatoses are divided into two main groups: **irritating** (orthoergic) and **allergic**.

The primary disease is **contact dermatitis**:

- irritative (orthoergic) contact dermatitis;
- toxic contact dermatitis;
- allergic contact dermatitis, incl. contact urticaria;
- phototoxic contact dermatitis;
- photoelectric contact dermatitis;
- pigmentary skin disorders;
- chlorine acne;
- professional dermatoses caused by plants;
- professional dermatoses caused by biological factors;
- professionally determined skin tumors.

10.1. IRRITANT CONTACT DERMATITIS

Irritant contact dermatitis is a non-immunological acute local inflammatory response characterized by erythema, edema or corrosion after single or multiple exposure to chemical substances on the skin. The clinical picture depends on the nature of the stimulus - organic solvents, machine oils, detergents, etc. Dermatitis caused by acids is characterized by rapid skin damage, while that of alkali is significantly slower. It is expressed by skin redness, drought, hyperkeratosis and rhagades. The disease is most often localized on the skin of the hands and forearms. Persons with endogenous eczema or other forms of atopic disease get easier dermatitis in contact with chemicals and paints: fitters, hairdressers, sanitary personnel, and others.

Irritating contact dermatitis has two main forms: acute and chronic.

Acute irritant contact dermatitis occurs after single or double contact with strong irritants, is not preceded by a latency period, and in most cases occurs only at the site of contact with the stimulus. Phototoxic contact dermatitis is a more specific type of chemical burn in which the irritant agent acts only in combination with sunlight.

The clinical picture is characterized by erythema, edema, sometimes with vesicles and bulls. Subjectivally, it may be accompanied by pain **(Table 1)**.

Diagnosis: A history of contact, possibly a biopsy. Skin testing is not done due to the danger of exacerbation and inadequate results. **Differential diagnosis:** arteficial dermatitis of another nature (physical, thermal, etc.).

Treatment: Immediately wash the affected skin thoroughly with water. Topical treatment: wet dressings with 10% Burov solution, potassium hypermanganate solution etc. two to three times a day. These compresses are followed by applications of topical corticosteroids. If dermatitis is disseminated, systemic corticosteroids and antihistamines are required. In secondary bacterial infection, local antibacterial therapy. In necrotic changes - surgical treatment.

Prevention: removing contact with irritating chemicals. Workplace survey for detecting harmful stimuli. Use of protective clothing and gloves.

Prognosis: Favorable in lighter forms.

Chronic irritant contact dermatitis is the most common occupational skin disease that occurs with multiple cumulative exposures to weak irritants: soaps, detergents, water, lubricants. Affects most of the hands. It is common among female hosts cleaners, who have day-to-day contact with these irritants.

Clinical picture: Disease changes are quite characteristic - erythema, scaly plaques with vague borders, fissures. Subjective complaints are itching and often pain (mostly from the fissures available). Nails are transverse. This particular picture gives reason to call it the "hostess' hand". People with a family history of atopy are more likely to be affected. On this basis, subsequent individual sensitization to the same substances that have triggered earlier irritative changes or new allergens, is often developed. Some workers may also develop relative resistance to irri-

Tab. 1. Types of contact dermatitis (by D. Birmingham)

Features	Irritating contact dermatitis	Allergic contact dermatitis
Receipt mechanism	Direct cytotoxic effect	Late cellular immunity type (Gell and Coombs type IV)
Potential risk	Everyone	Minority
Begining	Progressively, after repeated long exposure	Fast, after 12-48 hours in sensitive patients
Clinical changes	Subacute to chronic eczema with erythema, desquamation and fissures	Acute to chronic eczema with erythema, edema, bulls and vesicles
Symptoms	Pain and burning	Itching
Concentration of the stimulus	High	Low
Research	History and research	History and research, Patch test

tants with which they have been in professional contact (eg cement, lime, chromates, etc.). This phenomenon is called non-specific hardening. In these cases, the worker may continue to work and do not require re-employment.

Differential diagnosis: it is done with allergic contact dermatitis (history and positive skin samples), atopic eczema, dyshidrotic eczema.

Treatment: local corticosteroid ointments and creams, general treatment - antihistamines and others.

Prevention: Workers with a family history of atopy are at high risk of developing chronic irritative dermatitis, which should be considered when choosing a profession. Use personal protective equipment to avoid contact with irritants (clean working clothes, gloves, protective creams). Avoid highly irritating skin cleansing agents.

Forecast: Safeguarding. In heavier cases, re-employment is recommended. With good prophylaxis, the work process can last a long time.

10.2. ALLERGIC CONTACT DERMATITIS

Consists of 20% of workers with professional contact dermatitis. Appears after 48 hours to two weeks after contact with a chemical (hapten). The latter binds to the skin protein to produce a complex antigen and develops a cell-associated immune response in the body. Upon further re-exposure to the same antigen a lymphocyte-cell inflammatory skin reaction (late-type allergic reaction) is obtained. Changed working conditions and the intervention of new industrial allergens also lead to changes in the dynamics of the professional dermatoses with a tendency to increase the allergic contact skin damage compared to the irritative and infectious ones **(Table 2)**.

Clinical picture: Characterized by the onset of erythema, edema, desquamation, and papule crusts. Sometimes there are vesicles and bulls as a result of the accumulation of fluid between epidermal cells (spongiosis). Localization: most often at the point of contact, but it also occurs in remote, more sensitive areas. A generalized measles-like rash, called "auto-sensitizing dermatitis," or "ID" reaction, occurs in a preserved disease process at the site of contact. Photo-allergenic response occurs when the site of contact is exposed to sunlight with a wavelength of more than 320 nm. Sometimes the nature of the allergen determines the clinical picture - so contact with plants may lead to abruptly limited linear erythems and bulls; some chemicals used in the rubber industry, e.g. phenyl-isopropyl-para-phenylenediamine may cause Purpura like eczema; the developer of colored film strips causes a eruption resembling Lichen ruber planus and so on.

Diagnosis: History, skin tests, exposure and elimination test, histological examination.

Differential diagnosis is difficult between chronic irritative contact dermatitis and allergic contact dermatitis **(Table 3)**.

The most common causes of occupational allergic contact dermatitis are being investigated worldwide through good professional history and data from the so-called testing with a standard series of allergens, specific to individual production. Testing with the standard series, which is a sort of screening, does not replace individual research with other allergens, but complements it.

Treatment. For the successful treatment of professional dermatitis (irritant and allergic) it is necessary to eliminate the risk factors of the professional environment on the one hand and to increase the resistance of the skin to their impact.

Topical treatment is based on the stage of the disease. During the acute phase, compresses (wet bandages) with potassium hypermanganate (1: 5000) or a solution of oak bark, chamomile, etc. are made every 1-2 hours, then aniline dyes (Solutio Fuchsini 1%, Solutio Pyoctanini 1%) and others are coated. After stopping wetting and passage in subacute stage,

Tab. 2. The most common professional irritants in some important productions (J.B. Olishifski, 1988)

Chemicals	Primary irritation	Sensitization	Skin and other changes	Production / Profession
Acids				
Acetic	x	?	Dermatitis and ulcerations	Acetate silk and coloring, vinyl plastic
Phenol	x		Corrosion, local anesthetic effect	Production of phenol, disinfectants, paints, pharmaceutics, plastics
Chromic	x	x	Ulcus, inflamation and perforation of septum nasi	Production of organic chemistry, dyeing, silverware
Formic	x		Irritation with blisters and ulcerations	Rubber, detergents, cellulose and lubricants
Hydrochloric	x		Irritation, ulcerations	Production of bleaching agents in refineries, chemical industry
Lactic	x		Ulcerations at high concentrations	Adhesives, plastics, textiles
Nitric	x		Burning, ulcera	Production of nitric acid, metal cleaning agents, paints
Oxalic	x		Skin corrosion, cyanosis, nail fragility	Leather fur, indigo, oxalic acid production
Picric	x	x	Erythema, dermatitis, yellow skin and hair coloring	Production of explosives, painting, mining
Sulphuric	x		Corrosion of the skin inflammation of the mucous membranes	Chemical production, vinegar, galvanization
Alkalis				
Calcium cyanamide	x		Irritation and ulcerations	Farm workers, production of nitrogen compounds
Calcium oxide	x		Dermatitis, burns or ulcerations	Working with lime, producing calcium salts, glass, fertilizing
Potassium hydroxide	x		Skin corrosion, deep persistent ulcus, loss of nails	Production of KOH, paper, soaps, printing inks
Sodium hydroxide	x		Skin corrosion, deep persistent ulcus, loss of nails	Manufacture of NaOH, soaps, paints, bleaching, oil refineries, plastics
Sodium silicate	x		Skin corrosion, ulcera on fingers	Cement, water treatment, detergents
Salts and elements				
Antimon	x	?	Irritation and licheniform skin eruptions	Extraction of antimony, glass, aniline, fireworks, alloys
Arsen	x	x	Spotty pigmentation, perforation of septum nasi, carcinoma, hyperkeratosis of the hands, dermatitis, pustules	Leather, insecticides, glass, agriculture, tanning, alloys, oils
Bromine	x		Iritis, vesicles, ulcus, acne	Extraction of bromine, bromine salts, paints, medicaments, photo materials
Chrome	x	x	Punched ulcera, dermatitis, perforation of septum nasi	Chrome, paints, chrome manufacture, tanning
Nickel	x	x	Folliculitis, dermatitis	Nickel production
Zinc chloride	x	?	Skin ulcera and septum nasi	Manufacture of chemicals, paper, disinfectants, paints

Solvents				
Acetone	x		Dry skin	Celluloid industry, artificial silk and leather, working with varnishes and sprays, garage
Benzol	x		Dry skin	Chemical and rubber Industry
Trichlorethylene	x	?	Dermatitis	Chemical intermediate products
Turpentine	x	x	Dermatitis	Artists working with polishes and varnishes
Oil and tar				
Petrol	x		Dermatitis, folliculitis	Engine-drivers, mechanics working with oil
Resin and asphalt	x		Dermatitis, folliculitis keratoses, skin carcinoma	Production of resin, asphalt
Tar	x	x	Dermatitis, folliculitis, skin carcinoma, keratitis	Production of tar, roof paper, construction and repair of roads
Paints				
Paraphenylen-Diamine (Ursol)	x		Contact dermatitis (Erythema, edema, blisters)	Painters, cosmetics
Insecticides				
Creosote	x	x	Dermatitis, folliculitis, keratoses, hyperpigmentation, skin carcinoma	Production of preservatives for wood, railway rails, tar, resin
Fluorides	x		Dermatitis, burning	Production of insecticides, enamelling
Pyrethrum	x	x	Dermatitis	Production and application of insecticides
Rottenon	x		Dermatitis	Dentists, paper processing
Natural resins				
Shellac	x		Dermatitis	Cosmetics, cladding
Colophon	x		Dermatitis	Dentists, paper processing
Explosives				
Nitrates, Nitroglycerine	x	x	Iritation, dermatitis, pigment changes	Production of explosives
Others				
Vinyl chloride	x		Dermatitis, acro-osteolysis	Polyvinyl resin, chemical markers
Formaldehyde	x	x	Dermatitis	Biologists, textile workers

weak corticosteroid creams (1% hydrocortisone, etc.) are used. In the treatment of chronic forms, corticosteroid ointments, which can be combined with salicylic acid or tar, are recommended. When general treatment is needed, antihistamines are used and for short period systemic corticosteroids with high dosing at the start and gradual reduction.

Rehabilitation. High-mountain and sea climate therapy, balneotherapy has a beneficial impact. It is due to non-specific action and changes in reactivity, immune mechanisms, neuro-vegetative system, etc.

Prevention. Change in the technology by eliminating or replacing the substances with high sensibilization index with other protective (hydrophilic or hydrophobic) protective creams, skin protection from oils, solvents, petroleum products, hydrocarbons, paints, varnishes and the like. Easy to wash after work. Hydrophobic pastes, liquids or aerosols are water insoluble and have a chemical instability to the acids and substances such as turpentine, potassium bichromate, formalin and the like. It is recommended washing with a soft, non-irritating soaps, fuller's earth, followed by coating the skin with 2% pined or normal vaseline (do not use abrasive detergents), PPE - working and protective clothing, gloves and the like.

Prognosis. If the disease process is not affected, it is necessary to work readjustment or change the profession.

Contact urticaria occurs with edema, erythemic, itching fast transient papules or raised (angioedema) lesions at the site of the contact. The latency period is from a few seconds to minutes after the contact, and the duration of the rash is 1-4 hours.

Etiology. Changes may be due to a specific antigen or result from a pharmacological reaction that

Tab. 3. Differential diagnosis between irritant and allergic contact dermatitis (P. Mihaylov, J. R. Nethercott)

	Acute irritative contact dermatitis	Chronic irritation contact dermatitis	Allergic contact dermatitis
Begining	Suddenly, at first contact, no incubation period	Slowly in weeks	After a phase of sensitization and a latency period
Number of affected persons	Everyone who was in touch	In most persons who were in contact	Only individuals
Dependence	From the nature of the primary stimulus and its concentration		From the reactivity of the body
Clinical picture	Erythema, edema, vesicles, bulls, chemical burns	Dryness, lichenification excoriations	Erythema, edema, vesicles
Subjective complaints	Pain	Pain, weak itching	Itching
Histological picture	Epidermolysis, epidermis necrosis, polymorphic nuclear and leukocyte infiltrate		Spongeosis, vesiculation, acanthosis, lymphocyte infiltrate
Passing	A short time	Long, with periods of abatement	A short time
Mechanism of appearance	Non-immunological	Non-immunological (cumulative)	Immunological
Skin tests	Negative	Negative	Positive
Causes	Strong acids and alkalis, solvents	Soaps, detergents, water, oils	Nickel, Chromium, Plastics, Paints, Plants, Medicines, Cosmetics
Treatment	Quickly	Slowly, with a tendency to relapse	Anti-allergic
Prevention	No redevelopment	No redevelopment	Often work readjustment is required

occurs in the various occupations, and IgE response may induce contact urticaria. For this type of reactivity, the release of histamine and other vasoactive substances from mast cells is characteristic. However, some substances (sodium benzoate, etc.) can lead to direct release of histamine. There must be intense initial exposure without a latency period.

Contact urticaria may also occur upon contact with platinum salts, ammonia, formaldehyde and others. Inhalation urticaria occurs in gas-related and volatile chemical compounds (hydrogen sulphide, ursol, etc.). They can also be combined with the onset of respiratory allergy, rhinitis, bronchitis, asthma.

Contact urticaria occurs also in contact and in touch with proteins from various organs of animals, body fluids, blood, saliva, epithelium.

Contact urticaria develops and in contact with certain exotic trees and plants (chrysanthemum, mahogany) in bakers and flour workers, hairdressers (contact ursol, ammonium persulfate), hosts (from latex gloves, etc.), drugs, cosmetics (acetone, ammonia, nail polish and hair, lipsticks, etc.).

Urticaria par effort occurs in athletes, farm workers. Cold urticaria occurs in swimmers and heat urticaria - in metallurgists.

Diagnosis. It is not difficult - the appearance of rapid transient rash and its passage without leaving traces.

Differential diagnosis: urticaria and angioedema.

Treatment. Topical-cooling compresses, general treatment - antihistamines (Diphenhydramine 25-50 mg 3 times a day, Hydroxyzine 10-50 mg daily, Terfenadine 60 mg daily, etc.). Corticosteroid therapy, local or systemic, is not recommended.

Prognosis - good.

Prevention. Personal protective equipment.

10.3. PHOTOTOXIC CONTACT DERMATITIS

Most photoreactions of the skin are phototoxic. Natural and artificial light sources alone or in combination with different chemicals, plants or medications can cause a phototoxic or photosensitive response. The phototoxic reaction is mainly related to the light-exposed parts, whereas the photosensitive response is more common in the exposed parts of the body.

Phototoxic contact dermatitis occurs in contact with certain substances (by a non-immunological mechanism) that come into contact with ultraviolet light. These substances cause reactions in all persons at first contact, provided they have a sufficiently high concentration and absorb enough ultraviolet energy.

Clinical picture. It is characterized by the occurrence of edema, in the more severe cases with vesicles and bulls resembling sunburn and localized on

the exposed parts of the body and the photodynamic substance. If the substance is volatile, the reaction may spread beyond the limits of the directly exposed areas. Sometimes skin changes can be linear in places that have been in contact with the grass and flowers and the sunlit (i.e. Dermatitis pratensis, variant phytophotodermatitis). The phototoxic reaction resolves rapidly with residual hyperpigmentation.

The most common professional phototoxic substances are: tar, resins, creosote, furocoumarines (psoralens), dyes (eosin, acridine), drugs (sulfonamides, barbiturates, griseofulvin, etc.).

10.4. ALLERGIC CONTACT PHOTODERMATITIS

It is encountered in a small number of persons sensitized to a given substance under the simultaneous effects of light. A certain latency period is required for the occurrence of the reaction. The photoallergic reaction develops in the same immunological mechanism as the contact allergic reaction of the late (late) type. Photoallergens show properties of a contact allergen only in the presence of light rays of a certain wavelength.

Clinical picture. It is expressed by erythema, infiltration, papules, sometimes vesicles. They are located in places that have been in contact with photosensitizing substances and are irradiated with sunlight (mostly the skin of the face, neck, chest, forearms). The most common photoallergens are: medicines (phenothiazine derivatives, sulfonamides, antimicrobials, salicylic derivatives), eosin, dichlorophen, antimycotics and others.

Treatment. In light forms, local treatment with anti-inflammatory agents such as zinc oil, white mixture, cooling cream, corticosteroid creams, etc. is recommended. The severe forms that occur with vesicles, bulls and general symptoms (temperature, fever, etc.) treatment are common - with antipyretics, antihistamines, corticosteroids, and local.

Prevention. Avoid sunlight. Use of photoprotective creams with an adequate UV protection factor.

10.5. SKIN PIGMENTATION

Occupational Vitiligo, Leucoderma professionale.

Appears with white spots on the skin at the site of contact with depigmenting chemicals **(Table 4)** as a result of inhibition of melanin biosynthesis. In most cases, disconnection leads to slow repigmentation.

Clinical picture. Blurred depigmentation of the sites of the contact with the most frequent localization on the skin of the hands, folds and genitals, often hair. Best seen in the light of Wood.

Diagnosis. Pre-existing contact in history, postpartum repigmentation, presence of similar manifestations and other workers, histological examination.

Differential diagnosis: idiopathic vitiligo, Pityriasis versicolor alba, post-inflammatory hypopigmentation and others.

Treatment. Discontinuation of contact with depigmentation agents and exposure to ultraviolet light results in repigmentation, reception of methoxylene in combination with ultraviolet light at 320 nm to accelerate repigmentation. Local corticosteroids may also be used.

Prognosis. Repigmentation occurs in a few months, but in some cases it may not be complete.

Prevention. Avoid exposure to known depigmenting agents.

Riehl Melanosis (Melanosis Riehl, Kriegsmelanose).

Tab. 4. Reasons for Professional Vitiligo (by J.R.Nethercott, 1990)

Hydroquinone
Hydroquinone monobenzyl ether
Hydroquinone monomethyl ether
p-Cresol
p- tert – Amyl phenol
p- tert – Butyl phenol
o-Phenyl phenol
o- Benzyl –p- chlorophenol
4-tert-Butyl catechol

Described in 1917 by Riehl among workers in the arms industry. Contact with untreated tar products, vaseline, etc., which also have photosensitizing action.

Clinical picture. Erythema, later chocolate-grayish-colored hyperpigmentations, which extend and form a reticular mesh, are seen on the skin of the lateral face and temporal area, in front of the ear -conchs and lateral parts of the neck. The skin is dry, rough, with slight desquamation. It is accompanied by pruritus and mild burning.

Treatment: topical corticosteroid ointments and hydroquinone preparations.

Prognosis: unfavorable.

Photochemical skin changes (Solar Elastosis, "Farmer's Skin", "Sailor's skin").

Sun exposure leads to degeneration of elastin in the dermis and atrophy of the epidermis. It occurs with the appearance of wrinkles, yellowish pigmentation with hair follicles passing and the formation of comedones. The changes are localized on the face, V-shaped on the neckline, nape, shoulders and dorsal surface of the hands. Damage to the skin pigmentation of the skin leads to persistent hyperpig-

mented spots on the exposed face of the sun and the dorsal surface of the hands, which are called solar lentigo. Degeneration of elastin is histologically proven.

The presence of these changes is an indication of harmful sun exposure and the need to monitor for skin malignancies.

10.6. CHLORACNE

Chlor acne, also called "Perna disease" or "Perchlornaphthalin" or "chlorine scab", was described by Herxheimer in 1899. The largest number of people affected were described during the Second World War. Appears after contact with chlorinated hydrocarbons (chlorinated naphthalenes, azobenzenes, dibenzofurans, dioxin, polychlorinated biphenyls). The contact is always professional.

Clinical picture. Affected adults develop comedones, straw-colored cysts, miliums and papules with localized facial, neck, ear-conchs, shoulders, abdomen, genitals, legs. There is also blepharitis with granular changes along the edge of the eye. Hepatotoxic changes, Porphyria cutanea tarda, peripheral neuropathy and hypertriglyceridemia are also observed.

Differential diagnosis - see **Tab. 5**.

Treatment. Removal of contact with polynaphthalene products. Local treatment with anticomedonical preparations with evening application, abrasive cleaning agents and procedures.

Prognosis. Continuous backward development after interruption of the contact.

10.7. PROFESSIONAL DERMATOSES FROM PLANTS (PHYTODERMATITIS)

Contact with many plants can cause contact dermatitis irritating or allergic. Irritating action has leaves, stems, fruits and roots. Various substances are believed to play a role - alkaloids, essential oils, glycosides and others. For example, Ragweed causes contact dermatitis due to the water-soluble oleoresin content. Other plants that can cause allergic contact dermatitis are Chrysanthemum, Philodendron, Oleander, Tulipis, Narcissus and others. In Europe, Primula obconica and, above all, the primine component is the most common cause of plant contact dermatitis. In contact with primrose gardeners, pharmacists and oth. get erythema and edema on their fingers and eyelids. In the preparation of fig-jam confectioners and hosts, they get very characteristic dermatitis (Feigenbaum-dermatitis), localized to the fingers and wrists. There is erythema, edema, and in more severe cases blisters and erosions, and after their resumption - hyperpigmented macular plaques. For the occurrence of plant dermatitis, in many cases, besides plants, sun exposure also plays a role. The combination of both factors results in the so-called phytophotodermatitis. Most of them are the result of touching the plants to moist, sweaty or wet skin.

Plant dermatitis is a professional disease for gardeners, farmers, pharmacists, confectioners at work with plants. In the summer, gardeners, land workers and others the so-called Dermatitis pratensis appears when the grass is in contact with damp and sweaty skin and at the same time they are exposed to sunlight. A few hours later, red lines and strips of grass and plants on which they lay appear on the skin. It is accompanied by severe itching and burning, which is due to ethernal oils that have a photosensitizing effect.

10.8. OCCUPATIONAL SKIN INFECTIONS

Bacteria, viruses, fungi, dermatophytes and parasites can cause professionally related skin infections **(Table 6)**:

The treatment of occupational infections is as usual in their development - general and local, according to their type and stage.

Prevention - protective measures against the possibility of infection in the workplace.

10.9. PROFESSIONALLY DETERMINED SKIN TUMORS

In the last four decades there has been a general dramatic increase in skin carcinomas. This phenomenon is also associated with changes in sunlight and thinning of the ozone layer. Professional skin tumors are mainly three types: spinocellular and basal cell

Diagnosis	Age	Localization	Clinical changes
Acne vulgaris	13-26 years	Face, breast, back, waist	Comedones, papules, pustules, cysts, crusts
Folliculitis	Every age	The whole body in contact with oils	Black comedones, papules, pustules, melanosis
Chloracne	Every age	Face, neck, ear lobes, breasts, legs, genitals	Weak stained cysts, comedones, miliums, papules, some systemic events.

Tab. 5. Differential Diagnosis of Chloracne (Adams)

Tab. 6. Professional Skin Infections (J.R. Nethercott)

Causes	Professional relationship
Bacteria	
Bacillus anthracis	Wool sorters, tanning
Brucella abortus	Veterinarians, meat production
Erysipeloid	Fishermen, meat production
Francisella tularensis	Farmers, veterinarians, laboratory workers
Mycobacterium tuberculosis	Pathologists, veterinarians, farmers, meat producers
Mycobacterium marinum	Fishermen, fishery
Fungi	
Candida albicans	People working in the food industry, health professions
Trichophyton,Microsporum	Health professions
Sporothrix schenki	Farmers, gardeners, child caretakers
Blastomyces dermatitidis	Farmers, laboratories
Coccidioides immitis	Farmers
Actinomyces,Nocardia (Mycetoma)	Agricultural workers
Viruses	
Pox virus	Shepherds, farmers, veterinarians
Paravaccina Virus (milker's noduli)	Milkers, farmers, veterinarians
Herpes simplex virus	Medical staff
Human Immunodeficiency Virus	Medical staff, police

(associated with epidermal keratinocytes), and malignant melanoma - from pigment-forming cells. In many industries and professions, the incidence of skin cancer is higher than that of the general population **(Table 7)**.

Basal cell carcinoma (Carcinoma basocellulare)

Basal cell carcinoma is the most common non-melanocytic skin carcinoma. It is due to proliferation of the basal layer of the epidermis. It can be seen as a solitary nodus or eroded as ulcus (Ulcus rodens). Sun-induced carcinomas are localized on the head and neck.

Professional arsenic exposure (gold mines) can cause multiple superficial basal carcinoma in unexposed locations as well.

Carcinoma spinocellulare (Squamous Cell Carcinoma)

After many years of recurrent sun exposure, skin keratinocyte abnormal proliferation is provoked so-called Actinic keratosis. Some of the actinic keratoses develop as spinocellular carcinoma of the skin, which can metastasize to the lymph nodes or the hematogenous dissemination pathway. The association with the profession is one of the oldest listed occupational diseases.

Spinocellular carcinoma can also develop in the non-exposed parts of the skin - under the influence of petroleum distillates or tar, e.g. scrotal carcinoma in chimney sweepers. This type of carcinoma is also described as a post-traumatic result of repeated mechanical trauma or burns.

Diagnosis: Clinical changes responding to non-melanocytic occupational skin cancers with mandatory histological confirmation.

Treatment: surgery, radiation, etc.

Prevention: reduction of injuries and interruption of long-term contact with carcinogenic substances. Personal precautions include personal protective clothing. Particular attention is paid to workers with a history of atypical dermatitis, scarring, seborrhoea, sun allergy. If necessary, change of work in time. Regular prophylactic examinations and studies for the detection of precancerous conditions.

Malignant melanoma (Melanoma malignum)

A relatively rare pigment tumor that develops as a slowly growing node in the solar lentigo or as a new rapidly developing black nodule (nodular melanoma). Multiple development can also be observed for several years as superficial spreading melanoma.

All pigment tumors of pigment cells develop in the exposed parts of the body on pre-existing actinic changes. Outdoor workers are at increased risk for melanoma.

Malignant melanoma has an aggressive development, originated from pigment cells and always on a pre-existing nevus. The tumor is usually from a few millimeters to a few centimeters in thickness, brown or black in color, can change color, bleed or ulcerate. Criteria for poor prognosis are: nodular subtype, tumor thickness, metastasis, ulceration, bleeding, tumor duration, localization (body or extremities), male gender.

Diagnosis is necessarily confirmed by histological examination.

Treatment: surgery, chemotherapy, biological therapy (Alfa Interferon, Interleukin-2) alone or combined.

Prophylaxis: systemic dermatoscopic monitoring of pigment lesions in patients in contact with chlorine, propane, synthetic adhesives, trichlorethylene, etc., sun exposure, etc. Use of individual precautions.

Tab. 7. Risky Professions (by D. J. Birmingham)

Carcinogenic materials or agents	Industry or risk	Process or risk group
Resin, tar or products	Aluminum covering	Production of terracotta, pottery
	Coal, gas, coke	Coke ovens, tar distillation, coal gas production, resin loaders
	Open production of fuels	Production of briquettes
	Asphalt	Road construction
	Using creosote	Running bricks and tiles, Woodworking
Soot	Chimney sweepers	
	Rubber production	
Lubricants and cooling oils	Glass blowers	
	Refining of shale oil	
	Cotton processing	Pipe cleansing
	Paraffin processing and waxes	Typesetters, turners working with automatic machines (cooling oils)
	Technical activities	
	Oil refinery	Distillers
Arsen	Arsenic insecticides	Production and use
Ionizing radiation	Radiologists	
	Others working in the environment	
Ultraviolet radiation (256-320 nm)	Running outdoors	Farmers, fishermen and others.
	Industrial UV radiation	welding, voltage arc, UV lamps, cutting, printing

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11 PROFESSIONAL DISEASES OF THE VISUAL ANALYZER

V. Zlateva

11.1 PHYSICAL AND CHEMICAL AGENTS AND LOAD

In spite of its many protective mechanisms, the vision is extremely sensitive to professional impact. In recent decades, the production and use of appliances that are sources of different types of non-ionizing and ionizing radiation have increased for industrial, military, consumer, cultural and medical purposes. At a given frequency, wavelength, power, and duration of exposure can cause different lesions in the human eye.

A large number of workers are in contact with chemicals. The possibility of eye diseases as a result of acute intoxications is now reduced at the expense of the chronic effects of low concentrations.

The main groups of professional factors that affect the eyes are:

I. Physical factors of the working environment

II. Occupational intoxications affecting the visual organ

III. Occupational work associated with increased visual load.

I. Damage to the visual analyzer by physical factors

The nature of the damage to the eye depends on the prevailing or isolated effect of one or other part of the spectrum on the radiation energy stream and on the duration of its action. In production conditions, the predominant effect of certain rays can also be observed. This explains the specificity of eye loss in persons with different occupations.

Ultraviolet radiation in production conditions

Rays with a wavelength of 200 nm to 280 nm (UV-C) have the strongest biological effect of denaturation of cellular proteins and lipids; the rays with a wavelength of 280 to 315 nm (UV-B) have a pronounced anti-rachitis effect, and with a wavelength of 315 to 400 nm (UV-A) they are biologically weakly active. Ultraviolet rays act on a photochemical mechanism on the eye structures. Industry is dominated by sources of ultraviolet rays with a wavelength of 200 to 280 nm (UV-C) - oxygen- and electric welding, electric furnaces, converters, apparatuses in quartz radiation cabinets, etc.

Workers in the metallurgy are at risk - scumbers, foundries, converters; those working in the production of radio lamps and mercury rectifiers, and in particular electric welders, as well as technicians and medical practitioners working with mercury-quartz lamps. Under intensive irradiation occur photoerythemas, photodermatitis, general toxical symptoms by affecting the central nervous system - headache, dizziness, hyperthermia, effects of agitation and the like. Electro-ophthalmia develops in the eyes. In the presence of photosensitising substances - the distillation of coal (anthracite, anthraquinone) and oil (resin, paraffin, fuel oil, etc.) may develop photosensitivity with frequent ophthalmia, combined with chronic dermatitis, folliculitis, eczema, atrophic changes etc.

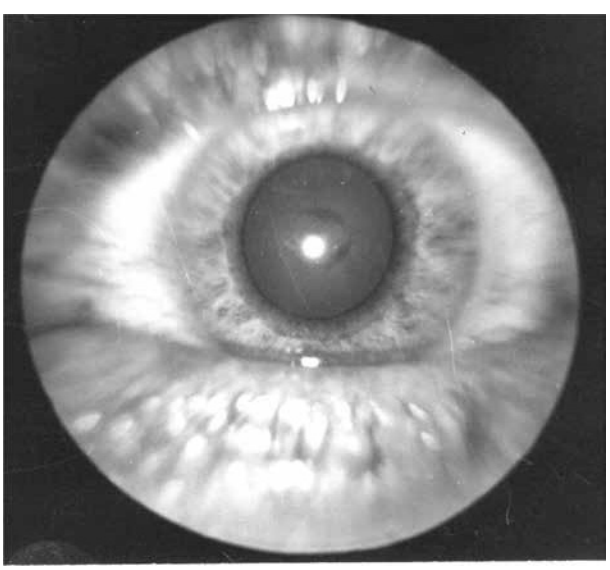
One of the most common diseases of the eyes by the impact of radiation energy is **electro-ophthalmia**. It occurs as a result of UV radiation during gas, arc or plasma welding and cutting of metals. Threatened occupations are welders, medical staff in physiotherapy cabinets.

Pathogenesis. The cornea and the conjunctiva absorb the great part of the UV rays - the cell nuclei, in particular the nucleoproteins, are predominantly damaged. Photochemically active UV rays practically do not cause serious changes in the retina with short-term effects.

Clinical picture: Inflammatory effects of the cornea and conjunctiva occur usually after a latency period of four to twelve hours. Symptoms include light-headedness, pain, tear, blepharospasm, hyperemia, and conjunctival edema. When the effect is short-lived or of lesser intensity and the corneal epithelium is not affected, the events resolve for 12 to 18 hours. When the epithelium is affected, biomicroscopy reveals a swollen cornea, sometimes with thin villi and a scarified surface layer. The vision is re-

duced as long as the acute phase lasts. Only one eye is extremely rarely affected.

Repetitive UV irradiation, develop a chronic form of elektroophtalmia, expressed in increased sensitivity to light and the effects of chronic blepharoconjunctivitis. Chronic UV effects on the eyes may be damaged and the lens - most often at a wavelength greater than 290 nm - UV-C (below 290 nm UV-rays are absorbed by the cornea and the front-camera fluid). **Occupational cataract** is a disorder of lens transparency caused by prolonged exposure of working environment factors. The lens of the eye is the only formation of the body that has no mechanism to release it from normal, damaged or unviable cells. Irradiated abnormal cells remain there, which is why it is sensitive to chronic irradiation. In **specific UVcataract** mainly rear cortical layers centrally are shaded, as in the initial phases changes are more gentle and better constrained by the transparent lens and at the evolution of the process feculences coarsened and distributed at periphery. This evolution is slow and is maturing for several years. When the changes are centrally located and are too dense,



Pic. 1. UV cataract

visual acuity decreases early in a narrow pupil (normal conditions) **Pic. 1.**

Cataract from infrared radiation in production conditions

IR rays are the part of the EM spectrum with a wavelength of 760 nm to 1 mm. It has been assumed that, because of their low energy levels, when absorbed by the tissues, the IR photons lead to an increase in the kinetic energy of the system - the radiant energy becomes heat.

The defeat of the lens of IR radiation is the subject of a detailed study of the early twentieth century for many years, and the name "cataract of glassblower" described by Wenzel in 1917 remains common name for various types lens changes in individuals working with heated bodies.

Threatened professions are metallurgists in various industries having contact with large surfaces of heated metal - for metal rolling, production of slabs, of cast iron founders; workers in the glass industry; Bakers, bakers, chefs and more.

Pathogenesis: According to Focht, the cataract develops as a result of the high temperature effect on the entire front part of the eyeball. The increased temperature of the tissues, especially the ciliary body, disturbs the feeding of the lens.

Clinical picture: The disease usually starts in one eye. At the rear pole of the lens there is an opacity in the form of a wedge or pan, abruptly limited by the strong lens. Opacity develops slowly for years, but early reduces vision because the cataract is centrally located. The magnitude of the opacity initially corresponds to the area not covered by the iris, i.e. of the broad pupil. It is also characteristic for the deposition of the surface layers of the lens capsule which are curved forward and may also "swim" in the anterior chamber. This condition persists for many years, but progressive development may also be observed with whitish non-homogeneous opacity in the front of the lens. Subsequently, the second eye is also diseased. This cataract occurs usually in older workers, as the sclerosed lens absorbs to a greater extent IR rays.

Cataract from the impact of ionizing radiation

It was first described in Halupetsky in 1897.

Threatened professions are gamma-defectoscopes of metals, the use of radioactive isotopes for control of production processes, research, use for production purposes of powerful high-voltage and accelerator apparatus, nuclear reactors, in medicine for diagnosis and treatment.

The lens is highly sensitive to penetrating ionizing radiation (especially to X-rays and gamma rays, but also beta-rays, neutrons). Opacity of the lens develops after prolonged latency (2-12 years). The cataract-induced absorbed dose in 1-5% of exposed individuals is assumed to be 5 Gy.

Pathogenesis: All types of ionizing radiation causing lens opacity cause similar lesions and therefore these cataracts may be clinically and histologically separate from the many other lens opacities. The primary effect of the rays is on the anterior epithelial cells having a high exchange and on the young fibers that are formed by them. These new fibers are located directly under the lens capsule, so the earliest damage should be sought there.

Clinical picture: Biomicroscopic study shows that the first opacities occurs at the back of the lens in the axial zone. In this place a polychromatic over-

flow appears on the background of which are visible vacuoles and tender graininess. Later the opacity becomes disk shaped in the posterior pole of the lens subcapsular. The disk of this opacity consists of several concentric layers. The complete cloudiness of gamma-lagers resembles the opacity of asbestos. Radiation cataract is characterized by clearly limited areas of cloudiness from the transparent areas. Later the specific picture is cloudy.

Electrical cataracts

Caused by high voltage electrical currents, atmospheric electric discharge, electric shock. The name electrical cataract encompasses several pathogenetic types that are not exactly distinct. In most cases it is about electrolysis due to the passage of an electric current through the eye or the heat that forms in it. Pathogenesis is sometimes unclear.

Clinical picture: The development of cataract and the corresponding decrease in vision develop after a latent period of 1 to 2 years. Opacity are located at the beginning subcapsular, sometimes in the front lens capsule. In form may be diffuse, punctuate, striped, often take the form of a rosette. Sometimes they remain unchanged for a long time, but they often progress to full opacity and blindness.

Differential diagnosis of occupational cataracts is mainly due to pathological and complicated cataracts, but with either a common disease or pre-existing or concomitant eye disease. Diagnosis is mandatory after an examination of patients with a biomicroscope mydriasis, as well as information on work experience and contact with occupational injuries.

The treatment of professional cataracts is operative. The possibilities of modern eye surgery allow the surgery to take place when the normal life and work of the patient are disturbed. Since there are no changes in eye bottoms, the surgical result is usually good. After appropriate labour expertise, workers may continue their employment under a regime consistent with post-operative outcomes.

Prevention: In the case of electric welding, the rays act harmful not only to the eyes but also to the exposed parts of the head - this is why not only safety goggles (in the case of gas welding) but also protection with a manual shield (screen). A shield attached to the head is also recommended when welding in hydrogen using argon or helium. IR rays separated from the source of radiation can be absorbed or reflected. The design of most safety glasses is based on their absorption by incorporating one or several chemical compounds (bivalent iron, chromium, nickel, etc.) into the glass (in the liquid glass mass). For X-ray and gamma rays-lead are used to protect, in glasses and glass-screens.

Effects of laser radiation on the eyes

Laser is the source of radiation in the optical range of the EM spectrum - infrared (IR), visible, and UV-radiation.

The eye is seen as a critical organ for laser radiation. The front eye segment is designed to concentrate and direct the rays to the retina.

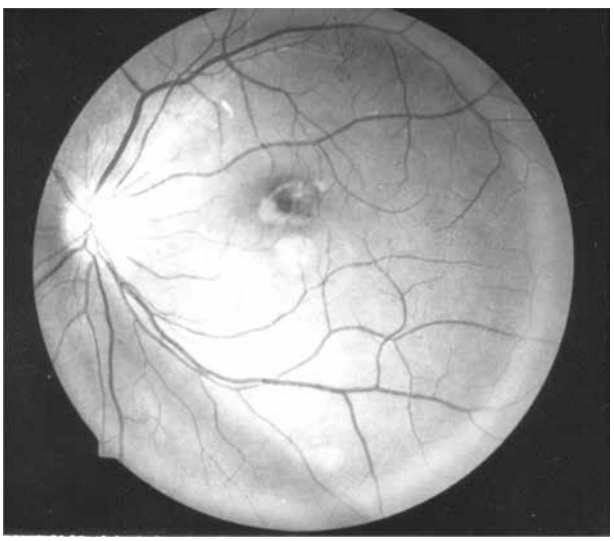
Threatened professions are in machine building - for cutting metals and hard materials, welding; in geodesy; for environmental control - deformation of the earth's crust, air pollution, water, etc.; in medicine - in ophthalmology, surgery, physiotherapy; in science - for holographic records, super-surface control, optics, microelectronics; for military purposes and others.

Laser beams can get into the eyes of laser-powered devices with direct or diffuse and reflected laser radiation. In the laser application technology and in the setting of laser devices using high power lasers, the damage to the eye tissues is most often caused by direct or mirrored reflected rays, - i.e. direct eye laser trauma. A second group of eye injuries is caused by diffuse laser light - these lesions are defined as indirect.

In recent years, using high-performance metal cutting lasers (Nd: UAG and CO_2 lasers), workers are exposed to reflected and scattered light not only in the act but also in the maintenance and service of the lasers. The effect on the eyes also depends on the material being handled. Nearly all types of lasers emitting in the visible or nearby IR spectrum (IR-A - between 400 and 1400 nm) are so bright that they pose a danger to the eye. Exceptions make some very small low-power semiconductor lasers that emit only a few microwatt power. As a result of focusing light on the cornea with wavelength of the visible and IR-A spectrum, its energetic density on the retina increases sharply.

Occasional incidents occurring during laser work are associated with reduced visual acuity as a consequence of burning the macula **(Pic. 2)**. Especially sensitive is the pigment epithelium of the retina in whose cells the most intense absorption is, and the heat released therein can lead to severe burns, and loss of vision. With direct laser beams, the **clinical picture** is very characteristic. Besides oculorbital pain at the moment of falling, followed by a feeling of dazzle, there is often a feeling of heat in the head and discomfort. Depending on the weight of the burning of the macula, transient or permanent central scotomas may be obtained, including a complete drop in the possibility of fine visual activity. Angio-fluorographic studies contribute to both diagnosis, differential diagnosis, and prognosis of impairment.

Not high energy densities cause reversible changes, but re-exposure can cause damage to the retina and other eye tissues (various eye structures can be



Pic. 2. Burning of the macula due to laser trauma

damaged depending on which structure absorbs the largest radiation energy for volume tissue).

The treatment is dependent on the severity of the lesions. With large edema in the macula - dehydrating agents such as manitol, glucose and local retrobulbar corticosteroids.

Prophylaxis: A basic safety device is suitably designed spectacles and the non-direct alignment of the laser beam to the eyes.

II. Damage to the visual analyzer by chemical factors

Chemical factors in the working environment can affect either the eye structures or secondary damage due to general damage to the body. Eye symptom is sometimes so characteristic that it contributes significantly to the early diagnosis, prophylaxis and therapy of occupational intoxications.

Occupational risk. The workers in the chemical, pharmaceutical, cosmetic, perfumery, alcohol, leather, furniture, woodworking industries, in the extraction, processing and application of heavy metals, pesticides, synthetic fibers, organic solvents, etc., are threatened by the damage to the visual organ.

Eye symptoms of **lead poisoning** are not observed in isolation but are accompanied by other signs of saturnism. It is possible to lower vision to complete blindness of both eyes. In case of lead encephalopathy (currently not present), visual disturbances are central - central hemianopsia or amaurosis with conserved pupillary responses. In the field of view, there are also changes - homonimic haemianopsia, central scotoma, concentric narrowing.

In chronic intoxication, lead is deposited subepithelial in the cornea. Optic neuritis with papillary edema and tightly narrowed vessels with retinal haemorrhages and focuses (reminiscent of retinitis albuminurica) can be observed. Retrobulbar neuritis or papillitis is usually a consequence of vascular lesions due to spasm, sclerosis or obliterating endarteritis. Paralysis of m.abducens, nystagmus and disappearance of pupil light reactions may occur. Vascular changes are often seen - atherosclerosis, endarteritis, hyaline degeneration. It is believed that the damage is due to primary degenerative changes in the optic nerve and also to vascular changes. Probably both factors work.

Ocular symptoms of **mercury poisoning** are most often not the direct effects of mercury. They are secondary to kidney damage. There are atrophic foci in the retina, bleeding, edema in the course of the vessels. In case of chronic poisoning inflammation of the tenon capsule, retrobulbar neuritis, endogenous deposition of the metal in the Descemet's membrane or corneal endothelium, in the conjunctiva and more often in the anterior sub-capsular layers of the lens can be observed.

Atkinson's Symptom - a peculiar color reflex from the front surface of the lens is found in people who have been continually working with mercury and its compounds. Biomicroscopic picture shows that the front of the lens has a glare that changes its color from brown to dark reddish brown. It is believed that this mark is a permanent and early diagnosis in chronic mercury poisoning. Visual acuity is normal.

In case of **methyl alcohol poisoning**, eye symptoms occur on the second-third day of acute poisoning when the patient is already out of the severe general condition. They appear fog in front of the eyes, and after a few hours the visual acuity sharply falls, often to complete blindness. The pupils are wide, with a faint reaction of light, a central scotom appears. In the optic floor there is a hyperemia of the papilla of the optic nerve, sometimes with large swelling and bleeding around the vessels and the vitreous. Later there is an atrophy of the optic nerve. Visual improvement may also occur after 4-5 weeks with appropriate treatment and in less severe cases.

Chronic poisoning with methanol vapors, along with headaches and gastrointestinal disorders, can also lead to eye damage - retrobulbar pains, which increase in eye movement and pressure on the eyeball; pupil extension; paralysis of the external eye muscles with diplopia; upper eyelid ptosis; absolute or relative central scotom. Ophthalmoscopy shows inflammation of the optic nerve with significant swelling and enlargement of the retinal veins or rapid early-onset paleness of the papilla. Chronic cases may also cause retrobulbar inflammation of the optic nerve leading to atrophy. There is individual sensitivity to methyl alcohol.

Aniline poisoning shows blue or yellowish brown conjunctival and corneal epithelial cleft colouration, less corneal erosions, and parenchymal opacity. If aniline is directly in the conjunctival sac, tissue necrosis can also be induced. Keratitis stay for many weeks, healing with solid leukomas. In the optic floor, bluish optic papillary staining and dilation of the venous vessels may appear. In severe poisoning complications are haemorrhages and veno-thromboses, rarely neuritis of the optic nerve with narrowing of the field of vision, central scotom and subsequent atrophy. If aniline is in the conjunctival sac, a flush should be made and then sub-conjunctivally injecting vitamin C. Antiseptic ointments (Vitaform, Nemybacin eye ointment 2-3 x daily) are placed.

In case of **carbon monoxide** poisoning, paralytic ptosis, paralysis of the external ocular muscles, nystagmus, conjunctival hyperemia, mydriasis or myosis may occur. In more severe cases, edema and haemorrhage in the retina, venous thrombosis, optic neuritis, homonymous hemianopsia, chromatopsias - seeing in yellow. In rare cases, carbon monoxide poisoning may result in almost complete vision loss, with the maintenance of normal pupil responses, most likely due to damage to the visual center cells in the occipital brain part. The disorder may be transient or lead to permanent blindness. Sometimes they are accompanied by visual hallucinations.

Upon poisoning with **hydrogen sulphide** and at negligible concentrations in the air, eye pain occurs, and at higher concentrations - photophobia, blepharospasm, tears - "hydrogen sulphide ophthalmia". As a result of corneal edema, the patient sees colored circles around the light source. The corneal epithelium is raised in the form of bubbles and easily slides. Chronic hydrogen sulfide impact a chronic recurrent keratoconjunctivitis occurs.

In **carbon di sulphide** poisoning, irritative and inflammatory changes in conjunctivitis, fine epithelial lesions in the intereyelid slit which occur with lacrimation, blepharospasm, light-beating ("the eye of the spinner") are observed. The cornea has spotted gray infiltrates that fall apart after a few days. The process occurs in the surface layers of the epithelium, so there are no scars in the cornea. If secondary infection occurs, ulcerations with hypopyon-irritis and secondary glaucoma may occur. Weak corneal and pupillary reflexes are observed in severe poisoning. Retrobulbar neurites, oculomotor palsy, ophthalmoplegia interna, and color vision may appear in chronic intoxications - the injured person sees in yellow or red. Retinal capillary micro-aneurysms, rarely complicated with exudates and haemorrhages - retinopathia sulfocarbonica of Japanese authors, are observed in the optic floor.

Ocular symptoms of **dimethyl sulphate** intoxication are characterized by severe pain and light-headedness. The conjunctiva is hyperemic, swollen. Lacrimation is abundant. Visual acuity is reduced due to edema of the corneal epithelium. Biomicroscopic at the beginning are superficial point-like opacities, and in severe forms - epithelial sliver from the upper corneal parts. The pupils are narrowed. Acute inflammatory events in conjunctiva resolve for 5-7 days, but in the cornea there are opacities in the stroma that are difficult to spread.

When liquid dimethylsulfate falls into the eyes, severe burns with loss of tissue and deep corneal infiltrates, often leading to blindness, occur.

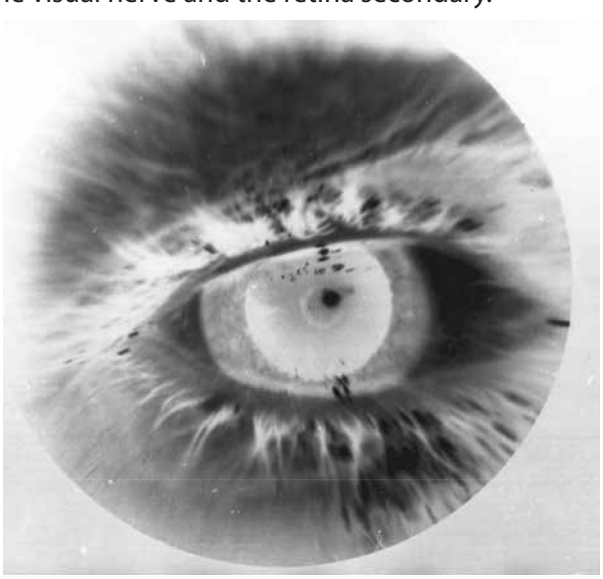
Trinitrotoluene cataract is the most characteristic manifestation of **trinitrotoluene** (TNT) on the optic analyzer.

Trinitrotoluene cataract is often the only symptom on the basis of which retrospectively can be judged for past intoxication. It has four stages of development. At first, at biomicroscopy in full mydriasis, scattered gray-whitish opacities are observed in the corneal layer of the lens, which gradually group together to form an uncompleted ring. In the next stages the ring thickens by forming triangles with peaks to the center. A tender central ring also appears. At the last stage, the available opacities are increasing in size. The triangle tips are moved to the central ring **(Pic. 3)**.

The vision diminishes. Treatment is operational.

Eye damage due to poisoning with pesticides.

Pesticides may have either a direct irritant effect on the eyes or, in the case of chronic effects, damage the visual nerve and the retina secondary.



Pic. 3. TNT cataracts

Chloro-organic compounds, due to their fat-soluble properties, can affect the visual nerve, as toxic neuropathy occurs with decreased vision, light- and

color perception. In severe cases, atrophy of n. Opticus with loss of vision. Chronic effects cause conjunctival irritation with tearing and photophobia.

Phosphoorganic pesticides such as cholinesterase inhibitors in chronic intoxication may provide autonomic optic neuropathies, resulting in bilateral decrease in visual acuity, concentric narrowing of the field of vision without or with central scotomas, red color vision. In more than 50% of cases, papillary hyperemia and congestion are observed. Rarely ends with papillary full atrophy. In acute intoxication, conjunctival hyperaemia, spasm of the eyelid muscle, myosis, ciliary muscle spasm with focal disturbances were observed. In severe cases transient amaurosis may occur.

Dinitrophenol pesticides, whose mechanism of action involves dissociation of oxidative phosphorylation with subsequent energy balance disturbances, selectively affect the ophthalmic lens in chronic intoxication. The specific cataracts are two-sided and start as fine gray opacities with spotty character on the front lens capsule. Opacity develop rapidly and sometimes lead to secondary glaucoma due to swelling of the lens. In acute intoxication, dilation of retinal vessels, as well as haemorrhages, is observed.

Methyl-mercurial pesticides - in addition to pronounced oral hypoesthesia and polyneuropathy, ataxia, speech disorders, a narrowing of the field of vision with intact central vision is observed. Occasionally, eye changes progress to blindness, especially in the terminal phase of the disease. More often, small scattered scotoms, disturbed vision, xanthopsia, etc. are more common. Atrophy of the brain cortex in the area of Sulcus calcarinus at postmortem study.

Diagnosis. In addition to specific eye changes and increased incidence of unilateral eye pathology among workgroups, the elimination and exposure test and response to detoxification therapy are often used to prove the diagnosis. Important in differential diagnostics is the bilateral disability of eye injuries. Specific eye symptoms and evidence of chemical agents or their metabolites in biological media help diagnose.

Treatment. If the anterior eye segment (keratitis, etc.) is affected, it is necessary to cause mydriasis (Sol Atropin - 1% collyr, Mydrumcollyr), drop antiseptic collars and ointments. (Ophthalmoseptonex collyr - 4 x 1-2 drops, Sulfacetamid 20% collyr, Chlornitromycin ung. opth.). Nonspecific anti-inflammatory agents - calcium, butapirin, vitamins, possibly corticosteroids and antibiotics are also used. In neuritis - dehydrating, detoxifying and anti-inflammatory agents, group B vitamins; in the chronic stages - nivalin, strychnine, dibazone, vasodilators, tissue therapy and others.

Prevention. Persons with chronic ocular disease are not allowed to work. In the case of established occupational eye diseases, post-treatment is appropriate temporary rehabilitation without contact with toxic substances. In case of recurrences or more severe complications, the possibility of re-qualification and determination of loss of working capacity should be provided.

III. Increased visual acuity load

Progressive short-sightedness

The great interest in short-sightedness is determined by the fact that it is the most common cause of visual acuity fail, while, according to various authors, between 10% and 25% of the adult population of the world have myopia. Also, myopia most often disrupts man's ability to work from an early age, thus limiting the choice of a profession.

Progressive short-sightedness is a severe illness that leads to a sharp reduction in vision (even after correction), and complications accompanying it lead to disability and sometimes to blindness.

Etiology and pathogenesis. Progressive short-sightedness is thought to be the result of the pathological extension of the sagittal axis of the eye due to prolonged nearby work and accommodation stress. The hypothesis (Kuschel, Filatov, Radsycshovsky) is plausible, according to which myopia develops as a result of weakening of the sclera as part of a general weakness of connective tissue. This attenuation is associated with physicochemical changes occurring in it, caused by metabolic disturbances in the eye (calcium metabolism disturbances are reported). Influence also exerts heredity.

Occupational risk. A major risk factor is work that requires continuous fixation of small objects from a small distance. Workers from the printing and textile industry, microscopists, designers, jewelers, watch makers and others are at risk.

Clinical picture. Long-range visual acuity is always reduced, and there is no strict correlation between its reduction and the degree of myopia. However, in the higher degrees of myopia, good visual acuity is not always achieved and after a proper correction.

Visual acuity for near is usually good because, in comparison to emetropes, the same size objects give larger images on the retina.

Muscle asthenopia - is due to the excessive effort of the internal straight muscles, realizing convergence, which is quite large in the proximal PP of the myopic eyes. It occurs in unpleasant sensations in the eyes and forehead after prolonged close work at medium and high degree of myopia.

"Flying flies" are often seen, when looking away on a well-lighten surface. Spasm of accommodation

is very common, especially in the rapid increase of myopia.

Objective changes. The lens at high degrees of myopia can be luxated. In old age, it gets a brownish hue in the kernel. The structure of the vitreous is split, with fibrils disintegrating. Opacities occurs, which may be due either to bleeding or to the collection of disintegrated fibrils.

In the optic floor, a "myopic cone" may be observed - a semilunar white or yellowish area. Severe alterations may occur in the area of the macula - white spots appear as a result of atrophy of the choroid, and pigment is gradually deposited around the atrophic areas. Pigmentation increases and an ophthalmoscopically visible oval or circular black spot - the so-called spot of Fuchs. Sometimes there are bleeding in the macula that is difficult to resorb. One of the most serious complications in myopia is ablatio retinae.

Prevention and treatment.

Prophylaxis of myopia involves the elimination of harmful externalities influencing its appearance and development, the timely detection and correction of myopia, proper wearing of eyeglasses and contact lenses, and the general strengthening of the body.

There is no need to remove young people with short-sightedness from work. For workers with advanced myopia, appropriate working conditions should be created - lighting, ergonomic workplace, correction of vision.

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11.2 ACUTE OCCULAR TRAUMAS

Professional ocular traumatism is a current and serious problem for ophthalmology due to its high frequency, variety and often unfavorable functional outcome of vision. Includes bodies in eye contusions, penetrating and non-penetrating foreign bodies in eyes, conjunctival and corneal erosions, eyelid ruptures, retinal and optical nerve damages, chemical and thermal burns.

Particular attention should be paid to eye traumas to the anamnesis. A good history can help both the effective treatment and the precautions for other workers in this production.

Even in cases where only one eye is thought to be affected, it is imperative to carefully examine both eyes. If the accompanying trauma of the eyelid impedes the eyesight of the eyeball, a single sterile local anesthetic may be used.

tissues, their destructive action can last for hours if they are not washed away by the eye.

In contrast, **acids** precipitate tissue proteins and quickly create a barrier against deep penetration, resulting in burning more often localized within the contact area.

The corneal endothelium, which is extremely important for its normal structure and function, is very vulnerable to chemical agents. If anterior chamber and drainage system is affected, secondary glaucoma may occur. Obliteration of conjunctival and sclera blood vessels may lead to severe anterior ocular ischemia.

It is almost impossible to predict the outcome of alkaline burning immediately after the accident - the degree of damage to the eye tissues and the eventual outcome of burning can only be estimated within 5-6 days.

I Type of trauma	II The severity of the trauma	Type of injured agent	IV Type of injury
Industrial	Lightweight	Chemical	An injury
Agricultural		Mechanical	Unpunctured without or with a foreign body
Transport	Moderately heavy	Thermal	
Sport		Other physical factors	Punctured without or with a foreign body
Others	Heavy		Destruction of the eyeball
V Trauma characterization			
Depth of trauma, concomitant lesions, presence of blood in the anterior chamber and vitreous, prolapse and loss of ocular tissues			

I. Chemical injuries

Chemical burns are among the most urgent eye injuries and are caused in the production environment by bases and acids or by chemical compounds of a basic or acidic nature. If they are not in high concentrations and if the medical help is timely, the disabilities are transient. However, there are also severe production eye burns that lead to loss of vision.

Etiology and pathogenesis: The most serious chemical burns are caused by: **alkaline** materials - sodium, calcium, potassium bases, ammonia, cement; **acidic substances** - sulfuric, acetic, sulfurous acids, industrial solvents, pesticides and others.

Alkaline burns are heavier than those with acids due to their rapid penetrating effect through the cornea and the anterior chamber (usually less than 1 min) due to the effect on the lipids of the cell membranes. Because of their slow neutralization by

The classification and prognosis of chemical burns of the eye are related to the degree of incineration:

CLASSIFICATION OF CHEMICAL BURNS OF THE EYE

Type (weight)	Clinical findings
Lightweight	Erosion of corneal epithelium, slight corneal edema is missing Ischemic necrosis of the conjunctiva and sclera.
Average	Corneal opacities engaging the stroma with increased corneal thickness, minimal ischemic necrosis of the conjunctiva and sclera, superficial neovascularization.
Heavy	Strong corneal edema, corneal opacities to full opacity, severe ischemic necrosis, and conjunctival and sclera whitening.

The treatment is conducted in three directions:

1. Emergency aid, aimed at removing the substances in the eye.

2. Prevent the development of infections or cornea breakthroughs.

3. Achieve maximum recovery of visual functions.

Immediately flush with water at the workplace. Health eye care should also be provided with physiological serum to ensure that the conjunctival sac is clean (in the presence of small particles of the chemical they must be carefully removed with a wet pad). Place an antiseptic ointment in the eye. The patient must be referred to an ophthalmologist for continued washing with a physiological, isotonic or neutralizing solution, including after reversing the eyelids. A single and sterile local anesthetic may be used.

Depending on the condition of the ophthalmic tissues, appropriate therapeutic approaches, including surgical, are applied.

II. Thermal injuries

Thermal burns, unlike chemical ones, are in a moment. The effect of defeat depends on the temperature of the substance. More often, the lyelid's skin is affected by the blinking reflex protecting the eyeball. Often, splashes of molten metal are fall directly into the eye when casting into molds, as well as hot metal particles (in forging workshops). Light metals - e.g. zinc, zinc alloys, have a low melting temperature and fall into the eye they cool quickly and stop their thermal action. However, if it falls in the eye of molten cast iron or steel (over 1000 ° C) while it cools, it continues to penetrate deep into the tissues and subsequently form deforming scars.

Treatment: For slight eyelid's skin burns, an antibiotic ointment and a sterile dressing are provided. With wider skin lesion damage, subsequent scars may lead to corneal desiccation due to insufficient eye lid closure. In order to protect the bulb from mechanical damage from rough scarring, trichiasis and the like a soft hydrophilic contact lens is placed on the cornea, or early plastic surgery is performed on the eyelids.

III. Mechanical traumas

Eyeball contusions. In production conditions, they can be caused by powerful air waves, water jets, various solid objects, and more. Dull traumas range from ecchymosis to severe intraocular damage: hyphema, lens subluxation, iridodialysis, traumatic mydriasis, haemophthalmia, edema and retinal haemorrhage, choroid rupture, retinal detachment can be observed.

The main goal of the treatment is to quickly restore haemodynamics and eye hydrodynamics and overcome local ischemia.

Foreign bodies in the cornea. In many professions such as turning, milling, carpenters, blacksmiths, machine and auto mounters, etc., when working without safety glasses or screens, metal shavings are often found in the eye. The worker must be sent immediately to an optician's office. Unlike burns where mutual assistance is urgent, it is not necessary to take the foreign body by at hand means.

Puncture eye injuries. They are one of the worst traumas. Three main points characterize puncture through eyeball injuries: the site of injury, the amount of rupture, the presence or absence of an intraocular foreign body. They are most often accompanied by the loss of intraocular tissue and the contents. Rare injuries only affect the cornea and the sclera. In most cases they are accompanied by damage to underlying tissues. Immediately occurring complications are most often haemophthalm, hyphema and traumatic cataracts.

To the obligatory questions of foreign bodies that have pierced the eyeball is the type of material used: magnetic - iron or steel, non - magnetic - aluminum or copper, organic - wood or plastic as this information is of particular importance for determining the method of treatment and prognosis. Soluble metal salts of iron and copper, contained in foreign bodies can cause irreversible toxic damage to the retina if not removed in time. More inert materials such as aluminum, plastics, glass have a better forecast. Organic foreign bodies such as wood, parts of plants can cause severe intraocular infection with unfavorable prognosis.

TYPES OF FOREIGN CELLS		
METAL		NONMETAL
Toxic	**Nontoxic**	
Iron	Gold	Stone
Copper (bronze, brass)	Silver	Glass
Mercury	Platinum	Porcelain
Aluminum	Tantalum	Coal
Nickel		Some plastics
Zinc		
Lead		

Magnetic foreign bodies are removed by a magnetic device. Immagnites are removed with a special pinch for microsurgical subtraction.

The technique by which foreign bodies are localized in various eye structures includes biomicroscopy, ophthalmoscopy, X-ray, ultrasound diagnostics, scanner, and more.

The inability to remove iron and copper foreign bodies can cause severe visual impairment due to their toxic effect on the eye tissues. Inert materials, such as glass or plastics, cause mechanical damage

to the eye, but the absence of a local toxic reaction makes their long-term prognosis better (some of them may not be taken depending on their position in the eye). Foreign bodies can cause severe intraocular infection and lead to loss of eyeball.

As with most ocular incidents and also eyeball injuries, the worker should refer to an ophthalmologist. It is imperative to place a sterile dressing, anti-tetanus prophylaxis, analgesic therapy, haemostasis medication. Puncture eye injury is an urgent condition requiring surgical treatment.

One of the most serious complications of eye trauma is **"sympathetic ophthalmia"**, which occurs in the healthy eye, most often after a puncture injury involving the ciliary body. It occurs in 0.2 to 1% of the eye trauma. If immediate treatment is not initiated, sympathetic ophthalmia may result in complete vision loss of both eyes. Ophthalmia sympathica has been found to develop in 50-60% of cases after 3-12 weeks of injury, in about 80-90% of cases during the first year after the trauma, and only 10-20% at a later time.

Ophthalmia sympathica is believed to be an autoimmune disease arising as a result of sensitization to the antigen of the outer membrane of photoreceptors. This rarely autoimmune inflammation of the second eye can be prevented by rapid and adequate treatment of the puncture injury. Ocular traumatism leads to temporary or permanent, partial or total **disability**.

Prevention of industrial eye traumatism is limited to a system of events relating to the organization and rationalization of manufacturing processes, to professional selection and qualification, personal protection equipment, safety glasses, screening and good health education.

Knowing the damaging effect of chemical agents and protecting against the risk factors that may come into the eyes during work, the availability of washing and irrigation of the face and eyes at the workplace are important in the primary and secondary prevention of ocular traumatism.

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Zl. Panova

12 OCCUPATIONAL DISABILITIES OF FEMALE REPRODUCTION

Recent scientific data and analyzes indicate an increase in disease abnormalities in the reproductive function of women. It was found that one of the 7 married couples had infertility, approximately 20% of pregnancies ended with miscarriage, 7% of newborns were of lower weight and 7% were born with defects. It has also been shown that the relative share of loss of fertilized ova in both the pre- and the post-implantation periods is significant.

In working conditions in the working environment there may be harmful chemicals, noise, vibrations, high or low air temperature, electromagnetic waves, etc., as well as the woman being in a state of inconvenient working position, lifting weights, to endure nervous emotional intension, etc. All labour factors at a certain degree of intensity and duration may have an adverse effect on the reproductive function of the woman.

Characteristics of the female organism and reproductive physiology. The anatomical and physiological features of the skin, whose resistance to external effects changes during menstruation, pregnancy and climax, are important for the more intense introduction of chemical substances into the body of the woman. The characteristic morphological composition of the blood, the functional characteristics of the enzyme systems, its higher reactivity, and therefore, under the influence of the chemical production factors in women occur more often than men's adaptation failure with the development of anemic syndrome and other changes in health. The different sensitivity of the female organism to the factors of the labour and the environment are also due to some peculiarities in the intake, distribution and metabolism of toxic noxae and most of all - the presence of hormone-conditioned physiological rhythms. During pregnancy, the woman's microsomal system, incl. and the placenta and the fetus, can metabolize xenobiotic substances differently, which in some cases is important for the newborn and for the adequate lactation.

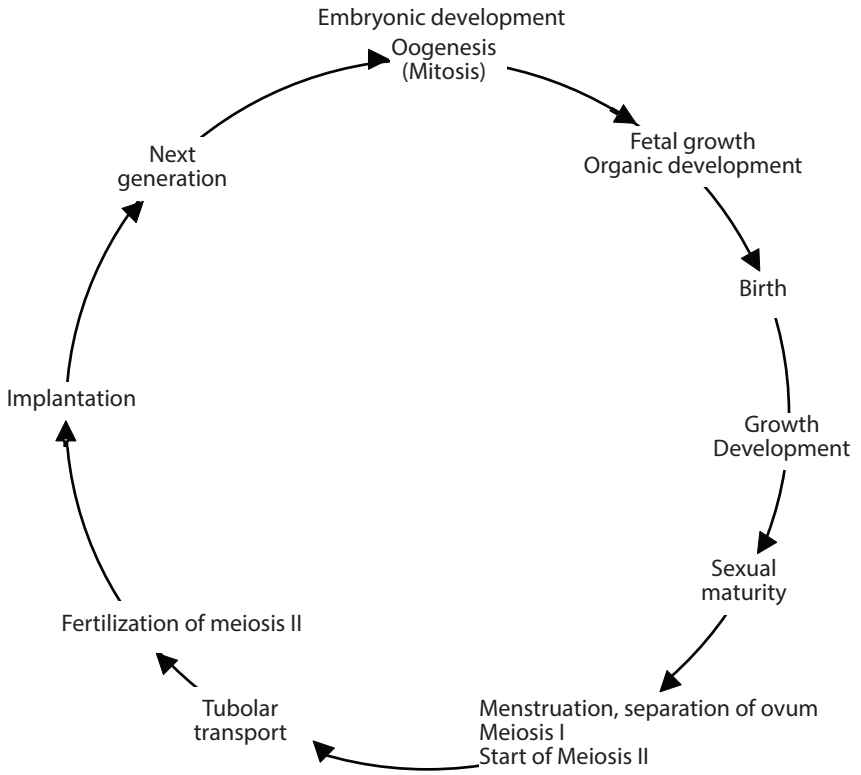
The reproductive cycle in the woman is extremely complex and is regulated by the neuro-endocrine system via the pituitary-ophthalmic axis **(Figure 1)**. There is a balance between follicle stimulating hormone (FSH), luteinising hormone (LH), estrogens and progesterone. For example, the intervention of toxic factors can occur at the earliest stage with a mutagenic effect (organic solvents, ethylene oxide, arsenic, nickel) or during tubular transport of the ovum, influencing the endometrium and inhibiting implantation accordingly.

A critical period for fetotoxic and teratogenic chemicals is the first trimester of embryo and fetal development. Cell differentiation leading to the formation of individual organs and systems can be disturbed and lead to malformations (spina bifida, palatum phisum), to abnormalities (skin growths, extra fingers) or functional deficits (delayed mental development). The second and third trimesters of pregnancy are characterized by toxic effects in the fetus, such as intrauterine maturation, skin defects, broad fontaneles. Functional disturbances in the endocrine, immune, urogenital and central nervous systems, which continue to develop during the second and third trimesters and are therefore easily vulnerable (lead, organic mercury compounds). In recent years, the transplanted passage of carcinogenic factors, as well as damage to germinative cells in parents in the pre-conceptual period (adenocarcinoma with diethylstilbestrol exposure), are not excluded. The question of the passage of some toxic agents into maternal breastmilk (perchlorethylene, halogenated hydrocarbons, dieldrin) is also not debatable. Some physiological changes during pregnancy can get worse. For example, in some disposition the presence of organic solvents may lead to preeclampsia in pregnant women.

In summary, the reproductive outcome associated with women's exposure to harmful production factors is manifested as:

- menstrual disturbances;
- changed fertility;
- single gene defects;
- chromosomal defects;
- spontaneous abortions;
- congenital malformations;
- intrauterine growth retardation;
- late fetal death;
- changed gestational time

Fig. 1. Reproductive cycle in a woman



- changed sex ratio;
- perinatal mortality;
- troubled development;
- behavioural disorder;
- carcinoma;
- carcinoma in childhood;
- female genital carcinoma;
- weakened libido;
- premature menopause.

Potential biochemical mechanisms of exposure to harmful production factors are associated with:

- mutations or chromosomal abnormalities;
- hormonal imbalance;
- direct cytotoxic effects;
- causing cell death;
- a defeat in cell interactions;
- reduction of enzyme biosynthesis;
- obstruction of morphogenetic activity;
- mechanical destruction of cells;
- interference through energy metabolism;
- depletion of energy;
- race in the location of the receptors.

When discussing the abnormal normogenesis of the fetus, it is not necessary to absoluted the harmful factors of production, as other causes - genetically determined, fetal hypoxia, rhesus conflicts, inadequate nutrition, previous infections and many others, have long been known.

Criteria for diagnosis. The lack of specificity in occupational obstetric-gynecological pathology makes early diagnosis difficult as well as timely preventive measures. Attention is usually directed to counteracting adverse late effects in reproduction, which confirms occupational risk, but has long time been realized with other, no less significant, health consequences. In addition to obstetric- gynecological, some of the most important profpathological methods of diagnosis are important for diagnosis:

- In-depth professional profile with data on the parameters of harmful working environment factors.
- Epidemiological study for outbreaks, seeking them from more than one person in the same team.
- Dynamics of pathological abnormalities using the baseline for the profpathology exposure and elimination test, ie. reversibility of the phenomenon after disconnection, and vice versa.
- Finding into the biological media the toxic agent, metabolites or other biotransformation products.

Other relatively specific criteria are limited to:

- The condition of the ovarian-menstrual function - too early signal for the adverse impact of occupational hazards. Clinical and gynecological endocrinology methods, objectively reflect this infertility pathology in women.
- The results of objective genital examination (examination and palpation) most often correspond to the type of hormonal cycle.
- Dosage of ovarian and other hormones in biological fluids may support the results of other studies, eg. colpo-cytological. For the purpose of practice, cytodiagnosis, determining the hormone receptivity in dynamics, is more prominent.
- Abnormal pregnancy: bleeding, spontaneous abortions, gestoses, premature birth, childbirth, malformations; Complications in the labour act - labour activity, bleeding in the placental period, etc.; primary and secondary functional infertility.
- Asphyxia in the newborn, which can not be explained by the conditions in which the child was born or other factors. It is also important to take into account the lag in the physical and

mental health of the newborn, as well as in the later development of the child.

The mentioned criteria for diagnosis of the professional obstetric- gynecological pathology do not exhaust the diversity of the type and degree of the adverse events under the influence of the production factors and processes. In the complex diagnosis it is important to take into account both the gonadotropic - teratogenic, carcinogenic effect and the remote consequences.

Epidemiological and clinical data on the impact of productive factors on reproduction. The influence of environmental factors on the mother-placenta-fruit biological system is varied. It has been proven that 4-6% of the births have abnormalities (severe and minor developmental defects, metabolic disturbance), etiologically 10% are due to chromosome changes, about 20% to other genetic abnormalities and approximately 70% - as a result of the impact of the environment or in combination with genetic factors.

The effects of **chemical substances** are associated with the greater incidence of functional sterility in terms of production contact with petrol, benzene, toluene, xylene, trichlorethylene, lead, mercury, arsenic, etc. Pregnancy gestations are more pronounced and more frequent in women contacting gasoline, benzene and homologues, trichlorethylene, carbon disulfide and hydrogen sulphide, dinil and caprolactam, and others. These chemicals can also cause spontaneous abortions, premature births in the early outflow of amniotic fluid, partus precipitatus. Transplantation of lead, mercury, phosphorus, gasoline and benzene, trinitrotoluene, carbon, manganese, cadmium, carbon monoxide, pesticides leads to a higher incidence of spontaneous abortions, deaths and those born with malformations. The penetration of toxic substances into maternal milk leads to increased incidence of children up to 1 year of age, lagging behind in their physical development, reduction of the immunobiological reactivity of newborns. For some toxic substances, it is not debatable whether circulating in the body of the mother both they and their metabolites are active, especially since they are contained in breast milk, blood, urine and fecal matter, placenta, amniotic fluid , In the ovum.

Clinical manifestations when working in an **ionizing radiation** environment are too early, before positivation of blood changes, mostly functional, and in some cases morphological with irreversible damage to the follicles - sterility or spontaneous abortions, infertility, malformations. Even small doses may cause menstrual disorders, mainly oligo-menorrhea, amenorrhea, menorrhagia, metrorrhagia. These data, and especially those related to genetic consequences, necessitate the periodic updating of radiation protection standards for workers with or near sources of ionizing radiation and the population as a whole. Other factors that affect production conditions, often alongside of forced labour, muscle over-strain, increased mental-psychological load, overcooled or overheating microclimate, are **vibrations** and **noise**. The faster development of "vibration-noise disease" in women has been established, with smaller parameters and with some kind of clinical picture. Vibrations cause spontaneous abortions, gestations of pregnancy, abortive birth bleeding, premature leakage of amniotic fluid. These pathological changes, incl. and changes in the menstrual cycle, are still in the earliest stage of development of vibration-noise disease. Self-**noise** as a production factor adversely affects the female organism much earlier than on the male, especially during menstruation and pregnancy. The suggestion of some authors to bring pregnant women out of workplaces with **high air temperature and infrared radiation** is warranted because hypotension, congestive phenomena in the small pelvis, fast heartbeat are more prominent in pregnancy gestations, delayed labour activity requiring frequent surgical birth, asphyxia of the newborn during labour and in early post-natal period. Arterial hypotension at birth, with all the resulting complications, is common also in those working under **electromagnetic fields** from the radio frequency range. And since the biological effects of RF EMF is reflected in the complex effects on the CNS, VNS, neurohumoral, hematopoietic and most to germinative, functions, complications in fertility is reduced to events representing contraindications for work pregnant in an environment with EMF. Interest is a study on the work of women of reproductive age in **windowless rooms** with static microclimate and lack of natural solar radiation. Except underlined genital pathology, in 36 pregnancies 16 has been pathologicaly, mainly threatening abortions complicated was born in 9 cases, 3 children were born preterm, 1 - full-term, but immature, 1 - with hydrocephalus and spontaneous abortion has occurred In 4 pregnancies (the limited number of observed subjects only draws attention to possible risk). In the case of **lasers**, there is still no conclusive evidence of impairment of reproductive function, and only in experiment there is evidence of induced hyperestrogeny. **Physical overload and lifting of overweight** have been shown to negatively affect pregnancy, and that is why they have first found a place as a ban in labour law.

Prevention of damage to the genitals and reproductive function in manufacturing contact with

chemical factors include organizational, technical and technological activities - rational design, construction and equipment utilization, organization and technology of manufacturing processes, coformable to the specificities of the female body and the need to preserve Its reproductive function. The safe handling of toxic substances requires strict adherence to the rules of personal and public hygiene. In medical prevention is an important professionalism at selection of incoming, through strict adherence to the contra indications - i.e. the women with impaired ovarian-menstrual function are contraindicated for use with lead, mercury, cadmium, and their compounds, organic solvents, carbon disulfide, styrene. In some productions with an increased risk for reproductive function there is a statutory prohibition to work for women of fertile age - up to 35 years of age in the synthesis and polymerization of vinyl chloride; up to 40 years of age - in contact with selenium and its compounds with styrene, ethylene and ethylene oxide and others. In periodic inspection of the risk contingencies, gynecological examinations is mandatory in contact with lead and its compounds, mercury and its compounds, hydrogen sulphide, and at indications: manganese and its compounds, arsenic and its compounds, unsaturated aliphatic hydrocarbons, aromatic hydrocarbons, amino- and nitroderivatives of aromatic hydrocarbons, hormonal preparations and others.

Hygienic and technological issues related to limiting production risk from adverse physical factors are also too complex. Special requirements are provided for limiting the production risk from the effects of noise, vibrations, EMF, lasers, etc. Periodic medical examinations require attendance of obstetricians-gynecologists in work in local and general vibrations, intense production noise, sources of radio frequency electromagnetic fields, etc. Although pregnancy is a physiological condition, it is a contraindication for a number of industries, which prohibit work in a hostile work environment - noise and vibration, in conditions of EMF, ionizing radiation, lasers, overheating and overcooling microclimate and others.

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Iv. Grozdev

13 OCCUPATIONAL IMPAIRMENT OF THE ORAL CAVITY AND TEETH

Most of the occupational hazards penetrate the body through the mouth. Some of them are deposited in the oral tissues and damage them directly. Others are absorbed through the lining or are excreted by the salivary secretion. Intoxications and other occupational illnesses are often manifested with characteristic dental symptoms of proven importance for their diagnosis and causal therapy and prophylaxis.

People with toxins and aggressive chemicals are at **risk** and, to a lesser extent, individuals under the influence of certain physical, physicochemical and biological factors. Direct engagement of the lips and teeth in the work process poses a risk of their immediate harm.

Etiology and pathogenesis. They are clearer with the consequences of direct physical and chemical effects - demineralization, abrasion, usuration, etc. of the teeth; pigmentation, cauterization, irritative inflammation, etc. mucosal lesions.

General occupational diseases and poisoning affect the mouth after disruption of metabolism, trophic and reactivity of tissues. Local expression is vascular dystonia, staining and inflammation of the lining, structural changes in jaw bone and joints, etc. Individual resistance or disposition to unfavorable exogenous influences plays an important role. The effect of harmful professional factors is also aggravated by bad oral hygiene and by the presence of other non-professional pathology (the latter is deepening under the influence of professional harm).

I. DISEASES OF ORAL CAVITY OF TOXO-CHEMICAL FACTORS.

1. Acids, irritants gases and bases are chemical substances with markedly aggressive action on chewing organs. Prolonged exposure to vapors and aerosols of inorganic acids and their anhydrides increases the saliva's acidity and causes demineralization of the frontal teeth with characteristic erosion of the enamel. Affected people complain of sour taste, hyperesthesia and dullness of teeth. Later the tooth enamel becomes cloudy, becomes rough and yellow-brown. Toothed columns become brittle and susceptible to abnormal abrasion (up to 55% of people working with acids). An open occlusion is formed in the front. The incidence of acid-damaged teeth ranges from 2 to 70% depending on the duration and intensity of the exposure. Erosion teeth remain vital, but are susceptible to recurrent caries.

Direct contact of the oral mucosa with concentrated acids and bases causes painful burns, followed by low sensation, taste dullness, hypersalivation and stomatitis. Later, specific stained scarring is formed. The chronic action of the caustic factors irritates the lining and leads to cheilitis, catarrh, pigmentation, desquamation, and hyperkeratosis.

Unlike mineral, organic acids exert a cariogenic effect on all teeth and contribute to mucosal and periodontal inflammations. Typical is so-called "sugar or confectionery" caries with circular, broad-leaved teeth neck injuries and frequent complications in food industry workers.

2. Heavy metals. Oral symptoms are among the cardinal signs of increased deposition or intoxication from these noxae. Exceptionally, classic heavy lesions in the mouth can be observed today.

Mercury is accompanied by metallic taste sometimes with hypersalivation, "cold" mucosal edema with mucous tongue and cheeks, tremor of the tongue withdrawn, non-specific periodontiopathies, very rarely, and a mercury strip on the gingiva.

Saturnism is currently developing with an unpleasant "lead" breath and a sweet-metal taste, anemia of the lips and mucous membranes, an increased incidence of dystrophic periodontitis (up to 63%). Relatively rarely, discrete gray-brown pigmentation of the mucosa occurs - depending on the extent of lead exposure and impact in 10 to 16% of workers. By way of exception, the classical endogeneous lead-sulfide garland on the gingival edge (Bourton's band) is now observed.

Cadmium intoxication occurs with metallic taste, dryness in the mouth and golden-yellow coloration of the teeth neck ("cadmium ring"). Osteoporosis of the jawbone rarely develops.

3. Organic solvents. Oral changes in benzene and gasoline intoxication are more characteristic. From the developing fatty dystrophy and damage to the capillaries, the tenderness and tendency of the lining to bleed increases. Inflammatory edema and hyperemia of the gingival edge and the rest of the

mucosa appear and are followed by minor traumatic and spotting bleeding. The decreased periodontal resistance increases to 80-90% the incidence of periodontal disease, which is difficult to treat. There are no-rare, cumbersome, post-extraction haemorrhages.

The direct irritant effect of phenol, chlorinated hydrocarbons, nitro compounds of benzene, and oth. causing chronic catarrh, erosion and burns of mucous membrane with disordered taste and salivation. Intoxication with trichlorethylene affects the sensory fibers of n. trigeminus with paraesthesias, glossalgia, shaking and falling teeth. Working with tar, asphalt, resins, aniline paints and mineral oils increases the risk of developing cheilitis and mucosal hyperkeratosis as the basis for malignant degeneration.

4. Other toxic-chemical damages can be expected in professional contact with:

Arsenic and thallium. Their excretion through the salivary glands causes metallic taste, garlic breathing, xerostomia with dysphagia, stomatitis, parodontosis, and a tendency to hyperkeratosis.

Phosphorus and fluorine: breath of garlic, hypersalivation, initially osteosclerosis, and later osteoporosis of the jaws. Under modern working conditions, classical phosphorus jaw necrosis and osteomyelitis are not developed.

Pesticides: change taste and salivation, irritate the lining of pain, hyperemia, haemorrhage and keratinization, potentiate periodontal disease.

Plastics (non-polymerized ingredients - monomers, plasticizers, etc.): irritate the mucous membrane and periodontal and cause non-specific, difficult to treat cheilitis, recurrent gingivitis, parodontosis and hyperkeratosis.

Vegetable and animal powders: change the taste and color of the teeth and mucous membranes, trigger the development of allergic cheilitis and stomatitis especially in bad oral hygiene.

II. DISEASES OF THE ORAL CAVITY OF PHYSICAL FACTORS

1. Mechanical traumas (from instruments, powders, vibrations) most often lead to the development of pathological dental abrasion, traumatic periodontiopathies with frontal teeth luxations and arthropathies. Chronic microtructures on the mucous membrane cause pigmentation, erosion, and hyperkeratosis. A characteristic occupational disorder of wind instruments and glass-blowers is the so-called stomatodynia that occurs with taste disturbances, paraesthesias, spasms and lip pains in contact with the tool. Sometimes the air blowing blows over and pervades the parenchyma of the parotid glands. A pneumocellulitis develops with a distinctly changed face, unpleasant popping and chest pain, temporarily or permanently impaired working capacity.

2. Radiant energy. Ultraviolet rays damage the lips after disturbing protein, mineral and water metabolism in tissues, especially in vitamin deficiency and the help of photocatalysts. In open-air fishermen, sailors, farmers, cheilosis and cheilitis ("cheilitis solaris") are often developed. **Ionizing radiation** causes mucosal dyskeratosis, dystrophic changes in dental pulp and salivary glands with characteristic xerostomia, multiple rapid "ray caries", rarely osteo-radionecrosis of the jawbone.

3. The sharp temperature fluctuations (overheating, overcooling) of the work environment are often the cause of n. facialis and n. trigeminus neuralgia and transient rheumatoid arthritis of the maxillary joints with an effect on working capacity at workers in furnaces, at overflow, refrigeration, etc. Combined heat and dust effects damage the lining and facilitate the formation of tartar and plaque - a prerequisite for an increased incidence of gingivitis and marginal parodonthopathies.

4. Barometric factors (abrupt decrease or increase in atmospheric pressure - in aviators, divers, caisson workers) are reflected in the mouth with sudden severe pain from carious or undercured teeth (aerodenthalgia), tendency to bleed, dryness and inflammation of the lining and the teeth support tissues.

III. DISEASES OF ORAL CAVITY OF BIOLOGICAL FACTORS

Professional **zoonoses and phytoparasitic diseases** are currently significantly less frequent among agricultural, veterinary, medical and other workers. Reflection in the mouth is caused by aphtae epizootical, actinomycosis, aspergillosis, oral candidiasis, anthrax, tularemia and other infectious diseases.

Diagnosis is based on evidence of a causal relationship between the pathological process and adverse work environment factors: specific occupational stigma and increased incidence of one-size-fits-affected disorder influenced by discontinuation or renewal of exposure; the effect of applied detoxifying therapy is monitored; the presence of chemical damages and their metabolites in the biological environment is investigated.

Prevention and treatment. With the preliminary dental examinations of workers with agents intervening in the oral cavity, professional selection is made on dental indications. Periodic reviews aim at early detection and early rehabilitation of occupational and banal diseases. To enhance the resistance of local tissues to intense professional effects, protective coatings and tooth fluoridation, system brush and shower massage of the gums, maintaining per-

fect oral hygiene, timely sanitation and removal of local irritants and injuries are applied. Healthy nutrition rich in vitamins, mineral salts and protein foods, improvement of the labour process with possible exclusion of the mouth from direct participation in working manipulations - in case of impossibility - use of suitable mouthpieces, safety splints, screens and other means of local protection.

Treatment - Physical procedures, rinse with light disinfectants, dyeing, high doses of vitamins (B, C, K, PP), novokain blocks, calcium preparations are applied to the tissues, stimulating their trophic and regeneration. General and local detoxification therapy. Specific requirements for workers: in overheating microclimate - non-metallic fillings; with cariogenic substances - corona photopolymers instead of amalgam fillings; of blowers (musicians and glass makers) - fixed prosthetic structures.

Temporary discontinuation of exposure (or redevelopment) has a beneficial effect on treatment.

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K. Kotseva

14 OCCUPATIONAL DISABILITIES OF THE CARDIOVASCULAR SYSTEM

Cardiovascular diseases are the most common cause of death in the developed world. In Bulgaria, this indicator has markedly unfavorable dynamics, and according to the WHO, it ranks first among the ten countries in the world where cardiovascular disease mortality is steadily rising. It is known that the major risk factors for coronary artery disease (IHD), arterial hypertension, smoking, hypercholesterolemia and diabetes can only account for about 50% of the disease. The rest depend on the so-called secondary factors for which data are contradictory. Among them, a particular place is attributed to the negative impact of some chemical, physical and manufacturing deficiencies.

The cardiotoxic effect factors and cardiovascular lesions are shown in **Table 1**.

Chemical Factors

Lead. The results of many experimental and clinical studies have shown that acute exposure to high concentrations of lead may cause rise in blood pressure and cardiac rhythm disturbances. Chronic effects increase the risk of developing arterial hypertension and increase mortality from ischemic heart disease and cerebrovascular disease. An increased incidence of non-specific electrocardiographic changes - rhythm, conduction and repolarisation disorders, as well as cases of cardiomyopathy in professionally exposed workers. The following possible mechanisms of influence of lead on the cardiovascular system are discussed: 1. Renal impairment; 2. Activation of the renin-angiotensin-aldosterone system; 3. Disturbance in the exchange of catecholamines; 4. Vasospastic effect and increase of peripheral vascular resistance; 5. Direct cardiotoxic effect; 6. Dislipoproteinemia with accelerated development of atherosclerosis. A positive correlation between the level of lead in the blood and the blood pressure values was found.

Cadmium. Occupational exposure to cadmium is discussed as a risk factor for the development of arterial hypertension. Experimental studies have identified renal tubular lesions as well as sclerosis and generalized thickening of the walls of arterioles and small and middle arteries of the kidney. In case of cadmium intoxications, the catecholamines in the blood and urinary excretion are elevated. A positive correlation was found between the level of cadmium in serum and systolic and diastolic blood pressure, as well as the severity of arterial hypertension.

Cobalt. The term "cobalt cardiomyopathy" was introduced in 1965-66, when it was observed in people who consumed beer with a cobalt sulfate stabilizer. The cobalt disturbs the oxygen consumption and the mechanisms of oxidative phosphorylation in the myocardium. Cases of cardiomyopathy and increased mortality from ischemic heart disease and workers exposed to cobalt have been reported. In patients with cobalt cardiomyopathy, increased cobalt content was found in the myocardium.

Mercury. In subjects with mercury intoxication, there are relatively frequent electrocardiographic changes - arrhythmias, Q-T interval prolongation, S-T segment depression and T-wave negative. It is believed that mercury may play a role as a risk factor for arterial hypertension and atherosclerosis. Pathogenetic mechanisms discuss the nephrotoxic action of mercury, a direct toxic effect on the vessels and the heart, and a lipid metabolism disorder.

Arsen. Acute arsenic exposure may cause repolarisation ECG abnormalities and severe ventricular arrhythmias, most likely due to direct cardiotoxic effects. Cardiomyopathy with heart failure was observed in more acute intoxication. An "exposure-response" relationship between arsenic trioxide exposure and mortality from ischemic heart disease has been found, with relativistic risk increasing with increasing duration of exposure.

Antimon. In case of acute impact of antimony, various rhythmic and conduction disorders as well as changes in repolarisation are recorded. There have been reports of sudden death in workers exposed to antimony trisulphide. The cardiotoxic effect of antimony has been demonstrated in experimental studies. Antimony production increases the incidence of electrocardiographic changes in the absence of cardiomyopathy or ischemic heart disease.

Beryllium. A positive correlation was found between beryllium exposure and IHD (relatively few

Tab. 1. Production factors with possible Impact on the cardiovascular system

Production factors	Effects on the cardiovascular system		Mechanism of impact
	Acute	Chronic	
1	2	3	4
Lead	Hypertension Arrhythmias	Hypertension Arteriosclerosis? Increases mortality from IHD and BVD. Cardiomyopathy? ECG changed	Nephropathy? Activation of the renin-angiotensin-aldosterone system. Disturbance in the exchange of catecholamines. Generalized angiospasm; Increased peripheral vascular resistance. Direct cardiotoxic effect? Dyslipidemia?
Cadmium	–	Hypertension?	Nephropathy? Activation of the renin-angiotensin-aldosterone system. Disturbance in the exchange of catecholamines? Increased sympatho-adrenal system activity. Increased peripheral vascular resistance. Increased sodium retention in the body?
Cobalt	–	Dilatational cardiomyopathy	Oxidative phosphorylation disorder. Disturbance in the exchange of catecholamines?
Mercury	ECG changed	Hypertension? Atherosclerosis? ECG changed	Nephropathy. Disturbance of oxidative phosphorylation. Dyslipidemia.
Arsen	ECG changed	Increases mortality from cardiovascular disease?	Disturbance of oxidative phosphorylation.
Antimon	ECG changed - Arrhythmias - Conductive disturbances - S-T changes	ECG changed	Disturbance of oxidative phosphorylation.
Yellow phosphorus	Arrhythmias Acute CHD	–	Direct toxic effect on the myocardium
Nitrates	Angina pectoris Myocardial infarction Sudden death	–	"Rebound" vasospasm when the exposure-is discontinue "Monday morning attacks". Disturbance in the exchange of catecholamines.
Carbon monoxide	Angina pectoris Myocardial infarction Arrhythmias. Sudden death	Atherosclerosis	Myocardial hypoxia due to formation of carboxyhemoglobin. Direct toxic effects on vessels and myocardium. Disturbance in lipid metabolism. Increase of aggregation of the platelets.
Hydrocarbons A. Chloroderivatives (methylene chloride, chloroform, carbon tetrachloride). 1.1.1. Trichloroethane, trichlorethylenes, etc.	ECG changed - Arrhythmias - Conductive disturbances Sudden death	Arrhythmias Cardiomyopathy	Myocardial sensitization to catecholamines. Depressive effect on contractility and conductivity. Disturbance of oxidative phosphorylation. Methylene chloride is metabolised to CO.
B. Fluorinated derivatives (Freon 11, 12, 113, 114)	Arrhythmias Sudden death	–	Myocardial sensitization to catecholamines. Direct toxic effect on the myocardium.
Vinyl chloride	–	Raynaud's Syndrome. Increases mortality from IHD? Hypertension?	Disturbance of peripheral microcirculation. Direct toxic effects on vessels and myocardium.

Carbon	-	Atherosclerosis Increases mortality from IBS. Hypertension?	Dyslipidemia, suppression of the function of the thyroid gland, reduction of fibrinolytic activity. Direct vasotoxic effect. Disturbance in the exchange of catecholamines.
Other solvents (Ethanol, phenol, toluene, benzene)	Arrhythmias Myocardial infarction	Dilated cardiomyopathy Atherosclerosis	Direct toxic effect on the myocardium. Myocardial sensitization to catecholamines. Dyslipidemia
Pesticides			
A. Chlororganic (DDT, lindane, aldrin, dieldrin, toxofen, epichlorhydrin and others.)	Arrhythmias	Atherosclerosis? ECG changed Hypertension Atherosclerosis?	Dyslipidemia. Direct toxic effect on myocardium. Increased sensitivity of myocardium to catecholamines.
B. Phospho-organics (parathion, intrathyon, chlorophos, zolone, etc.)	ECG changed - arrhythmias Acute HVD	Atherosclerosis? Arrhythmias	Inhibition of cholinesterase by acetylcholine accumulation Direct toxic effect on the myocardium. Electrolyte disturbances
C. Mercury Organic	Arrhythmias	–	Direct toxic effect on the myocardium.
D. Carbamates	–	Atherosclerosis?	Dyslipidemia
E. Herbicides (paraquat, TDDD, dioxin, etc.)	Acvute HVD	Atherosclerosis	Disturbance of oxidative phosphorylation. Dyslipidemia.
Noise (over 85 dB)	Hypertension	Hypertension Atherosclerosis?	The type of intermittent stress with activation of the hypothalamic-pituitary axis - adrenal cortex and the sympathic-adrenal system with enhanced release of catecholamines and glucocorticoids; Increased peripheral vascular resistance. Dyslipidemia.
Vibration	–	Hypertension Atherosclerosis?	Damage of nerve endings, perivascular connective tissue and microvasculature at the periphery. Vegetative and vascular dysfunction.
High temperatures	Angina pectoris, myocardial infarction, sudden death (with coronary-sclerosis).	Atherosclerosis?	Increase the oxygen needs of the myocardium. Disturbance of water-electrolyte exchange. Increased blood viscosity.
Low temperatures	Angina pectoris, myocardial infarction, sudden death (with coronary-sclerosis).	–	Increase the oxygen needs of the myocardium. Peripheral vasoconstriction. Coronarospasm. Activate sympatic adrenal system.
Non-ionizing radiation (radio waves)	NCD, ECG changed	NCD, hypertension? ECG changed	Differential dysfunction. Impaired neurovegetative regulation; vegetative and vascular dysfunction. Direct toxic effects on vessels and myocardium.

studies). In a retrospective cohort study of US workers, an increased mortality from IHD was also found due to exposure to beryllium.

Yellow phosphorus. Acute yellow phosphorus intoxication causes disturbances in the repolarisation phase and rapid progression of heart failure due to direct myocardial toxicity. There are no epidemiological studies on professionally exposed workers.

Carbon monoxide. The acute impact of carbon monoxide can cause myocardial infarction and sudden death as well as the onset or potentiation of angina pectoris - especially in subjects with pre-existing coronary artery disease. In experimental studies, chronic exposure to carbon monoxide has been shown to accelerate the development of atherosclerosis due to the combined effect of hypoxia, increased cholesterol deposition in arterial walls, and increased platelet aggregation.

It is known that smoking is a major risk factor for IBS and that the carbon monoxide contained in cigarette smoke is considered to be one of the major etiological factors. In patients with chronic exposure to high concentrations of carbon monoxide (with carboxyhemoglobin above 30%), cardiomyopathy with congestive heart failure has been observed. The results of some epidemiological studies show an increased incidence of angina pectoris, myocardial infarction, ischemic changes, and ECG rhythm disturbances, as well as increased IHD mortality, which decreased after discontinuation of exposure.

Carbon disulfide. Chronic exposure to abnormal serum carbon disulfide concentrations is considered as a risk factor for the development of atherosclerosis and IHD. The mechanism of action of serum is explained by a disorder of lipid metabolism and the development of dyslipoproteinemia. Other possible causes for accelerating atherosclerosis include decreased serum fibrinolytic activity, direct toxic effects on vessels, and the development of hypothyroidism and arterial hypertension. Studies in recent years have shown that the increased risk of IHD is reversible and reduces with decreasing exposure / discontinuation of exposure as well as strict control of other cardiovascular risk factors.

Organic nitrates. Many cases of angina pectoris, myocardial infarction, arrhythmias and sudden death are reported in young workers exposed to organic nitrates (nitroglycerin and ethyleneglycoldinitrate). Heart incidents are known as "monday morning attacks" because they occur 48-72 hours after discontinuation of exposure. Cardiac attacks are thought to be due to "rebound" vasospasm (no coronary atherosclerosis develops) caused by the temporary cessation of exposure at the end of the working week.

Other hydrocarbons (organic solvents). Their influence on the cardiovascular system is primarily associated with the possibility of causing arrhythmia and sudden death in the presence of high concentrations. The first messages are sudden deaths in people who are breathing aerosols with a narcotic action ("sudden sniffing death"). In young adults who inhaled adhesives containing organic solvents, severe ventricular arrhythmias have been observed. Sudden death and ventricular arrhythmias are also observed in workers exposed to 1.1.1 trichloroethane, trichlorethylene, chloroform, carbon tetrachloride, fluorohydrocarbons, ethanol, toluene, benzene and the like. Chronic effects of toluene and trichlorethylene have reported cases of dilated cardiomyopathy. The pathogenesis of cardiac lesions indicates: 1. Sensitization of myocardium to catecholamintitis, which causes arrhythmias and sudden death; 2. Suppression of sinus node activity and atrioventricular conduction, causing sinus bradycardia or arrest, and atrioventricular block; 3. Direct cardiotoxic effect. Increased incidence of arterial hypertension and increased morbidity and mortality from IHD have also been reported. Epidemiological studies in workers exposed to low concentrations are insufficient.

Pesticides. In case of acute intoxication with some pesticides, there were manifestations of increased myocardial excitement - occurrence of various rhythmic disorders.

The mechanism of action of **phosphoorganic pesticides** is associated with inhibition of acetylcholinesterase, with tachycardia and hypertension, and bradycardia and hypotension later in the initial phase of acute poisoning. Various rhythm and conduction disorders such as ventricular extrasystole, ventricular tachycardia, fibrillation and asystole, as well as non-specific changes in S-T segment and T-waves have been reported.

Some **organochlorine pesticides** have a direct toxic effect on the myocardium. Occupationally exposed workers are more likely to experience atherosclerosis of coronary and cerebral arteries - there is evidence that chronic effects of chlororganic pesticides and carbamates may cause lipid metabolism disturbances.

An "exposure-response" relationship has been found between the professional impact of phospho-organic pesticides and the frequency of "ischemic" changes in the ECG.

Polycyclic aromatic hydrocarbons (PAH). Polycyclic aromatic hydrocarbons are thought to have not only a carcinogenic effect but also increase the risk of IHD - this is in line with the monoclonal theory (EP Benditt et al.) according to which atherosclerotic plaques can be formed by proliferation of one cell and seen as benign tumors.

Workers exposed to PAH found an increased incidence and mortality from IHD and cerebrovascular disease.

Physical Factors

Noise. Production noise is seen as a risk factor for the development of arterial hypertension. A positive correlation was found between the intensity and duration of the noise effect and the incidence of arterial hypertension, serum cholesterol levels, and adrenaline and noradrenaline content in the blood and urine. The impact of noise is explained by the activation of the hypothalamic-pituitary-suprarenal gland and sympatho-adrenal system, generalized vasoconstriction. Experimentally, atheromatous changes were observed in the aorta and middle-sized arteries as well as morphological changes in the myocardium.

Vibration. Production vibrations damage nerve endings, perivascular connective tissue and microvasculature at the extreme periphery, cause vegetative and vascular dysfunction. Like the noise, their effects as a chronic intermittent stressor with activation of the hypothalamic-pituitary-adrenal cortex functional axis and the sympathic-adrenal system with enhanced catecholamine and glucocorticoid release and increased peripheral vascular resistance are possible. An increased incidence of arterial hypertension and IHD has been reported, as well as lipid disorders in professionally exposed workers.

High and low temperatures. In workers with coronary sclerosis, the acute effects of high and low temperatures (where myocardial oxygen needs suddenly increase) can cause ischemic attacks, such as angina pectoris attacks, myocardial infarction, or sudden death. Acute exposure to low temperatures may also cause peripheral vasoconstriction, coronarospasm, and sympatho-adrenal system activation. Working under high temperature conditions also leads to a disruption of water electrolyte equilibrium and increased blood viscosity. It is considered that overheating microclimate in long-term work can be considered as a risk factor for the development of atherosclerosis.

Ionizing radiation. Experimental studies have shown that ionizing radiation has atherogenic activity, as well as in patients undergoing radiotherapy, there is a premature development of atherosclerosis. However, there are insufficient epidemiological studies on IHD incidence and mortality in professionally exposed workers.

Non-ionizing radiation. Long-term exposure to electromagnetic waves may cause development of neuro-circulatory dystonia (NCD) of hypo-or hypertensive type. Functional changes may progress to sustained arterial hypertension.

Knowing the harmful effects of chemical and physical factors on the cardiovascular system is of great importance for the prevention of cardiovascular diseases. Their frequency can be greatly reduced in strict compliance with the limit concentrations and rates of chemical and physical hazards, as well as limiting the entry of workers with an increased cardiovascular risk - chronic cardiovascular diseases, diabetes mellitus, obesity, hereditary and oth. productions where there are factors damaging the cardiovascular system.

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15 PHYSIATRICS AND REHABILITATION OF PREFESSIONAL DISEASES AND INTOXICATIONS

15.1. OCCUPATIONAL INTOXICATIONS

15.1.1. REHABILITATION OF PROFESSIONAL METAL POISONINGS **

In professional metal poisonings sulfide mineral waters are specifically detoxicating agent, since they associate the metal with their sulfide component and eliminate it from the organism. Drinking treatment is with mineral waters with a sulphide content of 10-15 mg/l and fluorine at a dose of up to 20 ml/kg per day divided into 3 - doses. Sulfid mineral waters also make inhalations, baths and enemas. The baths have a water temperature of 36-37° C, duration of 10-15 minutes in a healing course 15 procedures applied each other day or with 2 rest days weekly. The sulphide mineral waters in Sapareva Banya are particularly suitable.

Both iodine and iodine-bromine natural and artificial water are used. The methodology of their internal and external application is as for sulphide mineral waters. Iodine-bromine artificial water is prepared as follows: for 100 batches 2.540 g of potassium bromide and 1.500 g of sodium iodide are dissolved in 10 l of water[1]. Place 100 cm^3 of the prepared solution in a bath of 200 liters of water, if necessary tempered.

Contraindications for balneo therapy treatment: acute infections; severe- and moderate - severe chronic intoxications; sensitivity to sulfur, iodine, bromine; cardiac decompensation; hypertension II degree; infarct; advanced atherosclerosis and the like. The release of metals from depots is also aided by thermal procedures in the liver and kidney. It is achieved by exogenous heat with paraffin, curative mud, infrared, sollux, as well as through endogenous heat with diathermy, VHF and microwave therapy. With these agents, active hyperaemia is achieved, metabolism increases and stimulates the detoxic function of the liver and kidneys.

Galvanic collar of Sterbach with calcium and bromine, electrophoresis with coffein and bromine, juniper baths and others are used to restore the functional fitness of the autonomic nervous system.

Kinesitherapy is a good result in patients with asthenovegetative and polyneurial syndrome.

Construction of kinesitherapeutic complex in patients with polineurial syndrome in chronic metal poisoning is dependent on the clinical picture that is too diverse by the prevailing symptoms and the effect on one or other peripheral nerves. In the prevalence of motor impairment, kinesitherapy was built on manual muscle testing. Through the general toning of the patient, the aim is to lower the threshold of excitability. It is necessary to choose optimal starting positions and to apply, depending on the damage, the method of the proceptive neuro-muscular facilitation with reflective influence of the injured structures, analytical exercises for the limbs together with relaxing techniques - all based on breathing and fortifier exercises.

A good effect is underwater gymnastics with a water temperature of 36-37 ° C.

In patients with astheno-vegetative syndrome kinesitherapeutic complex includes exercises combined isotonic and isometric mode extremities, abdomen and gluteal muscles, free active with relaxing effect exercises with upper and lower extremities, exercise for balance and coordination, to improve the sensory, breathing and general roborant exercises. We train the patient in the ways of general relaxation. The duration of the procedure is 30 to 60 minutes.

The best results are the complex treatment, which, depending on the leading syndrome, is:

When prevalence of polyneuritic phenomena occurs with paresis or paralysis of peripheral nerves, nerve cell electrophoresis, electrostimulation, kinesitherapy, underwater gymnastics, massage are most often used.

In astheno-vegetative syndrome is assigned balneotherapy, galvanic collar with calcium and bromine, physical therapy, massage collar, hygienic-dietary regime.

In predominance of gastrointestinal syndrome: hygienic - diet regime, drinking treatment, balneotherapy, thermal procedures.

Hepatic disorders recommended drinking mineral water from Hisaria, Gorna Banya, Slivenski mineralni bani, spa baths in the same resorts, heat treat-

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ments in the liver area and hygienic-dietetic regime.

When the prevalence of anemic syndrome, general UV irradiations 1/4 biodose daily - 20 days, drinking administration of iron-containing mineral water (Breznik) and hygienic-dietetic regime.

15.1.2. PHYSIOTHERAPY AND KINESITHERAPY IN ORGANIC SOLVENTS INTOXICATION

PHYSIOTHERAPY*

Physiotherapy is applied in chronic intoxications and possible detoxification, general body strengthening and neurasthenia syndrome affecting, which are differently manifested in individual intoxications.

Drinking mineral water treatment at 38-40 ° C, taken 3-4 times 200-250 ml, 45 min before meals for 4-6 weeks, was shown. The following mineral waters, poorly mineralized (Gorna Banya, Bankya, Hissar, Knyajevo, Pancherevo etc.), hydro-carbonates (Mramor, Ilientsi, Chepintsi, Birimirtsi, Kourilo), carbon-acid (St. Karadjovo, Mihalkovo), sulfide (Separeva banya, Kyustendil), salted (Mirkovo, Staro- and Novo Oryahovo, Shkorpilovtsi, Ovcha mogila). Balneological treatment is applied in Velingrad, Bankya, Narechen, Varshets; Mountain climates (Pestera, Kurtovo, Yundola, Borovets, Pamporovo), sea treatment and Black sea resorts. A good effect is treatment with medicinal baths with juniper, tripinate, brobalyl, rosemary, 36-37 ° C, 10-15 min, 12-15 procedures. It is a good response with: common iontophoresis with calcium and bromine in Vermel (10-20 mA, 15-20 min, 10-12 procedures) or galvanic collar on Shcherbak calcium and bromine (6-16 mA, 6-16 min, 10-12 procedures) brush body massage (15-20 min, 10-12 procedures) psychosensory relaxation of impact of speech, music, colors and flavors (45-60 min, 8-12 sessions) and acupressure in the following points: 3CM 14, 16, 19, 20, CPM 6, St 36, DP6, MD4, 11, 3E5, P6, 1-2 min at a point, 10-15 procedures.

In the advanced stage of chronic poisoning with carbon disulfide and the occurrence of lesions of the type of vegetative-sensory polyneuritis, physiotherapy is applied for this kind of lesions.

Similarly to the above, in the presence of chronic bronchitis in chronic phenolic intoxications, physiotherapy relevant for this condition is administered.

KINESITHERAPY **

Kinesetherapy is used as a prophylactic agent when working with organic solvents and for the treatment of chronic poisoning. The number of kinesitherapeutic procedures is unlimited. The patient is trained in the methods of general relaxation and in the so-called autoselect method. It shall apply from different starting positions (occipital, lateral leg in semireclining and sitting position), begins and ends with abdominal - diaphragmatic breathing and exercises to strengthen the respiratory muscles. Orthostatic and clinostatic responses, equilibrium and coordination are trained. In sensory disturbances, exercises are given for superficial and deep senses.

In patients with chronic intoxications, a good effect on neurasthenic syndrome and vegetative dysfunctions is underwater gymnastics with a water temperature of about 36 ° C, as well as underwater jet massage - 1.5 at (0.15 MPa). Analytical exercises for the limbs in isotonic and isometric mode are based on breathing and tonic exercises. Field terrain treatment, nearby tourism, sport discipline parts apply.

15.1.3. PHYSIOTHERAPY AND KINESITHERAPY IN POLYMERS INTOXICATION

PHYSIOTHERAPY *

Physiotherapy is applied at the chronic stage and has the following tasks: possible detoxification, increase of the general resistance of the organism, influence of the different syndromes (neurasthenic, astheno-vegetative).

Methods of physiotherapy are described in the section "Physiotherapy for poisoning with organic solvents".

With vinyl chloride intoxication, besides the above tasks, physiotherapy aims to also influence the available osteoporosis and Raynaud's syndrome. They are efficient:

Balneotherapy in Haskovo mineral bani, Momin prohod, Pavel banya, Kyustendil, Sapareva banya. Specific effects on arterial spasm, and osteoporosis is the use of low-frequency magnetic field (12,800 A/m, 1 Hz, 0,2 s, 1-20 procedures).

Chronic poisoning with ethylene available angiodistoniya affected by carbon-acid, pearl, oxygen bats or medicinal baths with juniper, brobalil, tripinat (36-37 ° C, 10-15 min, 12-15 procedures). The spa treatment with radon waters is effective in Momin prohod, Strelcha, Pavel banya, Velingrad and others.

KINESITHERAPY **

The kinetisi therapeutic complex in chronic polymer intoxications is dependent on the predominant syndrome.

With Raynaud's syndrome with kinesitherapy, we aim to restore and maintain the blood supply in affected areas. This is achieved with simple in coordination attitude gymnastic exercises for developing type all parts of the body, carried freely and rhythmically full amplitude and slow to medium pace, relax-

ing and breathing exercises. Games, swimming and nearby tourism in proper dosage also have a place in the motoring program. Local therapeutic impact is achieved through special exercises for the affected limbs. These are active, rhythmic exercises of their distal parts running freely or against moderate resistance and rhythmic short, isometric exercises. The best results in this respect are obtained by proper rotation of exercises against adequately and relaxation techniques, such as active swinging and shaking of the limbs, the withdrawal of the limb along the longitudinal axis - method Schaarschuch, surface soothing massage, gently stroking, squeezing and rocking). Underwater gymnastics with a water temperature of 36 ° C ± 0.5 ° and a submersible jet massage of 1.5 (0.15 Mpa) also improve haemodynamics.

In the presence of osteoporosis, the gravitational burden and the tension of the affected bones are carefully measured. In osteoporosis of the spine is the most suitable is fixing corset of Hohmann, who has a metal frame supporting pelvic bones and armpits. Wearing warm woolen "belts" is more elemental and widespread. For limbs use dynamic frames that stabilize the bones and allow movements in the adjacent joints. Keep in mind that orthoses weaken muscles and are always applied with kinesitherapy. The most suitable for this purpose are analytical exercises in isotonic mode of 7-8 s pause and contraction in a ratio of 1: 1. They are performed repeatedly during the day 30-40 times for each muscle group. Analytical exercises in isotonic mode are from relieved baseline. Underwater gymnastics has a water temperature of 30 ° C. No thermal procedures are given. The basic kinesitherapeutic principles of gradual, systemic, distraction, etc. are respected. Occupational therapy (entertaining, functional, professional) is also applied.

In case of sclerodermic skin changes and thyroid hypofunction, a sauna is administered 1-2 times a week in combination with hydrotherapeutic heat (paraffin or mud application). It can be applied once a week and cryotherapy for both reflex action and immediate action of cold factor on the tissue trophy. Mandatory manual massage and kinesitherapy including isothermal and isotonic analytical exercises, breathing and general tonic exercises are mandatory. Underwater gymnastics and underwater jet massage have a water temperature of around 36 ° C. The kinesitherapeutic program also includes field treatment, nearby hiking, swimming.

15.1.4. PHYSIOTHERAPY AND KINESITHERAPY IN PESTICIDES INTOXICATION

PHYSIOTHERAPY IN PHOSPHORGANIC COMPOUNDS AND CARBAMATES *

In acute poisoning, physiotherapy is pathogenic by the application of cholinolytic agents - atropine injections/prepared atropini sulfurici 0,0025 g and aqua destilata 20,0 ml of which for one inhalation, 2 ml, 10 min, 10-12 procedures are taken.

In chronic poisoning, physiotherapy aims to detoxify and influence the presence of vegetative dystonia with predominance of parasympathetic, toxic cerebrovascular and chronic bronchitis. Balneotherapy with iodromatic waters (Slanotrun, Shabla, Shkorpilovtsi, Staro and Novo Oryahovo), radon water (Narechen, Momin prohod, Strelcha, Pavel banya, Velingrad) is used. Effective is also bats with juniper, tripinate, brobabyl, rosemary (36-37 ° C, 10-15 min, 10-12 procedures), total brush massage (15-20 min, 10-12 procedures), acupressure at points: St 36, MD4, 11, P6, PM10, POM6, 3CM14, 20, 1-2 min at a point, 10-15 procedures.

For treatment of existing bronchitis, see "Physiotherapy of Chronic dust Bronchitis".

PHYSIOTHERAPY IN CHLORORGANIC INSECTICIDES *

Physiotherapy is applied at the chronic stage for the purpose of detoxification and increase of the general resistance of the organism. Drinking mineral water (250 ml, 38-40 ° C, 3-4 times 45 minutes before meals) and baths with the following mineral waters: low mineralized (Bankya, Gorna banya, Knyazhevo, Pancharevo, Hissar, Banya - Pazardjik), chlorides (Mirovo, Ovcha mogila, Staro and Novo Oryahovo, Shkorpilovtsi), fluors (Velingrad, Strelcha, Blagoevgrad, Pavel banya). Water treatment with juniper, salted, salimar, tripinate baths (36-37 ° C, 10-15 min, 12-15 procedures) is recommended. Effective is general ionogalvanisation of calcium by Vermel (10-20 mA, 15-20 min, 10-12 procedures), total brush massage (15-20 min, 10-12 procedures), acupressure at points: D4, 11, P6, 3E5 , 3CM14, 16,20, PCM6, St36, DP6, 1-2 min at a point, 10-12 procedures.

KINESITHERAPY FOR PESTICIDES **

Chronic poisoning with pesticides has been successfully applied: autogenous training from various starting positions, breathing gymnastics, including exercises for general influence, breathing rate control, improved mobility in the diaphragm strengthening; means for training the orthostatic reactions; coordination exercises; means for regulating blood pressure; isometric exercises for pelvic floor training, as well as nearby tourism, occupational therapy, etc.

The basic principles of kinesitherapy are respected and, after using the mentioned means, these are applied without limitation of the procedures in the treatment course.

15.1.5. PHYSIOTHERAPY AND KINESITHERAPY IN IRRITANT GASES INTOXICATION

PHYSIOTHERAPY*

In acute poisoning, physiotherapy aims to overcome the inflammatory process and added upper respiratory tract infections. Heat-moisted inhalations with 2-3% sodium bicarbonate (2 ml, 10 min), inhaled etheric oils, antibiotics, sulfonamides, expectorants are made. Mineral water inhalations are effective: (Mihalkovo, St. Karadzhovo), alkaline (Hissar, Bankya, Knyazhevo, Velingrad), low mineralized (Gorna Banya, Pancharevo, Banya - Panagyursko, Banya-Karlovo), sulphide (Sapareva banya, Kyustendil). Indicated are: breathing with oxygen, abduction means (chest synapses, suction cups).

Chronic poisoning is recommended for mountain climates in warm and dry weather (Pamporovo, Yundola, Peshtera, Kurtovo) as well as balneotherapy in Velingrad, Sandanski, Momin prohod.

KINESITHERAPY **

In the case of those working in a harmful environment, it is necessary to build a kinesitherapeutic program for the prevention of poisoning, including: increasing the mental tone and the general training with kinesitherapeutics (general tonic exercises, sport discipline items, puli therapy etc.) targeted respiratory gymnastics for rhythmic breathing , breathing with extended expiratory phase, for active movement of the diaphragm and strengthening of the respiratory muscles; exercises to improve peripheral haemodynamics; training in local and general relaxation. The sports disciplines most suitable for the threatened professions are: hiking, swimming, skiing.

In case of pronounced chronic poisoning, we strive to normalize the functions of the cardiovascular, respiratory, nervous systems. Depending on the clinical picture, methods of blocking the diaphragm, the thoracal cage and the spine are used, using functional respiratory reserves; exercises to improve peripheral haemodynamics; autogenous training; field treatment and more.

15.2. OCCUPATIONAL DISEASES BY PHYSICAL FACTORS

15.2.1. PHYSIOTHERAPY AND KINESITHERAPY IN COLD ANGIONEUROPATHY

PHYSIOTHERAPY *

Physiotherapy aims at influencing the limb frostbite, vascular spasm and obliterating thrombangitis. The most efficient and specific action is the low-sensitivity magnetic field applied topically (12,800 A / m, 1 Hz, 0,2 s, 15-20 min, 15-20 procedures). Ultrasound is also effective applied paravertebral of the upper limbs at the C3-T4 level, and to the lower limbs at the level L1-S2, 0.2-0.4 W / cm2, 4-5 minutes for each zone. Interference currents (constant frequency 100 Hz, 15 min, 10-12 procedures), sympatholytic-acting diadynamic currents applied in the areas described above under ultrasound (100 Hz, 4-5 min, 10-15 mA, 10 Procedures). Microwaves (0.36 W / cm2, 10-12 min, 10-12 procedures), ultra-short waves (oligothermal dosage, 10-15 min, 10-12 procedures) are shown. Etiopathogenetically is the effect of externally applied sulphide mineral waters (Sapareva banya, Kyustendil) as well as the sulphate-sodium and fluorine waters of Haskovo mineralni bani. It also recomended the iodinebromine mineral waters (Slanotrun, Shabla, Novo and Staro Oryahovo, Shkorpilovtsi). Efficient acidulae, oxygen, pearl baths, as well as four-chamber bathtubs with tripinate, brobalil, salimar (37-38 ° C, 10-15 min, 12-15 procedures).

At the chronic stage, mild muddy applications (38-40 ° C, 20-30 min, 10-12 procedures) are carefully applied in the stabilization of the condition, taking into account the individual tolerability.

KINESITHERAPY **

Kinesitherapeutic agents have a beneficial effect on the functions of the autonomic nervous system, contributing to the normalization of vascular and muscular tone, improving the metabolic and trophic processes in the limbs. The kinesitherapeutic program for the prevention of cold diseases includes: isotonic and isometric exercises for the limbs, relaxing exercises, breathing exercises, and exercises from different starting positions at different amplitudes at the beginning of the workday (so-called inaugural gymnastics). In the middle of the working day, analytical exercises are performed in isotonic mode at a ratio of 1: 1 with free, active, relaxing effect, breathing and general tonic exercises in baseline, different from the working posture. At the end of the day work massages (self-massage), underwater gymnastics with elements of swim sport or swimming, underwater massage, etc.

At angio-neuropathy is used exercises to support peripheral blood circulation, antiedematous exercises and sensory exercises. Analytical exercise in isotonic mode is up to 90 ° armpits and in a 1: 1 or 3: 1 ratio with free active and relaxing effect exercises. In more severe cases, there is a segmented-reflective effect on vegetovasal disorders (the method is described in vegetative polyneuropathy). The patients are trained in autogeneous workout with the so-called "Autoselect method".

15.2.2. PHYSIOTHERAPY AND KINESITHERAPY IN THERMAL SHOCK, THERMAL CRAMP, THERMAL EXHAUSTION

PHYSIOTHERAPY * In heat stroke, physiotherapy is applied as a first aid. Place cold compresses (ice) of the head, pouring, shower or bath with cold water, wrapping a sheet wetted with cold water, adopting a cold liquids, acupressure points PSM24, ZSM20, 28, P9 methodology finger - needle 30 S to 1 min at point.

In case of chronic overheating it is necessary to provide bathrooms for cold water procedures (baths, showers, swimming pools). It is not only a cooling effect but also a hardening effect. A good effect is the sharp temperature changes of the water jet at the Scottish shower (hot jet 40-43 ° C, 20-30 s, followed by cold jet 15-20 ° C, 5-10 s, each repeat 5-10 times, 15 -20 procedures.

KINESITHERAPY **

For workers in industries with overheating microclimate is prepared kinesitherapeutic program aimed at improving the adaptation capabilities of the organism. The program includes exercises and techniques with a strong influence on the general and local blood flow - and lymphatic circulation. used: rhythmical exercises in isotonic and isometric regimen from different starting points: breathing exercises (chest, diaphragmatic, yogic breathing). Rhythmic muscle contractions and movements of the joints against adequate resistance from proximal to distal extremities act as muscle pump and support the flow of venous blood to the heart. The program includes underwater gymnastics with a water temperature of 27 °, underwater jet massage with a water temperature of 35 ° C, cryotherapy, autogenic training from different starting positions, swimming, cycling, hiking, etc.

15.2.3. PHYSIOTHERAPY AND KINESITHERAPY IN RADIOWAVE DAMAGES

PHYSIOTHERAPY *

Asthenic syndrome and hypotensive type vegetovasal dysfunctions, are influenced by physiotherapeutic agents with a tonic and generalized supportive effect. The following are shown: sea treatment in the Black Sea resorts, mountain climate in Pamporovo, Borovets, Peshtera, Kurtovo, Yundola, balneotherapy in Bankya, Narechen, Varshets, Banya - Karlovo, Velingrad. Effective are: bats with brobalil, tripinate and juniper (36-37°C, 10-15 min, 12-15 procedures), galvanic collar of Shcherback with calcium and bromine (6-16 m A, 6-16 min, 10-12 procedures), brush body massage (15 -20 min, 12-15 procedures), acupressure at points: DM20, PM10, MD4,11, St36, DP6, P9,3E5, 1-2 min at a point, 10-12 procedures .

In vegetative-vascular dysfunctions of the hypertensive type, sea and mountain climatic treatment is carried out in the above-described resorts during the cold seasons. Also shown are carbonic wather and oxygen baths (35-36 ° C, 10-5 min, 10-12 procedures). Hypotensive effects are expressed by the four-chamber at Hauffe baths, especially combined with juniper, brobalyl, tripinate (37-42 ° C, 15-20 min, 10-12 procedures). It also uses a galvanic collar of Shcherback with calcium and bromine, brush body massage, psychosensory relaxation with speech, music, color, aromas (45-60 min, 8-12 procedures), acupressure in points: P6, 9, 3CM20, MS4, 11, 15, DP6, 1-2 min at a point, 10-15 sessions.

KINESITHERAPY **

In radio wave injuries, kinesitherapy contributes to the increase of the body's general tone, normalization of the basic nervous processes, ortostatic reactions, vascular tone with improvement of blood circulation and the psycho-emotional tone of the patient. Depending on the predominant syndrome, a kinesitherapeutic program is being developed. Teaching in autogenous training from different starting positions with and without musical accompaniment is required. Respiratory gymnastics includes: exercises to improve passability of airways; to regulate airflow and breathing rhythm; exercises for training and strengthening the respiratory muscles; exercises to improve diaphragm mobility, etc. The clino - and orthostatic reaction of the patient, the balance and the coordination is trained. Good effect is swimming, cycling, skiing, tourism and more.

15.2.4. PHYSIOTHERAPY AND KINESITHERAPY IN RADIATION DISEASE

PHYSIOTHERAPY *

The decorporating (etiopathogenetic) effect on the radionuclides of drinking (3-4 times 250 ml, 45 min before meals, 38-40°C, 4-6 weeks) and bath application of the following bulgarian mineral waters: hydrocarbonate-sulphate (Merichleri), low mineralized and gas waters (Birmirci, Kyustendil), weak mineralized (Gorna banya, Pancharevo), fluors (Sandanski, Haskovo mineral bats, Sliven mineral baths)

At initial stage are used hardening procedures, thalasstherapy in the Black Sea resorts, mountain climate treatments in Kurtovo, Pamporovo, Peshtera, Yundola, Borovets, general brush massage (15-20 min, 12-15 procedures), cold showers, bathing, rubbing, sauna, etc.

KINESITHERAPY **

The use of the kinesitherapeutic program depends on the nature of the damage and the stage of the radiation illness. Kinesitherapy increases the body's general tone, stimulates cortical processes, helps normalize the functions of different organs and systems, and improves the patient's psycho-emotional

tone. Applied: music therapy, psychosensory relaxation or autogenic training from different starting positions; breathing gymnastics, including exercises to improve the strength of the respiratory muscles and the chest, to strengthen the diaphragm; exercises and techniques with beneficial effects on the cardiovascular system and on the musculoskeletal system; terrain treatment; underwater gymnastics with a water temperature of 34-35 ° C; occupational therapy (functional, professional) and in the first stage of the disease and mobile games and elements of sports disciplines.

15.2.5 PHYSIOTHERAPY AND KINESITHERAPY IN CAISSON DISEASE

PHYSIOTHERAPY *

After exiting the recompression chamber physiotherapy helps to separate the nitrogen from the tissues by: general classical massage (30-40 min.), hot dry air or water procedures (steam bath, hot tub - 39-40 ° C, 8-10 min) or lighter (SOLUX) - 15-20 min.

In chronic forms, physiotherapy appropriate to developed syndromes is employed: e.g. Meniere's syndrome(See Physiotherapy for vestibular analyzer damage), which distorts arthrosis (see Physiotherapy of musculoskeletal system disorders).

KINESITHERAPY *

Kinesitherapy is dependent on the clinical form of caisson disease. Muscular - articular forms are administered: treatment by means of a situation in which the posture of the limb is changed and maintained in a physiological situation, isometric training in different modes (1: 3 or 1: 1 - contraction: pause); respiratory gymnastics to improve ventilation and circulation in the lungs, to regulate respiratory rhythm, airflow, and to strengthen the respiratory muscles and diaphragm. Relaxing and drawing methods used in respiratory gymnastics and autogenous training are used. After pain relief, the exercises are in isotonic mode in a 1: 1 ratio - relaxing. They also apply to general tonic exercises from different starting positions.

In case of pronounced hemiparesis or hemiparalysis with aphasia, ataxia, etc. the kinesitherapeutic complex includes: treatment by position; the specialized methodologies of Kabat, Bobat, etc.; cryotherapy; massage (relaxing), and at aphasia and exercises to restore speech functions.

In case of Meniere's syndrome, autogenous training is applied in the patient's preferred position; exercises for training the vestibular apparatus from relieved baseline at the beginning of the treatment course; coordination exercises; breathing and general tonic exercises.

In neuritis of the facial nerve after testing, the patient performs mimic gymnastics under the control of the kinesitherapist.

15.2.6. PHYSIOTHERAPY AND KINESITHERAPY IN MOUNTAIN SICKNESS

PHYSIOTHERAPY *

Physiotherapy aims to increase the general psychophysical sustainability of organism through general fortify procedures.

Apply: climatic impact of the high mountain associated with physical activity and sharp change of the mountain altitude, achieved with specially selected tourist routes. Shown are: balneotherapy with slightly mineralized waters (Bankya, Varshets, Knyazhevo, Gorna Banya, Stara Zagora mineral baths Banya - Karlovo, etc.), sun - and sea-prophylaxis, hardening bath treatments (sauna, pouring and rubbing with cold water, brush massage the entire body (15-20 min, 10-12 procedures), acupressure points in: St36, DCH4, DP6, 3E5, 3SM20, 1 min at any point 10-12 procedures.

KINESITHERAPY **

When operating in conditions of reduced atmospheric pressure prepares a long-term kinesitherapeutic program for training and adapting the body requirements of the working environment. The program includes exercises and techniques with beneficial effects on the cardiovascular system (improving venous and lymphatic flow, to regulate arterial pressure, supports heart, training orthostatic effects, etc.), breathing exercises with training in abdominal-diaphragmatic, thoracic and yogic breathing; exercises and methods for nervous system response and special ones for the visual, auditory and tactile analyzers; Exercises to develop muscle strength and endurance, coordination and balance; exercises acting relaxing and mobilizing on certain segments of the back muscles, etc.; sport disciplines (swimming, cycling, skiing, basketball, etc.).

15.2.7. PHYSIOTHERAPY AND KINESITHERAPY IN VIBRATION DISEASE

PHYSIOTHERAPY*

Physical therapy is administered throughout the course of the disease, but the most effective is in stage I and II. Physical agents are applied segmentarily in the root zone of C3-T4. Shown are: ultrasound paravertebrally (0,2-0,4 W/cm^2, 4-5 min, 10 procedures), electrophoresis with novocain, pymadin, nivalin, potassium iodide, calcium chloride (10-15mA, 10-20 min, 10 -12 procedures), Shcherback collar with calcium, bromine, magnesium (6-16 mA, 6-16 min, 10-12 procedures), diadynamic currents (1 min DF, 3 min KS, 3 min DC, 5-15 mA, 10 -12 procedures), microwaves (0.36 W/cm^2, 10-12 min, 10-12 procedures), ultraviolet erythemas in the form of a sea collar (1-3 bio-doses, 5-6 procedures).

Has expressed anti-pain and improving vascular tone effect of low-frequency magnetic field (16,000

A/m, 0,3-0,4 s, 1 Hz, 15-20 min, 15 procedures), in the same acupuncture points; of laser puncture (6 mW, 30 s to 1 min at point) and acupressure by 1 min at point: points ZHM20, PM10,11, DCH4,10, 3E5, P6, St36, TCH11, 10-12 procedures.

In resort situation apply: four- camera baths, or baths with juniper, brobalil, thymiane, sulfidium, sulfolil (36 ° C, 10-15 min, 12-15 procedures), sulfide baths (Separeva banya, Kyustendil), radon baths (Momin prohod, Narechen, Pavel banya, Strelcha) iodinebromine baths (New and Old Oriahovo, Slanotran, Shabla Shkorpilovtsi), sea treatment, mud treatment (38-40 ° C, 20-30 min, 10-12 procedures), general brush massage (15-20 min, 10-12 procedures) or underwater massage with tangentor (36-37 ° C, 1 atm, 20-30 min 10-12 procedures).

KINESITHERAPY **

Kinesitherapeutic complexes in vibration disease prepares on the basis of the syndrome leading to the clinical picture and the stage of the disease. The kinesitherapy procedure begins and ends with a total relaxation of 10 minutes. In patients with stage I, the ratio between analytical exercise in isotonic mode and free relaxation-active exercise is 1: 1. In II and II-III stage of disease at the position of general relaxation, forearms are resting on the pads and raised to 40 °, with the ratio between the analytical and relaxation exercises is 3: 1. They are based on breathing and general low tonic exercises.

In the II-III stage of the disease, the method for segmental-reflective action on vegetovasal disorders (described in vegetative polyneuropathy) has a very good effect.

Underwater gymnastics in vibration disease is conducted at a water temperature of 36 ° C ± 0.5 ° C, 8 to 25 minutes, 15 procedures. Analytical exercises for upper limbs in isotonic mode at different rates and amplitudes have a shoulder belt in the aquatic environment. They are followed by breathing exercises where the heart area is above the water. The basic principles of kinesitherapy are followed by exercising with the lower limbs and the corpse. After 3-4 procedures, elements of swimming and water polo games are also included.

15.2.8. PHYSIOTHERAPY IN PROFESSIONAL DEAFNESS *

The disease is refractory, which is why physiotherapy is early and multi stage. It aims to improve blood circulation and conduction of the nerve. In the initial "acute" stage ionophoresis with nivalin, pymadin novocain, potassium iodide, stugeron, 0.5-1.2 mA, 10-15 min, 10-15 procedures is assigned.

The chronic stage has beneficial effects with a low-frequency magnetic field (12,400 A/m, 0,2 s, 1 Hz, 15-20 min, 20 procedures). Efficient is a complex acupressurec acupressure combined with techniques of classic massage - rub, caress the neck and the area around the ear). Acupressure is applied at the following points: PM10, DM2, 8, 12, 20, 19, 3E 5, 19, 20, 21, 23, 2 min at a point, 15-20 procedures.

5.3. PROFESSIONAL DUST RESPIRATORY DISEASES

PHYSIOTHERAPY IN SILICOSIS *

It is a long-term and aim delaying the course of the disease, fighting infections and complications.

In the initial stage particularly efficient methods for suppressing silicotic fibrosis are: aerosoltherapy with polyvinylpyridine - N oxide (R 204) 2 ml, 10 min, 15 procedures suberythemal UV irradiation of the thorax (10-15 procedures), ultra high currents (oligotermal dosage, 10-15 min, 10-12 procedures). For elimination of the powder, expectorating and antiinflammatory apply: warm-damp, salty-alkaline and alkaline inhalations (2 ml, 10 min, 10-12 procedures) aerosoltherapy with radon mineral waters (Momin prohod, Hissar, Krasnovo, Pchelin, Strelcha), hydro-carbonates (Velingrad, Birmirci, Knyazhevo, Mramor, Gorna Banya), chloride (Mirovo, Varbitsa, Novo and Old Oryahovo, Ovcha mogila), iodine-bromine (Shabla, Slanotrone and Shkorpilovtsi) and low mineralized waters (Hissar, Bankya, Pancharevo, Varshets, Banya - Karlovo). Inhalatory, bronchodilatory, mucolytic, antibacterial preparations are also used. Climate and balneotherapy are very important, especially in the I and II stages of the disease. Specialized resorts are: Varna Black Sea Coast, Bankya, Kyustendil, Dolna Banya, Levochevo, Smolyan, Velingrad, Shipka, Troyan. Significant role plays the primary and secondary physioprophylaxis performed in the prophylactorium. The following are used: bronchial inhalation "toilet" with low-mineralized and alkaline waters, ultraviolet irradiation in solariums, water-sun- and aeroprocedures with hardening effect.

PHYSIOTHERAPY IN DUST CHRONIC BRONCHITES *

It is anti-inflammatory and has a bronchodilatory effect. In the phase of exacerbation apply: aerosoltherapy with sulfonamides, mucolytics, bronchodilators (2 ml, 10 min, 10-12 procedures), or with mineral waters: alkaline (Bankya, Velingrad, Pavel bani, Hissar Knyajevo) sulfide (Separeva banya, Kyustendil), radon (Momin prohod, Narechen, Krasnovo, Strelcha, Pchelin) iodine-bromine (Slanotran, Shabla, Shkorpilovci), salty (Mirovo, Old and New Oryahovo, Varbitsa, Ovcha mogila), carbon-acid (St. Karadzhovo, Mihalkovo Ilientsi, Mramor), low mineralized (Varshets, Banya - Karlovo, Gorna Banya - Sofia, Stara Zagora mineral baths). Also recommended are: suberythemic thoracic radiation with ultraviolet

rays (10-12 procedures), ultrashort wave (oligothermic dosage, 10-15 min, 10-12 procedures), low-frequency magnetic field (16,000 A/m, and 1 Hz, 0, 2 s, 15-20 min, 10-15 procedures), chest massage with vibrating and percussion techniques, acupressure in points: BD 1, 7, 9, MS4, PM13, 3CM14, MD11, 2 min at a point, 10-15 procedures, total Vermell calcium iontophoresis (10-20 mA, 15-20 min, 10-12 procedures).

In the phase of remission are shown aeroionotherapy (10-15 min, 10-12 procedures), aerosolotherapy with expectorant and anti-inflammatory drugs, sea treatment, mountainous climate treatment (Levochevo, Smolyan, Troyan), balneotherapy (Sandanski, Momin prohod, Velingrad). The healing procedures are important: sunny - air baths, sea baths, bathtubs and showers with cold water, sauna and others.

KINESITHERAPY IN PROFESSIONAL DUST DISEASE OF RESPIRATORY SYSTEM **

Kinesitherapy is shown predominantly in I and II (compensated and subcompensated) stage of the disease. Tasks for general positive impact and improvement of the functions of the cardiovascular system are solved with general - developed exercises and dosed training with walking, climbing and descending on stairs, swimming, biking, etc. Special tasks on respiratory function are solved with purposeful breathing exercises in the form of systemic breathing gymnastics.

The parts of the lung with sustained elasticity are moved by appropriate starting positions and various ways of assisting chest excursions and the unfolding of the pulmonary parenchyma.

Patients with pneumosclerosis and leading restrictive syndrome, respiratory failure with diaphragmatic breathing, tachypnea and shallow breathing, should not be reduced much respiratory rate, and to find optimum frequency, depth and rhythm of breathing under which consumes minimum energy to overcoming resistance.

In case of gas diffusion disturbances, the use of a characteristic breathing technique with pause in maximum breathing is indicated. Patients with interstitial fibrosis, accompanied by chronic bronchitis and emphysema, who have respiratory disturbances at rest, are usually able to perform a significant volume of muscular work. Physical effort can reduce bronchospasm. Assisted ventilation is applied not only to normalize air distribution but also to reduce respiratory rate within certain limits, extend the exhalation phase and reduce residual air, when increased. Attention is paid to the relaxation and enhancement of respiratory muscles. In this way breathing is economized. In cases of increased secretion, drainage situations and exercises are used. Respiratory gymnastics should be combined with oxygen therapy and aerosolotherapy.

15.4. OCCUPATIONAL OVER-TENSION

15.4.1. PHYSIOTHERAPY AND KINESITHERAPY IN MUSCULOSKELETAL DISEASES

PHYSIOTHERAPY *

Physiotherapy attempting to remove inflammation, pain, improve circulation and trophic tissue, restoring the function of the hand. Physiotherapy is applied in all three stages of the disease by hand overtension.

In the first stage are preferably administered antiinflammatory and anti pain means: diadynamic currants (local and segmentary - reflex zone of the C3-T4,1 min MF, 3 min KC, 3 min DS, 5-15 mA, 10-12 procedures) interference currants (constant frequency 100 Hz, 15 min, 10-12 procedures), ultraviolet erythemas as a "cuff" over the pathological process and in the form of a "mariner's collar", (1-3 bio-doses, 5-6 procedures), galvanic collar of Shcherback (6-16 mA, 6-16 min, 10-12 procedures). The anti-pain effect of acupressure at the points: D4, 11, 15, TH3, 11, 3E5, 14, GM 20,21, PM 10, 11, 60, St 36,P 6, 1-2 min in point, 12-15 procedures.

In a second stage in the phase of exacerbation besides the above-described physiotherapy agents are also such to improve blood circulation and trophic: ultra high frequency currents (oligohermic dosage, 10-15 min, 10-12 procedures), microwaves (0,36 W/cm^2, 10-12 min, 10-12 procedures), ultrasound or phonophoresis with ointments with voltaren, indometacin, hydrocortison (0,4-0,5 W/cm^2, 5-6 min, 10-12 procedures), ionophoresis with novocain, lidocain, potassium iodide , nivalin, calcium and others. (10-15 mA, 15-20 min, 10-12 procedures). With expressed anti-pain and anti-inflammatory effects include: acupressure (described above), laser puncture at the same points (6 mW, 30 s - 1 min, 8-12 procedures), low-frequency magnetic field (14,400 A/m, 1 Hz, 0, 5-0.6 s, 15-20 min, 10-12 procedures). In the phase of subsidence of acute phenomena apply baths with sulphide waters (Separeva banya, Kyustendil), radon waters (Momin prohod, Narechen, Velingrad, Pavel banya), iodine-bromine waters (Shabla Shkorpilovci), salted (Mirovo, Vurbitsa, Slanotran), paraffin procedures (30 min, 55 °, 10-15 procedures), mud applications (40-42 ° C, 20-30 min, 10-12 procedures).

In the third stage, physiotherapy is predominantly symptomatic - against pain, contracture, degenerative changes, adhesions, reduced volume of movement, etc. Apply: phonophoresis with aminosin,

voltaren, indometacin, ionophoresis with potassium iodide, novocain, lidocain, low-frequency magnetic field, UHF microwave, lye compresses (38-40 ° C, 20-30 min, 12-15 procedures), mud, applications acupressure, cryotherapy (with an ice bar, rounding for 1 minute, repeated 3-4 times, according to individual tolerance, 8-10 procedures).

KINESITHERAPY **

The construction of individual complexes in kinesitherapy is based on functional disorders in the limb and the localization of injuries. Depending on the stage of the disease, a healing-sparing, healing-tonic or healing-training regime is applied.

In the healing-sparing regime, damaged structures influenced indirectly in the first days. The patient is trained in the techniques of general relaxation, which is applied for 10 minutes at the beginning and end of the procedure. His attention is directed to localized pain for the application of the so-called auto select method. The analytical exercises for the analogous muscle groups of the injured are aimed at their reflective repercussion. They are without and with damages, with manual resistance at different rates and from different starting positions. For the damaged limb, the first days are given only free active with relaxing effect exercises. Exercise from lighter position eliminates pain, relaxes muscles and improves haemodynamics. When the muscles of the forearms are damaged, the movements are performed initially in the shoulder joint. Later, flexion and extension of the elbow joint was provided, which involved the injured structures from relieved baseline. Movements are combined with breathing exercises. General tonic exercises for the carcase and lower extremities with high intensity are applied to the general principles of kinesitherapy and to the improvement of general blood and lymphocirculation.

The healing-toning regime is progressing gradually. Affected structures are involved in movement first as synergists and later as agonists. The action of the muscles in the fingers is performed with the synergistic activity of the muscles in the wrist and vice versa. Isometric training is in different modes. At the beginning the contraction: pause is 1:3, and later changes 1:1. Analytical exercise in isotonic mode is 1:1 ratio, with free, active relaxing exercises. Exercises for the contralateral limb are without appliances, with appliances and appliances against maximum resistance. The complex includes breathing, co-ordinating exercises and games that improve not only the general blood- and lymphocirculation and the psycho-emotional tone of the patient.

The healing-training regime is applied after pain relief. Affected structures are included as progressive loading agonists. The exercises are without appliances, with appliances and appliances, against adequate resistance. Gradually, isotonic movements acquire the character and approach the rhythm of the productive activity with the active involvement of the damaged structures.

15.4.2. PHYSIOTHERAPY AND KINESITHERAPY IN PERIPHERAL NEURVOUS SYSTEM DISEASES

PHYSIOTHERAPY IN LUMBOSACRAL AND CERVICO-BRAHIAL RADICULITIS *

Physiotherapy aims to combat pain, improve the inflammatory process, nervous conduction and waste motor and sensory manifestations. Anti-pain and anti-inflammatory apply diadynamic currents (or diadynamophoresis with novocain, lidocain, 1 min DF, 3 min KC, 3 min DS, 5-15 mA, 10 procedures), novocain and potassium iodine ionophoresis (10-15 mA, 20 min, 10 procedures), interference currents (constant frequency 100 Hz, 15 min, 10-12 procedures), low frequency magnetic field (16,000 A/m, 0,2 s, 1 Hz, 15-20 min, 12-15 procedures) , ultrasound/phonophoresis with indometacin, voltaren, hydrocortisone, 0.5-0.6 W/cm^2, 5-6 min, 10-12 procedures), ultraviolet erythemas (1-3 biodoses, 3-4 fields, 6-8 procedures), microwaves (0.36 W/cm^2, 10-12 min, 10-12 procedures), ultra-short waves (oligothermal dosage, 10-15 min, 10-12 procedures). A good application effect (at the same acupuncture points) of acupressure and laserpuncture (6 mW, 1 min per point) was found. The cervico-brachial radiculite dots complex is: JM12, 20,21, PM10, 11,13,3CM 14, 3E 5,14,15, TH3, 9-15, D4, 11, BD1, and for lumbosarral radiculitis: 34, 36, 40, 60, 62, DM 30, 1 minute at a point, with the soothing acupressure method, 12-15 procedures.

For elimination of waste phenomena and improvement of nerve conduction: ionophoresis with pymadin, nivalin (10-15 mA, 10-20 min, 12-15 procedures), stimulation with low-frequency currents (according to individual parameters) or with sinusoid modulated Currents (also on an individual sheme).

In discopathy are administered by manual therapy, extensive therapy, acupressure (as described above).

In the chronic stage, balneotherapy with sulphide waters (Sapareva Banya, Kyustendil), radon water (Momin Prohod, Pavel Banya, Velingrad) is performed.

KINESITHERAPY **

The aim of kinesitherapy here is by stimulating the regenerative processes to restore nerve conduction, preventing the formation of secondary deformations, and in case of irreversible defects, compensation is formed.

Kinesitherapy begins the first few days with exercises for the healthy limb against manual resistance and instruments to reflexively affect damaged structures. For paretic muscles passive exercises are given several times a day, for undamaged muscle groups - active. It starts from the proximal to the distal parts of the limb. Passive and active exercises improve circulation, reduce venous- and, lymphatic stasis and concomitant edema. Movement in the joints and pulling of the paretic muscle groups prevents adhesions and joint contractions. Treatment is also applied by a position in which the joint is in a functional position.

The kinesitherapy and massage procedure should be light, non-traumatic, as the denerved muscle fibers are sensitive to mechanical trauma, that accelerates atrophy and leads to degeneration.

With the onset of active movements, the following additional tasks are set: elimination of substitution and compensatory movements; improvement of exteroreceptive sensitivity and coordination; complete restoration of muscle function and muscle strength (note that during the restoration phases, all senses in the limb acquire a hyperpathic tinge). A good effect is underwater gymnastics. Massage techniques paravertebrally on cervical and thoracic vertebrae have a reflective effect on injured structures. Kinesitherapeutic procedures and massage procedures should not cause pain as it leads to increased muscle tone and contractions. Procedures choose the optimum starting position. Active moves are sought, maintained and developed. In the presence of willing contractions by isolated accurate movements (analytical gymnastics with adequate dosage), a given muscle is trained. Passive movements to maintain the volume of movement are also applied. The method of proprioceptive neuromuscular facilitation is also used, where the contractions of the paretic muscles are facilitated by those of the preserved muscle groups. During the procedure, the paretic musculature is contracted and the antagonists are stretched. In definitive changes, when nerve damage is not recovered, attention is directed to training in substitution movements.

15.4.3. PHYSIOTHERAPY AND KINESITHERAPY IN VEGETATIVE POLINEUROPATHY AT THE UPPER LIMBS

PHYSIOTHERAPY *

It aims at increasing resistance and improving the local pathological process - influencing vascular, vegetative and trophic disorders; pain relief; improved hand functions. It is recommended that the segmental-reflective application of the physical factors in the paravertebral zone of C3 - T4 is recommended. In the first stage to improve the general condition, bats with juniper, trypinate, brobalil(36-37°C, 10-15 min, 10-12 min, 10-12 procedures), Scherback galvanic collar (6-12 mA , 6-16 min, 10-12 procedures), general brush massage (15-20 min, 10-12 procedures). They have anti-pain effect: ultraviolet erythemas (1-3 biodoses, 6-8 procedures in the form of seaman's collar), diadynamic currents or diadynamophoresis with novocain (1 min DF, 3 min KC, 3 min DS, 10-15 mA, 10-12 procedures). Amplification of trophic and vascular changes with other physical factors: low-frequency magnetic field (16,000 A/m, 1 Hz, 0,2 s, 15-20 min, 15 procedures); Acupressure in points: DM20,21, PM10,11, 3CM14, MD4, 11, Th3,3-15, 3E5, St36, DP6, 1-2 min at a point, 12-15 procedures; laser puncture at the same points, 6 mW, 30 s - 1 min at a point, 10-12 procedures. In the second and third stages except the above procedures are shown: sinusoidal-modulated currents (in alternating mode, III gen, 80-100 Hz, depth of modulation 25%, series duration 1-2 s), interference currents (with constant frequency 100 Hz, 15 min, 10-12 procedures), microwaves (0.36 W/cm^2, 10-12 min, 10-12 procedures), ultra high frequency currents (oligothermal dosing, 10-15 min, 10-12 procedures), ultrasound 0.2-0.4 W/cm^2, 5-6 min, 8-10 procedures), mud collar (38-40°C, 20-30 min, 10-12 procedures), paraffin application (55°C, 20-30 min, 10-12 procedures) - all described procedures applied segmentary-reflectory In the C3-T4 zone. Common baths or bath tubs for hands are also shown with: sulphide mineral waters (Sapareva Banya, Kyustendil), radon mineral waters (Momin Prohod, Velingrad, Strelcha, Pavel Banya). Climate treatment of the Black Sea resorts is recommended.

KINESITHERAPY **

In patients with pronounced angiospastic syndrome, the ratio of analytical exercises to isotonic mode and free, active relaxing exercise is 1: 1. In those with spastic-atonic and atonic vessels, analytical exercise in isotonic mode is up to 90 ° forearms and is in a 3: 1 ratio with relaxation exercises. Procedures begin and end with total relaxation.

The best and quickest effect on vegetovasal disorders in patients with moderate and severe disease is combined segmented-reflexology.

Segmentary-reflective effect is accomplished by:

1. General relaxation of occipital leg with thermal procedure performed by a cushion of wool cloth in the area of the cervical and upper thoracic vertebrae - 10 min.

2. A 15 minute procedure where the patient's head is outside the couch on the knee of the kinesitherapist for free access to the neck area. Performed: superficial and deep stroke; treatment of reflective changes in the skin, connective tissue, muscle and periosteum by appropriate toning or

sedative techniques, acupressure paravertebral at the base of the skull and on C4-5; manual resistivity of head movements and on distal parts of forearms; passively, flexion, extension, rotation and circles are performed, followed by low flexion of the head with a manual extension. Again in the position of general relaxation patient performs isometric training the muscles of the upper limbs and shoulder girdle, abdominal-diaphragmatic breathing and relaxation.

Under-water gymnastics also have a good effect.

15.4.4. PHYSIOTHERAPY AND KINESITHERAPY IN OCCUPATIONAL DYSKINESIAS

PHYSIOTHERAPY *

Physiotherapy is prolonged, multi-stage and aim to relieve congestive focus in the central nervous system and eliminate pathological reflexes by strengthening and sedative procedures. There are: mountain climates (Troyan, Ribaritsa, Yundola, Peshtera, Pamporovo, Borovets) and sea treatment during the cool seasons in the Black Sea resorts. Balneotherapy procedures are also displayed in Narechen, Varshets, Bankya, Velingrad, as well as medicinal baths with: juniper, tripinate, brobalil, rosemary (36-37 ° C, 10-15 min, 12-15 procedures).

Effective are: ultralow wave (oligothermic dosage, 10-15 min, 10-12 procedures), galvanic collar on Shcherbak with calcium, bromine, magnesium (6-16 mA, 6-16 min, 10-12 procedures), a general brush massage (15-20 min, 10-12 procedures), psychosensory relaxation with speech, music, colors, aromas (45-60 min, 8-12 procedures), acupressure at points: St36, DP6, 3CM20, 11, P6, 2 min at a point, 12-1 5 procedures.

KINESITHERAPY **

At coordination neuroses (dyskinesias) by means of kinesitherapy improve strength, mobility and steadiness of the main nerve processes, suppress be pathological reflexes and recovers dynamic stereotypes of the working movement. Kinesitherapy includes: autogenous workout, soothing massage, stretching of the limbs along their longitudinal axis using the Schaarchuch method, free active relaxing exercises, proprioceptive neuromuscular facilitation, exercises with antagonist replacement, underwater gymnastics, underwater jet massage, suspension therapy, field treatment, breathing gymnastics, swimming, tourism and more.

15.5 OCCUPATIONAL ALLERGIC DISEASES - BRONCHIAL ASTHMA

PHYSIOTHERAPY *

Physiotherapy apply: non-specific desensitization of the organism, anti-spastic, anti-inflammatory and decongestant effect on the bronchi, restoring the function of the external breathing, rehabilitation of foci.

During the attack, aerosol therapy with bronchodilators (astmopen, ventolin, salbutamol, berotec, inthal, etc., 2 ml, 5-10 min) is the most effective. Very effective is the application of various physical factors in acupuncture points (acupuncture, electroacupuncture, laserpuncture, acupressure). The following points are used: BD1,2,7, B27, Ст9,10,13,36, PMM 17,21,24, 3SM14, PM10, 43, GM20, 21, DC4,10, DP6. In case of attack, four chamber baths at Haufe (for redistribution of blood to the limbs and alleviation of the lungs) are also administered - 37-42 ° C, 15-20 min.

Outside attack applies aerosolterapy with anti- allergy (antihistamine) drugs such as: alergosan, sinopen, dimedrol and oth., mucolytics (mucosolvin, bromhexin, sodium bicarbonate, etc.) 2 mln 10 min, 15-20 procedures and with radon mineral water (Momin Prohod, Narechen, Velingrad), hydrocarbon sulphate (Sandanski). Effective are: air ionization (10-15 min, 12-15 procedures), low-frequency magnetic field in the adrenal area and local (16,000 A/m, 1Hz, 0,2 s, 15-20 procedures), ultrasound (segmentaryreflexly) in three zones : two fields paravertebral T_1-T_{12}, 0.2 W/cm^2, 2 min, VI and VII intercostal bilateral, 0.4 W/cm^2, 2 min, subclavicular areas 0.2 W/cm^2 for 1 min, 10-15 procedures, UHF (oligothermic dosage, 10-15 min, 10-12 procedures), ultraviolet irradiation of the thorax or in total in suberithemic dosage 10-12 procedures classical massage chest with vibrating and percusion devices (10- 15 min, 10-15 procedures). The resort treatment is essential in the specialized resorts: Sandanski, Momin Prohod, Velingrad, Levochevo, Smolyan, Troyan, Dolna Banya, Kostenets, Black Sea resorts.

Physioprophylaxis includes: aero-, sun- and hydro procedures hardening; rehabilitation of the inflammatory processes of the upper respiratory tract with physioprocedures (UHF, UAV, inhalations, etc.).

KINESITHERAPY **

Kinesitherapy in patients with bronchial asthma is applied in out-paroxysmal periods. It aims to: reduce the hypersensitivity of the body to allergies by destroying the pathological dominant and pathological reflexes; to normalize major nerve processes; to strengthen the patient's psyche by training him in proper, rhythmic breathing. Often, only relaxing of muscles improves breathing. Achieved by: psychic response (verbal, music, abducting therapy); with autogeneous side-bed, bed, seat, training etc.; with relaxing exercises for the upper limbs, the shoulder belt and the chest; rhythmic massage with vibration of the chest with shaking; with relaxing-pulling

poses and more. Respiratory gymnastics includes: breathing exercises for general impact; to improve the passing of airways; exercises and means for regulating airflow and breathing rhythm; exercises for training and strengthening the respiratory muscles and improving the mobility of the chest and diaphragm; exercises to unblock the diaphragm, as well as exercises to control the cough and to assist expectoration. Particular attention is paid to the exhalation, which should be slow, soft and prolonged, without impulses and unnecessary strain. In the presence of emphysema the patient is assisted in the expiratory phase. For respiratory gymnastics appropriate starting positions, poses and stands are used. It is important for the patient to develop habits for reconciliation of breathing with general development and applied exercises (walking, climbing and descending, body inclines and curves, etc.).

15.6. PROFESSIONAL MALIGNANT NEOPLASMS **

A complex rehabilitation program is needed in cancer patients. It is important to initiate early rehabilitation, patient co-operation and relatives, gradual transition from hospital to ambulatory and home rehabilitation.

Rehabilitation in patients with skin cancer

Kinesitherapy in skin-operated aids skin healing and prevents the formation of large scars that limits the function. With scars already formed in addition to gymnastic exercises in isotonic and isometric mode, massages, phonophoresis with anti-scarsing creams, ointments, gels (indomethacin, contratubex, madecasol, etc.) are also used.

In radical lymph dissections usually leave heavy and persistent lymph edemas that restrict limb function and lead to disability. With early targeted kinesitherapy, where rhythmic exercise in isotonic and isometric mode has a raised limb up to 45°C, lymphatic outflow is assisted and its functional fitness is maintained.

Rehabilitation in patients with lung cancer

With targeted kinesitherapy in the preoperative period of lung cancer patients, it contributes to: reduction of the purulent intoxication; improving the condition of the cardiovascular system and of the external breathing; strengthening the physical potency of the patient and increasing his neuro-mental tone. The exercises for the early post-operative period covering the first 15 days are also mastered. Respiratory gymnastics in a 1:1 ratio with mild, rhythmic isotonic exercise and early stand up of the patient (2-3 days after surgery), counteract pleural adhesions and have a definite psychological impact on the patient. Kinesitherapy during this period is also of great importance for the prevention of post-operative complications such as lung atelectasis, hypostathic or aspirational type bronchopneumonia, phlebotrombosis, embolism, etc.

With rehabilitation in the late postoperative period and in the home environment, the respiratory and cardiovascular systems and the general tone of the organism are maintained in the possible good state. There is also psychological adaptation of the patient in the family and society.

Rehabilitation in patients with nasal and sinus cancer

The development of the kinesitherapeutic program depends on whether radiotherapy or surgical treatment is performed. In radiation therapy the patient is trained in autogenous workout, rhythmic breathing and mimic gymnastics. After surgery, with subsequent prosthetics and speech disorder, kinesitherapy aims, in addition to restoring psychic equilibrium and possible correction of speech disorder. The patient is required to speak loud the vocals in front of mirror and then make syllables and small words like "alo", "echo", "mum", "sea" etc. combined with proper breathing. Autogenetic workout, mimic gymnastics, occupational therapies, which from amusement pass to functional and professional, are applied.

Rehabilitation in patients with larynx cancer

Rehabilitation is aimed at a possible recovery of the psyche and of the voice function. The patient is trained in autogenous training, rhythmic breathing with extended expiratory phase, abdominal-diaphragm breathing and isometric training of abdominal muscles. Rehabilitation of speech is accomplished through: training; voice prostheses; plastic surgical procedures. The so-called "esophagous voice" is best suited for this purpose, and training starts no later than 3-4 weeks after surgery (the air jet comes from the stomach and the esophagus and sounds forms at the entrance of the esophagus). In patients with larynx cancer, psychosensory relaxation, music therapy, occupational therapies (entertaining, functional, professional), hardening procedures are also applied.

15.7. OCCUPATIONAL DISORDERS OF THE VISUAL ANALYZER *

Good effect of the following physiotherapeutic complex: acupressure of specific points located around the orbit (PM1, 2, St1, 3E23, IM6 (yu-yao) 2min at a point, ocular relaxation (puts gently the palms of the patient himself on closed eyes for 10-15 minutes, on background of general relaxation),

exercise of ocular muscles with special exercises (close-up, look away, look down, up, left, right, circular clockwise and counter clock 10 to 15 times each exercise performed slowly, with breaks between them), 20-30 times, splash eyes closed with cold water every morning and after eye fatigue

15.8 OCCUPATIONAL CARDIOVASCULAR DAMAGE

PHYSIOTHERAPY IN HYPERTONIC DISEASE *

The hypotensive effect of sea climatic treatment in the cold seasons (Kiten, Nessebar, Varna) as well as the mountain climatic treatment associated with active kinesitherapy (Kurtovo, Peshtera, Borovets, Pamporovo) have been proved. Balneotherapy is applied in profiled resorts (Bankya, Narechen, Varshets). A specific effect is the four-chamber Haufeubaths, particularly suitable for a hypertensive attack (37-42 ° C, 15-20 min), as blood is withdrawn from the internal organs to the extremities. Manual baths with tripinate, juniper, brobalil (36-37 ° C, 10-12 procedures) are shown.

A positive response with acupressure was found in the following points: St36, 9, DP6, P6, 7, MS4, 11, 15, 3CM15, 16, 20, 23, DM20, PM10, PM14, 15, 25, 43, , CHD3, 1-2 min at a point, 12-15 procedures. Very effective is the following, tested by us acupressure method, which lowers blood pressure by 2.67 / 4.00 kPa. The patient is seated and even better lying down. Place the two pointers from the patient himself inside the ear canals (close the ears) and hold for 1 min. Remove the fingers and inhale several times very slowly and deeply. The manipulation is repeated three times.

Good effect: general brush massage (15-20 min, 12-15 procedures), underwater massage with tangentor - 1-1.5 at (0.1-0.15 MPa), 20 min, 36-37 ° C, 10-12 procedures; galvanic collar of Shcherback with calcium, bromine, magnesium (6-16 mA, 6-16 min, 10-12 procedures); general iongalvanisation of Vermell with calcium and bromine (10-20 mA, 10-12 procedures); psychosensory relaxation with speech, music, colors, aromas (45-60 min, 8-12 sessions).

KINESITHERAPY IN HYPERTONIC DISEASE **

Kinesitherapy contributes to the normalization of cortical processes and the elimination of stagnated excitability in the vasomotor centers, normalizing the dynamics of the autonomic nervous system, the cortical-visceral connections and the endocrine-humoral regulation of the vascular tone. In the first stage of hypertonic disease, the activities are group, everyday. Mostly free gym exercises are used for large muscle groups, performed without strain, fly, with maximum amplitude of movement and slow pace from all starting positions. Exercises with light devices (balls, clubs, hoops, dumbbells) and exercise of appliances (gymnastic bench and wall) are included in the main part of the procedure. A special place in the kinesitherapeutic complex occupies static and dynamic breathing exercises, performed slowly and rhythmically without breathing retention. Besides the positive effect on the respiratory function, they affect the blood flow and act soothingly. Similar is the effect of both passive and active relaxation with elements of suggestion and self-stimulation. The exercises with moderate dosage and isometric component have a stimulating effect on the blood supply and trophic processes of the heart muscle. In hypertensive patients with proven therapeutic effect are the equilibrium and coordination exercises, as well as the appropriate games and dances with appropriate musical accompaniment with adequate dosing of their emotional impact. With a marked normalizing effect on the functions of the cardiovascular system in patients with arterial hypertension, stage I is a dosing exercise for endurance with cyclic physical exercises: walking and running, cycling, swimming, walking and cross-country skiing, rowing, etc. Physical exercises should be in the so-called "steady state" (the oxygen supply of the body fully satisfies its needs) - aerobic workout.

In hypertensive stage II, the motor regimen is limited. Only components of sports disciplines are carefully used. They are made: massage collar and, if necessary, segmented massage or underwater jet massage, along with free, active, relaxing exercises at comfort start position. Blood pressure, pulse rate and breathing values are monitored.

In hypertensive stage III, the use of kinesitherapy is limited (consistent with the degree of cardiovascular failure).

15.9. OCCUPATIONAL DISABILITIES OF THE VESTIBULAR ANALYZER

PHYSIOTHERAPY *

Physiotherapy aims at improving the vestibular analyzers blood circulation, influencing neuro-vegetative and functional nervous disorders, influencing the general resistance of the organism. A good effect has been found in the application of a complex of: acupressure, low-frequency magnetic field, juniper baths. Acupressure is applied at points: 3CM15, 20, PM10, DM20, 2, 8, 12, 3E1, 4,5, 19, 21, 23, TC8, 19, CHD3, 1-2 min at a point, combined with rubbing and flattening around the ear, 12-15 procedures. The low-frequency magnetic field is applied in the ear area with parameters: 16,000 A/m, 0,2 s, 1 Hz, 10-20 min, 15-20 procedures. We also use juniper

or brobalyl baths (36-37 ° C, 10-15min, 10-12 procedures), general brush massage (15-20 min, 10-12 procedures), Shcherback galvanic collar with calcium, magnesium, bromine 6-16 mA, 6-16 min, 10-12 procedures).

Confounded bleeding of the vestibular analyzer responds well (especially if due to cervical osteoarthritis) by: paravertebral ultrasound in the C3-C7 area (0.3-0.4 W/cm^2, 3-4 minutes per field, 10 procedures), diadynamic currents (1 min DF, 3 min KS, 3 min DS, 5-10 mA, 10-12 proc.), ionphoresis with KJ at neck (10-15 m A, 15-20 min, 10-12 procedures), paraffinic applications at neck (55 ° C, 20-30 min, 10-12 procedures), mud application (38-40 ° C, 20-30 min, 10-12 procedures). Very good effect is the psychosensory relaxation with speech, music, aromas and color effect (45-60 min, 8-12 sessions).

KINESITHERAPY **

The aim of kinesitherapy is to lower the threshold of excitability of the vestibular and vegetative response by restoring the patient's stability and orientation in space and adapting it to physical stresses in different body positions.

In the case of significant vestibular disorders with violent vegetative disorders, the aim is to eliminate the psychogenic violation, to influence the vegetative nervous system, by the means of general relaxation in the patient's preferred position and breathing exercises. After the interruption of the attack in the initial period, at continuous eye and verbal contact, specifies the patient's tolerability to physical stresses and determines the plane where the exercise is most difficult to perform. Followed the vegetative reactions (fainting or redness of the face, sweating, increase or slowing of the pulse), blood pressure, the presence of spontaneous or latent nystagmus, statokinetic stability is determined. The special exercises revealing the particularities of the vestibular reactions are elementary, they are applied carefully to avoid an attack, with slow pace and pauses. The ratio of special exercise to respiratory and general tone exercises is 1: 2. The initial period is 2-3 days (with more pronounced vegetative reactions, the procedure is discontinued). Keep in mind that in middle ear disorders, movements in the frontal plane are difficult to handle; harassing of semi-circular channels-angular accelerations; in the case of the otolithic apparatus disorders - rectilinear acceleration.

The baseline period is 3-4 weeks. The aim is to restore the patient's statokinetic resistance. Special exercises are in proportion to the 1: 1 to breathing and general tone exercises in the first half of the healing course. The tempo is slow at the beginning and moderate at the end of treatment. In the first days the procedure starts and ends with the methods of general relaxation. The classes are individual. In the second half of the course, groups of three patients with similar disabilities and a close clinical picture are formed. Games and elements from various sports disciplines are also used.

In order to strengthen the established habits and prevent relapses, a home rehabilitation complex is being built.

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