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Occupational Diseases

Approved by the Ministry of Health of Ukraine as a manual for students and resident-physicians (intern) of higher medical educational establishments of the 3rd and 4th levels of accreditation

Kharkiv
2005

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Kostyuk I. and Kapustnyk V. Translated from the Ukrainian by _____
Occupational Diseases: Manual. – Kharkiv: Osnova, 2005. – 400 p.

Basic forms of occupational pathologies and their classification have been described in the manual. Issues on regional occupational diseases with the consideration of peculiarities and the structure of public economy of Ukraine have been considered. New data on pathogenesis, clinical symptomatology, development, treatment and organization of dispensary care have been suggested. Recommendations on rendering urgent medical assistance in case of some grave conditions and poisoning in clinics to treat occupational diseases have been provided. Principles on organization of conduct of medical examinations and solving expert questions in compliance with the regulations of recent acts have been analyzed.

For students and residents (interns)

CONTENTS

FOREWORD

Social and economic development of the society envisages improvement of labor conditions, as well as decrease of the level of general and occupational diseases. As to occupational diseases, their structure, character and progress have changed. Nowadays there is almost no severe forms of acute intoxication by industrial poison (nitric oxide or carbon oxide, hydrogen sulphide, aromatic nitrocompound or Oilsperse), as well as pronounced forms of chronicle poisoning with lead, tetraethyl lead, mercury, manganese, benzol, or other toxic matters. However, radically changing the work character, scientific and technological progress brings up new factors of production environment, which have negative effect onto the employees. In some spheres, there is a threat of the impact of existing production problems due to intensification of production processes. First of all, this concerns the expansion of the production of plastics, synthetic resins, leather, caoutchouc, organic dyes, chemical fertilizers, pesticides, as well as medicinal drugs.

Notions of factors of low intensiveness and combined impact of some harmful factors of production environment have appeared, thus a need have emerged for early diagnostics of occupational diseases.

In the occupational pathology, there is still an actual problem of dust diseases of lungs or pneumoconiosis, as in the structure of occupational diseases they are the most prevalent. The main form of lung dust pathology, as it was before, is black-lung disease or dust disease, and first of all, silicosis. Besides, together with black-lung disease, dust bronchitis has spread much.

Intensive development of electronics and radio technology conditioned the necessity to study the impact of electromagnetic emanation onto those, who work in these spheres of the economy with the purpose of timely elimination of its negative effect.

Implementation of high-pace equipment, which generates vibration and is a noise source, into various spheres of industry, causes vibration disease or cochlear neuritis. And in spite of the developed means and methods to struggle against these factors, vibration disease is one of the most prevalent diseases in the structure of occupational pathology.

Complex mechanization and automation of production processes together with the increase of labor efficiency allowed decreasing burden onto the muscle system. At the same time, partial mechanization and automation of some production lines causes physical overstraining for locomotor system, muscle overstraining, in particular, when monotonous movements are made in haste. That’s why occupational diseases of muscles, periphery nerves, and locomotor system can be met rather often and need timely diagnostics, treatment and preventive actions.

Thus, under conditions of modern production, occupational diseases still affect the health condition of working people. This situation needs correct evaluation of sanitary and hygienic conditions of work, intensiveness and duration of the influence of these factors in every case with the purpose of due diagnostics and treatment of patients with occupational diseases.

With the consideration of the aforementioned, collaborators of the Chair on Internal and Occupational Diseases of Kharkiv State Medical University have compiled this manual in compliance with the curriculum on occupational diseases for students of higher medical educational institutions of Ukraine.

Authors will appreciate any remarks and suggestions regarding the improvement of the manual.

Chapter 1

GENERAL ISSUES ON OCCUPATIONAL PATHOLOGY

Notion of Occupational Pathology as a Clinical Discipline

Occupational diseases with occupational pathology is a division of clinical medicine devoted to studying occupational diseases which appeared under the impact of harmful factors in the production environment or work process.

Main objectives of the occupational pathology are as follows:

- study of occupational diseases, their pathogenesis, symptomatology, progress, long-term consequences, therapy issues, medical rehabilitation, and labor ability expertise; early diagnostics of occupational diseases is gaining particular importance;
- study of non-specific action of occupational factors, their meaning for development, progress and consequences of general and non-occupational diseases.

The notion “harmful production factor” means a factor of production environment, as well as peculiarities of production process, which can cause harmful action onto the body of a working person and to cause diseases.

By impact of their nature onto a human body, dangerous of harmful factors of the production environment can be divided into physical, chemical, biological and psychophysiological ones.

Physical: machinery and moving mechanisms, moving elements of production equipment, falling rocks, high or low temperature of the surface, air in the work zone; increased level of noise, vibration on the work place; increased or decreased pressure within the work zone or its sudden change; increased level of ionizing emanation, etc.

Chemical: organic and non-organic compounds in the form of gas, vapor, aerosol or liquid.

Biological: biological objects, which include pathogenic microorganisms (bacteria, viruses, Rickettsia, spirochaetae, fungi, and photozoa), products of their life activity, as well as some organic matters of natural origin.

Psychophysiological: physical, nervous and mental strains, which in their turn can be divided into mental strain, analyzer strain, work monotony and emotional strain.

Appearance of dangerous and harmful factors in production can be conditioned by the following: wrong organization of work process (irrational work and rest regime, unnatural body position, extreme pressure on individual organs and systems), low production culture, lacking or insufficient working sanitary or technical devices and equipment; problems with sanitary and technological solutions at some productions (dust control in

coal and mining industries, normalization of microclimate at work places, and in deep mines); peculiarities of work processes, connected with strain in emotional sphere (complicated operator work under conditions of time deficit).

Unified classification of occupational diseases has not been developed until now. First of all, this can be explained by the fact that their clinical picture is often polymorph and can be characterized by the change of many organs and systems. Besides, they can be divided into specific and non-specific occupational diseases.

The former notion includes diseases, which can appear only in case of occupational factors. They can be also called “absolutely specific”. The second group of diseases includes “relatively specific” occupational diseases.

There are not that many absolutely specific occupational diseases: black-lung disease, vibration disease, or manganese intoxication.

Relatively specific diseases include intoxications, which sometimes have household origin, but more often they can be met in production conditions (intoxication with lead, mercury, arsenic, or pesticides), as well as radiation sickness, diseases of arms and hands due to functional straining. Particular clinical picture should also be taken into account.

Many diseases can be conditioned by not only occupational, but also harmful factors, though in specific professions and under the impact of specific occupational harms they can be met more often than in other conditions (bronchial asthma among fur makers and pharmacists; chronic bronchitis among workers of “dusty” professions, etc). These are non-specific occupational diseases.

The most specific classification of occupational diseases is etiological classification. In compliance with the etiological classification, groups of occupational diseases can be identified, which are conditioned by the impact of the following:

1) industrial dust (black-lung disease and dust bronchitis);
2) physical factors of the industrial environment (vibration disease, cochlear neuritis, affections caused by the action of various types of emanation, high and low temperature, etc);
3) chemical factors of production environment (various acute and chronic intoxication);
4) biological factors (infectious and parasite diseases, which develop in those, who are in contact with various infectious material or animals with some infections, as well as among those, who work in tuberculosis and other infection medical institutions; diseases, caused by antibiotics, fungi producers, etc).

Sometimes the classification of occupational diseases includes system and organ principle (occupational diseases of the nervous system, of respiratory apparatus, cardio-vascular system, blood, etc).

Diseases, which appear due to the impact of chemical compounds onto the body when working with it under conditions of production environment, are called occupational poisoning. It appears due to interaction of life organism and poisoning. The most popular classification of toxic matters, which reflects their practical utilization, is as follows:

1) industrial poisons used in production: organic solutions (dichlorethane), dyes (aniline), chemical reagents (methyl alcohol), etc.;
2) poisoning chemicals used against agricultural plant pests: organochloral pesticides (hexachloran, polychloropinen), organophosphorus insecticides (Trichlorfon, methylmercaptophos); organomercurous matters (granosan) and derivatives of aminoformic acid (Sevin);
3) medicinal drugs;
4) household chemicals used as food supplements (acetic acid), sanitary, personal hygiene and cosmetics means;
5) biological plant and animal poisons contained in plants and fungi, animals and insects (snakes, and bees); and
6) military poisoning matters (sarin, mustard gas or yperite, and phosgene).

Hygienic classification of poisons have been generally accepted, in the basis of which there is quantity assessment of toxic danger of chemical compounds based in experimentally determined fatal dose (DL_{50}) and permitted marginal concentration (MPC). In compliance with this classification, toxic matter corresponds to specific degree of toxicity, which is characterized by its stronger or weaker danger.

The most important for clinical toxicology is the division of chemical compounds on their toxic action onto the body (toxicological classification). The following poisons are distinguished: neuropsychic (organophosphorus insecticides), blistering (dichlorethane, hexachloran, arsenic, or mercury), general toxic (carbon monoxide), asphyxiating (nitric oxide), tear and irritant (vapors of strong acids and alkali).

Background of Development of Occupational Pathology

Studies of occupational diseases have deep roots. Specific data onto the impact of harmful conditions of work onto the human health, as well as existence of particular occupational diseases can be met in ancient documents. As an example, we can name ancient Egyptian and Chinese characters and a code of medical rules and lectures by Hippocrates, Aristotle, Lucretius, Ovid, Plutarch, Pliny, Juvenal, and Galen.

The first description of occupational pathology was done by Hippocrates (460 – 377 BC), who compiled a list of existing at that time the so-called lead professions and described the clinics of poisoning with lead in

detail. He also developed the description of negative impact of mining dust onto miners.

Significant growth of production in the late Medieval period (12^{th} – 15^{th} centuries), hard exhaustive labor in such spheres of industry as mineral resource industry and metallurgy, revoked interest in issues on labor hygiene and occupational diseases. In 1530, three-volume monograph was printed by an outstanding doctor and chemist Paracelsus “About Mountain Xerosis and Other Mountain Diseases”, where he specifically described labor conditions, as well as occupational diseases of miners in the result of action of dust, sulfur, mercury vapor and other metals onto human body.

Works of an outstanding German doctor, metallurgist, mineral researcher Agricola (Georg Bauer) cause paricular interest, and especially his work “De re metallica” (1556), which describes description of diseases of miners, stating reasons for their appearance as well as prevention methods.

One of the first books on lead poisoning is the edition of 1556 – a monograph by Stockhausen, which has detailed for that time consideration of pathology, which appear due to inbreathing lead vapor. In 1614, Martin Pans, a local doctor from Anaberg (Saxony) wrote a book on miner’s diseases. However, the first monograph, which had systemized description of issues on work hygiene of various professions and the description of specific diseases of workers of 52 professions, is the “Diseases of Workers” by an Italian doctor B.Ramazzini (1633 – 1714). It was published 25 times in various languages and brought world fame to its author.

Further development of industry in the Western Europe in the 18h and 19^{th} centuries and the beginning of the 20^{th} century was accompanied by the growth of professional diseases. Since 1782, in Germany and then in France, journals started being published, devoted to issues on general and, in particular, occupational hygiene. Articles told about the character of the impact of lead, copper, chromium, arsenic, phosphorus, as well as iodine onto a human body. Rather detailed monographs on dust, lead, phosphorus and other diseases of workers were published.

The founder of Russian science, culture and technology M.Lomonosov developed principles of occupational hygiene and occupational diseases in mineral resource industry. In the treatise “First Principles of Metallurgy or Ore Business” (1763), he provided specific detailed information on hazard impact of production factors onto the health of workers.

The most complete and systemized analysis of occupational diseases was done by A.Nikitin, doctor at Alexander’s Textile Mill in St. Petersburg in his monograph ‘Diseases of Workers and Their Prevention Methods” (1847). A.Nikitin used monograph by B.Ramazzini as the basis for his work, however he significantly changed and completed some chapters. The book played an important role in the development of hygiene and professional pathology.

The end of the 19th and beginning of the 20th are characterized by rapid development of capitalism in Europe. Millions of workers worked at large enterprises; work conditions and lifestyle were very hard. At that time many factory doctors conducted research on hygiene, studied the impact of production factors onto the health condition and diseases of workers. Valuable contribution in studying occupational pathology was made by representatives of rural medicine – F.Yerisman, Ye.Osipov, and O.Pogozhev. An original manual "Occupational Hygiene or Hygiene of Mental and Physical Work" by F.Yerisman, published in 1877, was in big demand. Scale and depth of research is well shown in the 19-volume edition "Materials on Research of Factories and Plants", compiled based on research of 1008 factories and plants. It includes data on the health condition and physical development of 114 thousand workers. This work, completed by F.Yeresman, O.Pogozhev, Ye.Dementiev et. al., had no analogue in the world practice of that time.

Significant contribution into he development of home science on occupational hygiene and occupational pathology was made by D.Nikolsky, V.Levitsky, V.Novrotsky, G.Khlopin, M.Kavalyerov, I.Liatschenko, O.Navakatikyan, G.Yevtushenko, M.Paranko, and A.Shevchenko.

Among outstanding scientists, who had big impact onto the development of professional pathology as a science, it is necessary to mention such scientists as M.Vigdorchyk and S.Kaplun. M.Vigdorchyk published a number of important works on occupational pathology and statistics of professional diseases; he was also a director for Leningrad Institute of Occupational Hygiene and Occupational Diseases. S.Kaplun was an organizer and the first leader of the Central Institute on Labor Protection.

Together with the realization of practical activity in occupational pathology, research and development work was widely implemented as well. In 1923, Ukrainian Institute on Occupational Medicine (now, Kharkiv National Institute on Occupational Hygiene and Diseases) was created in Kharkiv; in 1924, there appeared Moscow Institute on Occupational Diseases named after Obukhov, and in 1925, Central Institute of Labor Protection, and Leningrad Institute of Occupational Hygiene. In the following years, research and development institutes in occupational hygiene and occupational diseases were formed in Kyiv, Donetsk, Dnepropetrovsk, Sverdlovsk, Krivoy Rog, and Karaganda. Almost all the medical institutions on refreshing courses conducted the work in this direction for doctors, as well as departmental institutes.

Establishment and development of industrial toxicology is connected with the names of M.Pravdin, M.Lazarev, G.Shkavera, O.Cherkes, and L.Medved; studies of production microclimate are connected with the names of A.Letavet, G.Shakhbazyan and M.Karnaukh; S.Andreyeva-Galanina, V.Artamonova and G.Balan studied impact of vibration ontot a human body;

and professional pathology in agriculture was studied by Yu.Kundiyev and O.Krasnyuk.

Nowadays, issues on scientific development in the sphere of professional pathology are studied at R&D Institutes on Occupational Hygiene and Diseases. The main institute among them is the institute in Kyiv. Nowadays, four institutes like that function in Kyiv, including one in Kharkiv. As one of their research directions, they all study clinics of occupational diseases. Besides, study of occupational pathology is conducted by professors at chairs of medical institutes and universities, as well as institutes on refreshing courses for doctors. These chairs conduct research either independently (Donetsk Medical University), or together with chairs on Occupational Hygiene (National Medical University), or with chairs on therapy (Dnipropetrovsk Medical Academy, Lviv State Medical University and Kharkiv State Medical University).

Peculiarities of Clinical Examination and Diagnostics of Occupational Diseases

Diagnosing of occupational diseases is a responsible and often a complicated business. People with occupational diseases have a number of social advantages, including pension based on medical list, and payment for medicinal drugs. Clinical picture of the majority of occupational diseases, in particular on early stages, does not differ much from the clinical picture of similar forms of non-occupational diseases.

To consider and solve issues on the presence of occupational diseases, the following documents should be present:

1. Assigning of the medicinal establishment with the purpose of medical examination.
2. Extract from an ambulatory card of the sick person (medical and sanitary part, policlinics and dispensary), put together by the doctor, who attended the patient.
3. Sanitary and hygienic characteristics of labor conditions with the description of specific unfavorable factors of the production environment, their parameters, duration of the contact of the patient with them. In case of combined impact of unfavorable factors, it is necessary to make a detailed description of the production process character. A sanitary doctor of the sanitary and epidemiological station, who carries out state sanitary supervision of the object, where the patient works, puts sanitary and hygienic characteristics of labor conditions together.
4. Extract from a Work Record Book of the patient, which would prove his/her work term at the enterprise, where occupational disease appeared. The extract should be authorized by the Human Resource Department at the enterprise where the patient works.

When diagnosing occupational diseases, especially on early stages, special clinical and functional, as well as biochemical, immunology, radiological and other methods of examination are very important.

Starting with the examination of the patient, first of all, it is necessary to find out what hazard production factors could have or still have unfavorable impact onto his/her health condition in the work process. It is necessary to study thoroughly the documents, the patient brought with him. The assignment for hospitalization should state the reason of hospitalization, and information on the disease progress before the hospitalization.

Particular attention should be paid to the ambulatory health history record of the patient, where the following should be given:

- all diseases, which were previously encountered, including professional ones;
- time of their appearing with the consideration of work under conditions of unfavorable factor, acting of the production environment;
- information on the health state based on results of previous and periodical medical examination;
- clinical picture of the present disease;
- results of the conducted instrumental and laboratory research;
- contents of the conducted treating and its efficiency.

Sanitary and hygienic characteristics of work conditions should include the information on hazard production factors and their intensiveness, results of measured on the containing of toxic matters, dust, noise parameters, and vibration.

When examining patient with occupational diseases, it is necessary to pay close attention to targeted questioning. Patients should have an opportunity to give a detailed narration on hygienic and sanitary work conditions, life conditions, and disease progress. During the examination, it is necessary to keep to requirements of medical deontology, remember about responsibility to the patient, necessity of attentive care about him/her as well as keeping medical secrecy.

Filling in the Medical History Record for a patient with an occupational disease has specific peculiarities. In the history of disease, the following information should be present:

Passport/ID part: Filled in when a patient arrives to the clinic. It should contain the time of the patient coming, his/her last and first names, age, profession, current work place, what medical institution assigned the patient to come to your clinic and what the diagnosis is.

Occupational disease history/anamnesis: It should start with consecutive list of professions of the patient during his/her career until the moment of visiting the doctor or work termination. After a brief chronological listing of main professions, character of the executed work, as

well as the main profession and total work term in unfavorable work conditions.

It is necessary to clarify intentions of the patient regarding continuing work under hazard conditions, as well as where he/she could work after the connection of the disease and work conditions is stated.

Sanitary and hygienic characteristics of labor conditions should include the following: 1) detailed description of work, which was carried out by the patient himself/herself due to peculiarities of the technological process (the so-called “detailed” profession); 2) presence of unfavorable production factors (long uncomfortable body position during the work, noise production, vibration, conduct with dust and toxic matters), category of production; 3) character of individual and collective means of protection implemented at the enterprise (ventilation, sealing-in degree of production processes, utilization of protective uniform, and respirators); 4) working day duration, lunch break, place where lunch is eaten, provision of additional free meals; 5) timeliness of vacations, its duration and its actual utilization, as well as additional vacation and its term; and 6) average salary of the patient.

Occupational anamnesis as well as sanitary and hygienic characteristics, written from the words of the patients, should be completed with the study of sanitary and hygienic characteristics of the work place, which is put together and signed by a doctor on occupational hygiene and is an official document, which confirms the possible impact of specific occupational hazards onto the organism of a worker.

Complaints of the patient: At this stage, questioning the patient should be purposeful. After a list of complaints, each of them should be clarified and worked out in detail. At first, they describe complaints, which refer to the main disease, and then accompanying pathology.

History of the current disease: It is necessary to question the patient in details regarding the beginning and progress of the disease, comparing its development with the work character at the enterprise. It is also necessary to clarify: if a patient was suspended from work in their occupation and for what term; what treatment and/or prevention measures were taken and how effective they were; if the patient went to a resort house to be treated there; if he/she was given disablement certificate (provide the group and character) and what occupation he /she got after then; if the disease had group character (especially, with acute intoxication), if such diseases were encountered before among other workers; if the disease was found during one of the obligatory medical examinations or if the patient addressed to the doctor himself/herself.

It is also necessary to clarify the general anamnesis of the patient: if there were indications of diseases until the beginning of wok under hazardous conditions, if there were periods of exacerbation or complications; what are the results of examination of the patient in other medical institutions.

Life history: It is necessary to find out how the patient developed in his/her childhood, what was the material situation, at what age and where he/she started his/her working activity, and where the military service took place (if applicable); what life conditions are now; what previous diseases he/she had and heredity. For correct evaluation of clinical results, it is necessary to clarify what was the patient's health condition before the work started at the given state of production; if he/she had diseases, which increased the sensitivity of the organism to unfavorable action of production factors.

Results of objective examination of a patient. General examination is carried out purposefully in the order developed by therapeutic clinics. However, examination in occupational pathological hospital has its peculiarities. It is necessary to pay attention to those systems, which are most sensitive to the action of specific hazard factors and make an attempt to specify symptoms and syndromes, characteristic to a corresponding form of an occupational disease. For example, fits of whitening of fingertips can be seen among people who work in the conditions of vibration, indications of hemorrhagic syndrome can be met among patients who work closely with aromatic hydrocarbon, "lead colic" can be among those who are in contact with lead and this compounds.

It is necessary to consider that specific difficulty appears at exposure of initial clinic disease indications, which are not always specific.

In such cases, it is necessary to consider patient's examination results in the dynamics with the utilization of additional examination methods.

Preliminary diagnosis: Grounding the diagnosis starts with the analysis of sanitary and hygienic characteristics of work, which is or was carried out and its comparison with the data of the professional anamnesis, complaints of the patient, disease anamnesis and patient's life anamnesis.

During the consideration of objective data, it is necessary to pay attention to a complex of symptoms or syndromes, which reflect changes of various organs and systems that are the most characteristic for the envisaged professional pathology. Also those symptoms should be assessed, which do not have significant impact onto the condition of the sick person, but are important ("radical") for diagnostics, for example, "lead" and "mercury" edging of gums, or "lead" color.

To prove the diagnosis, it is necessary to prescribe additional clinical, laboratory and instrumental methods to examine the patient.

Data after additional laboratory and instrumental examination methods: Important results are provided by clinical and instrumental methods of research, which are often decisive for early diagnostics of professional disease. Research of functions of external breathing, lung radiography, and if necessary, bronchoscopy in case of occupational disease of respiratory organs (black-lung disease, toxic pneumosclerosis, and dust bronchitis); exposure of

pain (algesimetery), vibration (palestesiometery) and temperature (termometery) sensitivity with vibration diseases, as well as electromyography – with affection of nervous and muscle apparatus – are widely used.

A number of toxicological and clinical-biochemical research has particular specifics. Thus, exposure of the very chemical agent into biological environments, which caused the disease or products of its metabolism, can be used as one of the indications to diagnose an occupational disease (poisoning). When diagnosing such peculiarities of poisoning, as the ability to accumulation, creation of depots, ability to combine actions wit other chemical or physical factors, which lead to the change of clinical picture of intoxication, presence of the hidden period, possibility of the development of diseases in many years after the termination of the contact with occupational hazards (e.g. late silicosis, or cancer of urinary bladder).

When examining a patient, it is necessary to pay particular attention to the increased sensitivity of some chemical and biological factors of the production environment. In such cases, utilization of skin and inhalation sampling with the consideration of the possibility of sensibilization of the organism to these matters in case of their repetitive action (e.g. chromium, Ursol, or products of organic synthesis) is particularly important.

Final diagnosis and its grounding. To make diagnosis of an occupational disease (intoxication), it is necessary to complete thorough analysis of the work activity of the patient throughout his/her whole life. It is necessary to determine work duration under conditions of possible influence of unfavorable factors of the production environment, as well as types of these factors (chemical, physical, biological, or production dust).

It is necessary to have detailed understanding of specific sanitary and hygienic conditions of work at work place, where the patient counters the impact of unfavorable factors.

Examination results are grounded based on the analysis of clinical examination data and general symptomatology of the disease – questioning of the patient, physical methods of examination, laboratory and instrumental methods of research.

Here, those symptoms and syndromes can be distinguished, which are encountered in the clinical picture of the envisaged professional disease in the examination of the patient.

Differential diagnosis of non-occupational diseases is carried out, if they have similar clinical manifestation.

Conclusions regarding the reason of the disease: After the diagnosis has been made, it is necessary to make a conclusion regarding the reason of the disease (occupational or non-occupational).

To determine the ability of the patient to work: if the patient is capable to work or not at the current occupation if keeping to specific conditions;

ability to work is limited – to show what types of occupation can be carried out by the patient; the patient is unable to work and needs specialized assistance.

Besides, it is necessary to provide recommendations regarding rehabilitation, treatment and disease prevention.

Doctor and labor expertise. Social and clinical conclusion is the basis for decision making by Doctor Consulting Commission (DCC) or Medical Social Expert Commission regarding the type of work ability disorder, disorder level and the character of disablement of the given patient.

After the patient leaves hospital, the conclusion is sent to the medical establishment that assigned the patient.

The list of occupational diseases (Appendix 1), approved by the order of the Ministry of Health of Ukraine and the Ministry of Labor of Ukraine, dated February 2, 1995 (No. 23/36/9), is the main document in compliance with which, the diagnosis of the occupational disease is made, as well as the connection with completed work or profession is done, issues on work ability expertise, medical and work rehabilitation, reimbursement by enterprise owners, establishments or organizations or agencies empowered by them to employees for the caused harm when fulfilling labor obligations are carried out.

The list includes occupational diseases, which appear only due to unfavorable production and professional factors (black-lung disease, vibration disease, intoxication, etc), as well as a number of diseases, the development of which is connected with the influence of specific unfavorable production and professional factor and only a clear influence of other non-occupational factors, which cause similar changes in the organism (bronchitis, allergies, cataract, etc).

It is necessary to remember that in the corresponding lines of the given document only an approximate list of enterprises, manufacturing enterprises and work done there, as well as etiological factors, which can cause disease, is provided there. Occupational diseases also include closest and distant consequences of occupational diseases (e.g. stable organic changes in the central nervous system after intoxication by carbon oxide). It is necessary to consider the possibility of the development of occupational diseases in long term after termination of contact with hazard factors (late: silicosis, papilloma of urinary bladder, etc). Occupational diseases can also include such diseases, which developed based on the occupational disease (e.g. lung cancer, which appeared with the patient with black-lung disease or dust bronchitis, and should be considered as an occupational disease, which is proved by histological changes in the mucus tunic of bronchi – diffused metaplasia with elements of displasia and development of epidermoid cancer, as a rule).

If occupational disease causes worsening of the development of non-occupational disease, what led to the loss of ability to work, it can bee

considered occupational (e.g. progressing of a form of arterial hypertension, which appeared on the background of vibration disease).

Diagnostics of acute poisoning include:

- clinical diagnostics based in the given anamnesis, results on examination of the event place and study of clinical picture of the disease to establish specific poisoning symptoms;
- laboratory toxicological diagnostics, quality and quantity definition of toxic matters in biological environment of the organism (in blood, urine, or cerebrospinal fluid);
- pathomorphological diagnostics, definition of specific posthumous poisoning indications.

Diagnosis of acute occupational disease (intoxication) can be stated by a physician of any medical establishment after obligatory consultation with a specialist on occupational diseases and a physician on occupational hygiene of a territorial sanitary and epidemiological station (SES). Acute occupational disease (intoxication) appears suddenly, after one time impact of relatively high concentration chemical matters (for not more than one shift), which is in the air of the work zone, or levels or dozes of other unfavorable factors. The connection of acute infectious diseases with the occupational activity of the victim in case of necessity to clarify specialized departments of hospitals, clinics of scientific and research institutes on occupational hygiene and occupational diseases after the obligatory consultation with physicians on occupational hygiene and epidemiology of the SES. Professional etiology of acute contact dermatitis can be established by a doctor of skin and venereal dispensary on the agreement with a territorial SES.

Diagnosis of chronic occupational disease (or intoxication) have the right to state first of all specialized treatment and prevention establishments of Ukraine: Donetsky R&D Center for Occupational Hygiene and Prevention of Traumatism, Institute of Medicine of Labor of the Academy of Sciences of Ukraine (Kyiv), Kryvorizky R&D Institute of Occupational Hygiene and Diseases, Kharkiv R&D Institute of Occupational Hygiene Institute on Medical Radiology, Ukrainian R&D Institute of Ecohygiene and Toxicology of Chemical Matters (in case of utilization of means of protection of agricultural plants), Donetsk Regional Specialized Clinical hospital on occupational diseases, Department on Occupational pathology of Lviv Regional Hospital, Department on Occupational Pathology of Cherkassy Regional Hospital, as well as the clinic of the Institute of Health named after Medved. Diagnosis of chronic occupational diseases should include its name, main clinical syndromes of affections, degree of affected organ function disorder.

Research and registration of professional diseases should be carried out based on the “Regulations on Research and Registration of Accidents, Occupational Diseases and Damages at Enterprises, Establishments and

Organizations", approved by the Cabinet of Ministers of Ukraine, No. 623, dated August 10, 1993.

In compliance with these documents, all first exposed chronic occupational diseases and poisoning are subjects to investigation and research. A notification should be compiled on each victim of an occupational disease by clinics of research R&D institutes on occupational hygiene and diseases, specialized departments of regional (city) hospitals, which have the right to establish final diagnosis of occupational disease.

During three days after the final diagnosis of an occupational disease is made, a notification should be sent to the enterprise where the patient works, as well as to sanitary and epidemiological station and medical establishment where the enterprise is serviced.

An owner of the enterprise should organize investigation of reasons on each cease of the occupational disease during seven days since the moment of reception of a notification of occupational disease. Investigation is carried out by a commission, which is appointed by the decision of the leader of the sanitary and epidemiological station. It should include: an officer of the sanitary and epidemiological station (head for the Commission), representatives of a trade union, a work collectives, medical establishment, as well as a specialist on occupational pathology of the local agency of the Department of Health and the owner of the enterprise. Representatives of the Ministry and other central governmental agencies, to the sphere of which the enterprise belongs, local agencies on public supervision of labor protection of and state executive powers, as well as specialists of R&D establishments and educational establishments of the Ministry of Health of Ukraine can participate in the investigation.

An owner of the enterprise should provide the investigation committee with the data on laboratory research of hazard factors of the production process with instrumental measuring of their meaning, necessary documentation on the process (technological requirements, regulations and normative acts regarding the safety), to provide the commission with the facilities, transportation and communication means, to organize publishing and copying of materials to be disseminated.

The investigation commission should compile a program on investigation of circumstances and reasons of an occupational disease, put together an investigation act with suggested activities to prevent the development of an occupational disease, provide normalization of labor conditions, as well as determine the responsibility of the enterprise and officials for the occupational disease appearing and development. The investigation commission should evaluate hygienic conditions of labor of the patient based on materials of the early conducted attestation of work places, results on examination and research, and if necessary laboratory research of hazard production factors with documentary measuring of their meaning. It

should analyze present medical documentation: ambulatory cards, disease history, conclusions of establishments, orders of agencies on federal supervision on the labor protection, etc. It has the right to receive written explanations from officials and workers on issues connected with the investigation of occupational diseases. The investigation act on occupational diseases is compiled by the commission in five copies within three days after the termination of the investigation, which are sent to the patient, enterprise, where the occupational disease took place, medical establishment, which services the enterprise and the trade union, the member of which the patient is. One copy of the act stays at the sanitary and epidemiological station to analyze and control the intended measures. Investigation acts are kept at the SES for 45 years, in the rest of organizations they are kept for two years.

Based on the results of investigation of appearing of occupational diseases, an owner of an enterprise should make an order with measures on prevention of occupational diseases whose fault was in violation of sanitary norms and rules, which caused occupational diseases. When realizing measures on prevention of occupational diseases, suggested by the investigation commission, an enterprise owner should inform the SES within the terms stated in the act.

Registration and records of people, who were among the first to have occupational diseases, are conducted in specialized record books in compliance with the form, approved by the Ministry of Health, which should be filled out:

- at enterprises and establishments on sanitary and epidemiological service based on notifications about occupational diseases and acts of their investigation;
- at medical institutions based on the medical record of a patient, extracts from the medical history, doctor’s conclusion on the diagnosis after examination at the hospital, as well as notification about occupational diseases.

Using the data of disseminated acts on occupational diseases, the SES compiles special record cards for keeping and analyzing occupational diseases with the assistance of computers. These compiled cards should be saved for 45 years in the SES.

In compliance with the documents, investigation is carried out on every acute occupation disease and poisoning. A witness, employer, who found out about each case like this, or the victim him/herself should notify the work coordinator, foreman or any other authority of the enterprise and provide first aid medical assistance. In his/her turn, the leader should do the following: organize medical aid to the victim immediately and deliver him/her to a medical establishment, also notify the enterprise owner about the accident; keep the original state of the work place and equipment until the investigation commission arrives, also take measures to prevent similar cases.

A medical establishment should notify the leader of the company about each accident like this, which was registered at the company where the victim works within one day, and in case of acute occupational poisoning (disease), they also should notify a sanitary-hygienic station, by sending urgent notification on each victim. The same type of notifications is sent to the owner of the company/enterprise to take urgent measures to prevent further similar accidents. A medical establishment, which clarified or changed the diagnosis of an acute occupational poisoning (disease), compiles an urgent notification and sends it to the sanitary and hygienic station which coordinates the enterprise the victim works at with changed (clarified) diagnosis, as well as its date, within 10 hours.

Upon receiving the notification about the accident, the company owner appoints a commission on investigation, which includes a head (specialist) of the labor protection service of the company (chairman of the commission), head for structural subsection or chief specialist. The commission should also include a representative of a trade union, member of which the victim is, and in case of acute occupational poisoning (diseases) – a specialist of the SES. If the victim is not a trade union member, then an authorized representative of a work team on labor protection should be in the structure of the commission.

Within three days since the moment of the event, the investigation commission is obliged to do the following:

- to examine the accident place, interrogate witnesses and those involved in the accident, and to receive explanations from the victim, if applicable;
- to consider the correspondence of labor conditions and production means on the project and passports, and also keeping to requirements of normative and technical documentation on operation of equipment and normative acts on labor protection;
- to find conditions and reasons of the accident; to determine responsible for the accident; and also to develop measures to prevent cases like this;
- to fill in an act, form H-1, in five copies, where fault for the accident of an establishment, victim or a third party should be stated; the copies should be sent to be approved by an enterprise owner.

Within a day after the end of the investigation, an owner should confirm all five copies of the act, form H-1. The act is sent to a victim or to a person who represents his/her interests, a foreman or another structural subsection, where the accident took place, to implement measures regarding prevention of similar accidents, to the state inspector on labor protection, trade unions of the enterprise where the accident took place, the head of the sector on labor protection, to who the act is sent together with other materials on investigation. A copy of an act, form H-1, in case of acute occupation poisoning (disease) is to be sent to the SES as well.

The SES fills in Records on Occupational Poisoning: (Disease) for recording and analysis based on the act, form H-1.

Organization and Conduct of Medical Inspection of Workers

Medical assistance to patients with some occupational disease is provided by clinics, parts of R&D Institutes, as well as medical and sanitary sectors (MSS). The main task of the MSS is to develop and conduct measures, targeted at improvement of labor conditions and life of the employees and officers, prevention and decrease of total and professional disease, provision of specialized medical assistance and realization of systematic dispensary care together with the administration of establishments.

As until now, amount a list of doctors, there has been no physician on occupational diseases, all the work on servicing the patients with occupational diseases were carried out by general practitioners (workshop general practitioner). Nowadays, when such speciality became officially known, occupational pathological service is at the stage of development.

To prevent occupational diseases, preliminary and periodical medical examinations of workers to start their career or those who work in close contact with hazard factors of the production environment. The order of their conduct is regulated by the Order of the Ministry of Health of Ukraine No. 45 dated of March 31, 1994 “About Approving Regulations on the Order of Conduct of Medical Examinations of Workers of Specific Categories”. The order envisages that the organization and medical examinations will be provided by:

- an owner of an enterprise, establishment, organization independently of pattern of ownership and types of activity;
- establishments and agencies of the Ministry of Health of Ukraine, treatment and prevention, sanitary and hygienic, R&D and medical institutions (universities), on the territory where enterprises, establishments, organizations, agricultural companies, agrofirms, rental, cooperative, small, joint venture companies; catering, industrial, and children’s sites, elementary schools and other objects.

The owner of the enterprise finances medical examinations, reimburses losses on care, occupational and medical rehabilitation of people with occupational diseases, examination of specific labor conditions to put together a sanitary and hygienic characteristics.

Preliminary medical examinations are dune with the purpose to state physical and psychological ability of a person to work in the specific area, speciality, position, when first admitted to work; as well as to prevent diseases and accidents, exposure of diseases (infectious and others), which threaten with contagion of workers and manufactured products, as well as admission to work for people under 21.

Periodical medical examinations are conducted in the process of labor activity for people, who are engaged in hard work, work with hazard or unsafe conditions in compliance with the list of matters, unfavorable production factors and works to implement which, obligatory medical examinations of employees should be carried out. They provide dynamic examination of the health state of workers, expose early signs of the impact of production conditions and hazards onto the body, and which do not give the opportunity to continue working on the given profession: prevent accidents, expansion of infectious and parasitic diseases. They can be conducted within the period of staying of an employee in the hospital or in cases when he/she asked for help.

Results of medical examinations should be drawn up within a month in a relevant act, which is made in four copies (for a medical establishment, owner, trade union committee and sanitary and hygienic station).

During the medical examination, examination in occupation pathologic centers, clinics of R&D and medical institutes (universities) to specify the diagnosis or to determine a role of production factors in the development of diseases, a job and average salary is kept for a worker.

The order of organization of medical examinations: The enterprise shall:

- determine a contingent of people, subject to periodical medical examination; and draw up a list of them in two copies with last and first names, and then approve it at the sanitary and hygienic station. One copy is sent to the medical establishment, and the other stays at the enterprise (with the organization responsible for medical examination) together with the sanitary and hygienic station and a trade union;
- assign people, who are sent to the enterprise or to change profession and work place to take preliminary medical examination in compliance with a proper document;
- introduce a new-comer to the hazard and dangerous production factors and matters, as well as normative acts on labor protection, which are specific to the profession;
- finance medical examination, and reimburse costs on examination and treatment of workers at occupational pathologic institutes, medical institutes (universities); as well as examination of work conditions with developing sanitary and hygienic characteristics;
- make an order to conduct medical examinations within the terms, approved by the treatment and prevention establishments, appoints responsible for organization of medical examination;
- assist the creation of improvement of material and technical bases of medical sectors, and medical establishments to conduct medical examinations, clinical and other research;
- apportion premises to conduct medical examinations;

- assign workers to have medical examination in medical establishments and controls the term of its implementation;
- provide implementation of the recommended rehabilitation and prevention measures.

A medical establishment shall:

- make an annual order to create commission to conduct medical examinations with a fixed term, place of conduct and list of physicians, clinical and other research; the commission will be chaired by a vice chief physician of the medical establishment, which trains doctors in occupational pathology;
- develop a plan – schedule of medical examination of workers, which should be approved by an owner and sanitary and hygienic station;
- conduct medical examination of employees, as well as clinical and other types of research;
- involve other specialists to participate in the medical examination; conduct additional clinic research, necessary to assess the health state of employees if applicable;
- inquire sanitary and hygienic characteristics of labor conditions of employees at sanitary and hygienic stations;
- control keeping to the terms of medical examination;
- make conclusions on the health state of each employee, who underwent a medial examination; make a decision regarding medical contraindications as to the possibility to continue work on the profession for those who have general or occupational diseases;
- inform an employee as to the state of his/her health, as well as the possibility to continue work on the profession based on the results of medical examinations or provide conclusions regarding transfer to another job;
- based on the medical indications, send an employee to further examination at medical centers, which have the right to make a diagnosis of an occupational disease;
- assign an employee to meet medical and social expertise commission (MSEC) based on medical indications.

It conducts:

- annual medical examination of people who terminated their work at the enterprise with hazard and dangerous factors, the impact of which can cause late development of occupational diseases;
- analysis and generalizing results on medical examinations, drawing up a final act, which is sent to the territorial SES, owner and a trade union of the enterprise;

- sending out a list with names of those, who are contraindicated to work under unfavorable conditions to an owner within a month after the medical examination took place;
- carrying out dispensary supervision over patients with occupational diseases, who continue their work activity, treatment and occupational rehabilitation;
- keeping records of patients with occupational diseases and poisoning.

A sanitary and hygienic establishment shall:

- determine authenticity of the record keeping made by the owner of hazard and dangerous factors and matters, work with which require medical examinations;
- confirm lists with names of people, subject to medical examination, as well as plans – schedules of medical examinations.

It participates in the following:

- in preparing and training specialists of a medical center; and
- in compiling the final act on periodic medical examination.
- in expert evaluation of the organization and the quality of medical examinations.

It sends decisions as to elimination of exposed violations and drawbacks in the organization and conduct of medical examinations;

- considers issues on temporary termination of medical examination in case of isolation of the existing situation;
- compiles sanitary and hygienic characteristics on work conditions;
- applies with proposals as to prevention of occupational diseases to territorial state administrations.

Research and Development (R&D) institutes on labor medicine, labor hygiene and occupational diseases, as well as chairs of medical universities (institutes) shall:

- develop normative and methodical documents on scientific and organizational principles of conduct of medical examinations, expertise of their quality and evaluation of results; criteria of determination of contingent of people – subjects to medical examinations; indications of the risk of occupational disease development and criteria of determining diseases as occupational ones;
- are engaged in elaborating issues on prevention, early diagnostics and treatment of occupational diseases, medical rehabilitation of workers with the risk of development of occupational diseases and patients with occupational diseases; definition of distant consequences of the impact of hazard and dangerous production agents onto the health. They also carry out

refreshening and upgrading courses on occupational diseases in the form of provision of information, training and seminars, and also make final decision as to the connection of the disease with work conditions.

Chapter 2

PROFESSIONAL DISEASES OF RESPIRATORY ORGANS, CONDITIONED BY DUST IMPACT

Black-lung disease

Black-lung disease is a respiratory disorder, a type of pneumoconiosis caused by repeated inhalation of coal dust over a period of years and dust depositing in lungs, and is also characterized by the development of diffusion fibrosis.

Classification of dust diseases of lungs changed many times during a long period of time. But at first, dust diseases of lungs were united under the common name “consumption” with giving some specific occupation of the patient. Later, specific forms of the disease were distinguished, like “byssinosis” or “dust eczema”. Then, the term “pneumoconiosis” was introduced (from Greek: “lung” and “dust”), which generalized all forms of dust diseases, which were accompanied by the development of fibrosis process in lungs. For a long time, there was not unified thought regarding the possibility of the development of pneumoconiosis process without inhaling production dust of silicon dioxide, i.e. considered that silicon dioxide is actually a synonym of silicosis. There was not any unified thought as to the existence of dust bronchitis as it is.

Based on modern clinical, radiological and pathological and anatomic data, the dust disease was considered in a wider aspect, what was reflected in its classification as well. Clinical forms of dust diseases are disseminated processes in lungs - silicon dioxide, granulomatoses, exogenic allergy, alveoli disease, which appear under the impact of corresponding types of dust, dust bronchitis, and bronchial asthma.

After introduction of radiological method of lung examination into clinical practice, there was an opportunity for more differentiated approach to determine the presence and character of lung dissemination when the patient is still alive. Thus, particular attention was paid to the development of classification of pneumoconioses as a form of dust diseases of lungs, which are the most difficult to be diagnosed.

As to the evolution of views at the classification of pneumoconiosis, it is necessary to mention that their first classification was adopted in 1930 at the International Conference on Silicosis problems. Then, three stages of silicosis were determined based on mostly radiological picture. In the future, this classification was reconsidered many times (1950, 1958, 1968, etc), it was specified and changed, what let characterize other types of pneumoconiosis, variety of radiological expression of diseases, as well as the presence and character of indications of complications and some accompanying lung diseases. Various radiological and clinical indications in

classifications were coded and marked by symbols. The last variant of the International Classification of pneumoconiosis was approved in 1971.

Home classification of pneumoconiosis, in contrast to international ones, reflected not only radiological indications of the disease, but also the clinical picture, as well as the degree of compensation process. According to the first classification, developed by I.Kavalerov in 1925, the following clinical variants of pneumoconiosis were specified as bronchitis, emphysematous, pleurotic, interstitial cystitis and cardiogenic ones.

So, according to the modern classification (2002) the following types of pneumoconiosis are distinguished:

1. Silicosis

2. Silicatosis

3. Metalloconiosis

4. Carboconiosis

5. Hypersensitivity pneumonitis (dust diseases)

Taking account of dust aggresivity three groups of dust diseases are distinguished:

I. Dust diseases developing from a dust with high- and medium-fibrogenious influence (containing SiO_2 or asbest more than 10%) - a silicosis, silicoantracosis, silicosilicatosis, silicosiderosis, asbestosis.

II. Dust diseases developing from a dust with mild-fibrogenious influence (with the contents free silicon dioxid less then 10%, bonded SiO_2 or without SiO_2) – silicosilicatosis (kaolinosis, talcosis etc.); carboconiosis (anthracosis, grafitosis, pneumoconiosis of coal dust, diamond pneumoconiosis); metalloconiosis (siderosis, kaolinosis, baritosis, manganoconiosis, aluminosis etc.); a dust disease of the electric welders, gascarvers and working other welding professions, polishing, grinding, foundring etc.

III. Hypersensitivity pneumonitis (dust diseases) from a dust (aerosolum) with toxico-allergic influence - berylliosis, pneumonites from influence of chrom, nickel, platinum and others rare-earth metals and alloys; pneumonites from a dust of plastic, polymeric pitches, medicinal preparations (toxico-fibrogenious alveolites - TFA); a byssinosis, bagassosis, papricosis, exogenous allergic alveolitis (EAA) woodworkers, poultrers etc.

The classification also includes indications, which characterize various variants of clinical and radiological picture, functional disorders, complications of pneumoconiosis progress (Table 1 and 2).

Clinical and radiological characteristics of pneumoconiosis are very multi-sided and depend not only on the type of dust, which caused pneumoconiosis. Thus, when making a diagnosis of pneumoconiosis, it is not important only to state the etiological factor – from dust. It is very important for treatment and solution of questions of work ability of patients with pneumoconiosis to know the evidence, form, and speed of progressing of fibrous process in lungs, presence of respiratory compromise and cardiac

decompensation, which accompany the main disease. Based on this, the classification of pneumoconiosis, the following clinical and radiological indications were included, which characterize morphological, functional and clinical peculiarities of various forms of pneumoconiosis.

Radiological characteristics of main indications of coniotic fibrosis of lungs include the following elements: character of shadow (form, outline and size), their expansion, thickness and density. Each radiological indication is coded by a corresponding symbol. Codes of radiological indications are marked with letters from Latin alphabet and Arabic numerals, and stages of pneumoconiosis are marked with Roman numerals (Refer to Table 1).

Table 1

Radiological Characteristics of Pneumoconiosis

Code	Characteristics of shadows (form and size)	Spreading, density and territory of shadows	Stage
	Absence of pneumoconiosis Control		0
p	Small nodular (small rounded shadows)	Double	0 – 1
g	Nodules, up to 1.5 mm in size	Small number of shadows	I, II
r	Nodules, from 1.5 to 3.0 mm	Moderate number of shadows	
	Nodules, from 3 to 10 mm	Numerous shadows	
	Interstitial (small shadows of irregular form)	Double, diffusive	I, II
s	Linearly and cellulary changes	Blurry outlines	
t	Pulled changes	Clear outlines (lung picture is clear)	
u	Sharply expressed pulled changes	Numerous shadows (lung picture is not defined)	III
	Big nodular (big rounded shadows on nodular or interstitial background)	Double or one-sided	
A	Small nodular – diameter of nodules from 1 to 5 cm	Territory of spreading is not more than 5 cm^2	
B	Big nodular – diameter of nodules from 5 to 10 cm	Territory of spreading is not more than 1/3 of the lung field	
C	Massive – diameters of nodules is over 10 cm	Territory is over 1/3 of the lung	

Absence of pneumoconiosis indications on an X-ray photograph is marked with zero (0). If there are some doubts as to the change of lung picture in the way of its some intensification, then when describing the X-ray photograph, it is marked “0-1”, what actually means suspicion of pneumoconiosis presence. To specify the diagnosis, it is necessary to conduct additional research: increased side photographs, tomograms, as well a repeated radiological examination in 6 to 12 months.

Table 2

Clinical Characteristics of Pneumoconiosis

Clinical and functional characteristics	Disease progress	Complications
Bronchitis	Fast progressing	Tuberculosis: a) with separating forms of tuberculosis (according to the classification) b) without noting the form of TB (small nodular, big nodular and massive TB-silicon) Pneumonia Brochnoectatic disease Bronchial asthma Pneumothorax Atrophic arthritis Neoplasm
Bronchiolitis	Slowly progressing	
Lung emphysema, stage I, II and III	Regressing	
Lung failure, stage I, II, and III		
Cor pulmonale, compensated, decompensated, stage I, II and III		

As to the character of forms, sizes and outlines of shadows on the radiograms, there are the following fibroses: interstitial, small nodular and big nodular ones.

Interstitial fibrosis on an X-ray photograph is characterized by small change of a lung picture in the form of its intensification and deformation due to the development of perivascular and peribronchial fibrosis, as well as fibrosis of interalveoalr septum and interlobular partition.

Depending on the stage of reflection and localization of fibrosis shadows: linear and cellular (s), heavy (t) and roughly heavy (u).

Interstitial fibrosis of lungs is usually double sided and diffusive. As to the thickness and density of shadows on X-ray photographs can be not much spread (1); much spread, when the picture does not differentiate and there are numerous shadows of irregular forms.

Nodular fibrosis on an X-ray photograph is shown as small rounded shadows, which are conditioned by coniotic nodules. As to the size of nodules, they can be divided into three groups (up to 1.5 mm (p), from 1.5 to 3 mm (q), and from 3 to 10 mm (r). Nodular shadows are rounded with clear outlines; their intensiveness depends on dust, which caused pneumoconiosis. Nodular process is double sided, as a rule. As to the number of nodules, there are three categories: small (1), moderate (2), and numerous (3).

Big nodular pneumoconiosis is characterized by the presence of big shadows of rounded or irregular form with clear or unclear outlines on the background of nodular or interstitial shadows on X-ray photographs.

As to the diameter of nodular shadows and the territory, they cover there are A-small nodular variant when nodule diameter is from 1 to 5 cm, with the total territory of spreading – not more than 5 cm^2, B – big nodular

process, when the diameter of nodules is from 5 to 10 cm, with the total territory of spreading – not more than 1/3 of the lung territory; and C – massive pneumoconiosis, diameter of nodules is over 10 cm with the total territory of spreading – over 1/3 of lung territory.

In compliance with home classification, there are three stages of pneumoconiosis – I, II and III. Main criteria to determine stages, are X-ray indications, though clinical and functional indications are considered as well.

As to the character of pneumoconiosis process, there are the following forms: 1) fast progressing; 2) slowly progressing; 3) late; and 4) "regressing".

With fast progressing pneumoconiosis, stage I, the disease can be diagnosed in 3 to 5 years after starting work with dust, and the acceleration of pneumoconiosis process, i.e. transfer from pneumoconiosis, stage I to stage II, can be seen in 2 to 3 years. This form of pneumoconiosis can include the so-called acute silicosis, which is a fast progressing form of silicosis as it is.

Slowly progressing pneumoconiosis develops in 10 to 15 years after the beginning of work in contact with dust, and with transfer from stage I to stage II, the disease can last for not less than 5 to 10 years.

Pneumoconiosis, which develops in several years after the termination of contact with dust, is called late. Regressing forms of pneumoconiosis can be met only when X-ray contrast dust is accumulated in lungs, which created an impression of more intensified stage of fibrosis in lungs. In case the patient terminates his/her contact with dust, there is partial withdrawal of x-ray contrast dust from lungs. This explains the "regress" of pneumoconiosis process.

Clinical and functional indicators of pneumoconiosis include TB, pneumonia, bronchectatic disease, bronchial asthma, atrophic arthritis, and spontaneous pneumothorax (refer to Table 2).

Silicosis

Silicosis is pneumoconiosis, caused by inhaling dust with free silicon dioxide (SiO_2). This is the most spread form of pneumoconiosis, the progress of which is particularly complicated. The disease got much spreading in the end of the 19^{th} century, mostly due to the development of metal mining industry and machine engineering, where in the process of production, dust is created, which include free silicon dioxide.

Silicosis can be most often met in the following areas:

1) in metal mining industry – among people, who are engaged in mining gold, tin, copper, tungsten and other minerals, which is in the ores with quartz (drill-operators, tunnellers and workers of tunneling teams); in machine engineering among the workers of foundries (fettlers, shakers, etc);
2) on the production of fireproof and ceramic materials – among workers, engaged in production of dinas, fire clay and other fireproof products,

as well as repair of industrial furnaces and other operations in metallurgic industry.

3) among workers engaged in tunnel boring, sand grinding, processing and treatment of quartz, granite and other ores, which contain free silicon dioxide.

Changes in lungs due to action of dust take place, as a rule, after a long period of work there. Disease development depends on the amount of dust, which got into the organism content of free silicon dioxide, as well as tendency to the disease. The last thing is very important considering the fact that not all the people who inhale quartz dust will have silicosis provided common work conditions.

The boundary permitted concentration of dust, which contains over 70 % if free silicon dioxide, is 1 mg/m^3, from 10 to 70 % - 2 mg/m^3 and from 20 to 10 % - 4 mg/m^2.

Many research showed that the most pathogenic are the particles of dust with the size less than 5 micrometer as they achieve bronchiole or alveoli, and stay there.

In the development of silicosis, the importance is not only in getting dust into lungs, but also its retention there. At the preliminary stage of fibrosis process, in the result of the fact that the amount of dust, retained in lungs, increases the amount, which can be take out, what creates the so-called “dust depots”. Cleaning of lungs from silica is taken out mostly via bronchitis, but a smaller amount of particles is taken out via lymphatic nodules.

In average, the period from the beginning of action of dust until the development of the disease takes from 10 to 15 years, though under unfavorable conditions of work it can reduce to 2 to 6 years, thus we have the so-called fast progressing silicosis.

This form of silicosis is characterized by a fast progress and rather unfavorable forecast. As a rule, it develops among workers, who work in the most silicosis dangerous professions (tunnellers, polishers of lenses, and before these were sandblasters). They thing that the fast progressing, especially “acute”, silicosis with short period of dust exposition (up to six months) develops in case of the action of highly aggressive dust onto young people of asthenic constitution with clear inflammatory process in lungs.

Pathogenesis. Mechanism of appearing and development of silicosis is very complicated and is not completely opened. If to consider this question in historical aspect, then first of all, it is necessary to tell about the so-called mechanical theory of appearing and development of fibrosis process in lungs. From the point of view of followers of this theory, fibrosis changes in lungs are the result of mechanical irritation, microtraumatizing of lung tissue. Soon it was rejected, though even today, mechanical factor is considered valuable in the development of dust pneumosxlerosis.

Mechanical theory was later replaced by toxic–chemical theory, which explained the development of silicotic fibrosis by toxic action onto the lung tissue of silicic acid, which appears when gradually dissolving silica.

Further, many hypotheses were suggested as to the mechanism of appearing and development of fibrosis (infectious and piezoelectric), which did not received the necessary support and thus were rejected.

And nowadays, the following two theories are most known: colloid-adsorbing and immunological theories.

The essence of colloid-adsorbing theory is in the recognition of the role of silanol groups of the surface of silica in primary interaction with cellular elements. Thus, pneumoconiosis from the view of the theory followers is considered as chronic disease of lungs, caused by long inhaling of air with high concentration of practically non-dissolved aerosols, which have pathological impact in hard accumulated states based on processes, which take place on the surface of dust particles.

Mechanic destruction of silica is obligatory connected with breaking of many oxygen bridges between oxygen atoms; as in silicon dioxide, four atoms of oxygen surround silicon atoms, and each oxygen atom belongs to two silicon atoms simultaneously. Thus, silica is a continuous link of silica-oxygen tetrahedrons $(SiO_4)^{-4}$.

Thus, with breaking oxygen bridges on the surface of breaking, there are two types of active centers: one of them is unsaturated oxygen atom of a broken pair; the other one is an unsaturated atom of silicon. In the air, under the impact of water vapors, kept in them, and particularly fast in water environment, aquation on the silica surface takes place. Unsaturated oxygen atom is joined with ion of hydrogen, turning it into a silanol group.

An unsaturated atom of silicon attracts hydroxyl group $(=Si-)^{+}(-O-H)^{-}$. Besides, on the surface of silica break, there is also the third type of active center. This is an oxygen atom of non-broken marginal siloxane bridge.

Researches show that particular cytotoxicity of silica is conditioned by all active centers of the surface of dust particles, capable to create hydrogen links, but silanol groups have the most important meaning. The proof of the important role of these groups in the considered process, there is immediate depressed biological activity of silica at their substitution with inert methyl radicals.

Influence of silica and other fibrous dust particles onto a human organism is done in three stages. The primary and obligatory link in the total chain of pathological changes, observed in lungs when inhaling with fibrous dust, is extensive activity and damage of macrophages with ingestion dust particles. At the second stage, vital products of activated macrophages and matters, which are emanated at the destruction, stimulate fibroplasts and extensive synthesis of collagen in respiratory organs.

Besides, it is necessary to remember that the interaction of dust particles with phagocytes creates free radicals of oxygen, which also cause destruction of lysosomal ferments of phagocytes.

Making conclusions, it is obvious that the primary action of fibrous dust, has significant impact onto the character of pathological developing changes is grounded by on the one hand, mechanism of interaction if the surface of dust particle with the membrane of phagocyte, and on the other hand, by the peculiarity of activation of its oxidase metabolism, and besides, by the ability of the marginal layer of particles to catalyze reactions of transformation of active forms of oxygen.

Immune theory can be well connected with the colloidal–adsorption theory. Significant place in the mechanism of a corresponding reaction onto the dust action is in the interaction: antigen – antibody. Certainly, an antigen with silicosis is a structurally changed protein of coniophagus cytoplasm. After the destruction of coniophagus, particles go beyond the cell environment, and thus the release of structurally changed protein takes place, which is capable to impact the human organism as an autoantigen. Released particles again go through phagocytosis with further destruction of coniophagus, what conditions continuous creation of autoantigens, which stimulate the antibody production. There are also ideas that with the destruction of macrophage dust particles in the result of phagocytosis, liposaccharide complexes are released, which are capable to activate the creation of antibodies non-specifically.

Thus, silicosis can be considered as a disease of non-specific immunogenesis, where phagocytosis and the destruction of coniophagus is an obligatory condition of the development of silicotic reaction.

There is a supposition that lipids or products of their re-oxidation, which are released from destructed dust cells, possess fibrous activity themselves. In the fibroblasts under their impact, oxidation of amino acid of praline into oxiproline takes place, which is considered an important link in the pathological collagen creation.

Lately, works appeared, where the role of fibronectin in the pathogenesis of silicosis is considered. It was shown that fibrogenic dust stimulates synthesis and release of fibronectine by leukocyte and lung macrophages, securing their aggregation and adhesion when forming silicotic granulomas. Besides, in the pathogenesis of silicosis, an important role is played by biologically active matters of tissue basophiles. Based on this position, silicosis is a particular variety of the inflammation – chronic granulomatous inflammation.

Pathological and anatomic picture: With silicosis, changes take place not only in lungs, but also in upper respiratory ways, bronchi, pleura, lymphatic glands and lung vessels.

In the mucous tunic of scroll-bones, larynx and trachea, subtrophic, and later atrophic and sclerotic changes can be seen. The mucus tunic of larynx and trachea are thickened and plethoric, and in the submucuous space – hyperplasia of glands with the expansion of their outgoing flows, which are full with mucus and exfoliated epithelium.

At the early stages of silicosis, there are areas in lungs with deposits of dust particles, mostly around bronchi, vessels, and in lumens of alveolar ducts and in alveoli. In these areas there are precolagen and collagen fibers, which are a primary stage of forming of silicotic nodules. Together with it, there is a weakly outlined sclerosis in the form of outgrowing of conjunctive tissue around bronchi and vessels, as well as in alveolar septum.

Diffusive – sclerotic (interstitial) form of silicosis is characterized, first of all, with the presence of numerous bars of collagen tissues and marked sclerosis around bronchi, vessels and in interalveolar septum. Often there is marked emphysema of lungs. Silicotic nodules are absent, but with time in those areas, real silicotic nodules can appear.

Nodular form of silicosis can be met much more often and is characterized with the presence of concentrically located, practically hyalinized fascicles of conjunctive tissue, which look like silicotic nodules. As a rule, they are round or oval, of grey or grey – black color. Silicotic nodules are mostly placed in alveoli, and can be met in peribronchial and perivascular lymphoid nodules.

Clinical picture. Patients with silicosis mostly complain on three things: pain in chest, dyspnea and cough.

Complaints on pricking in the chest, mostly in scapula area and underneath them have non-permanent status on early stages of silicosis. Clinical character of pain proves their pleural origin, microtraumatizaton of pleura and the development of conjunctive tissue reaction on deposited sand in them. Along the development of silicotic process, commissures of interlocar and visceral pleura, as well as sclerosis of subpleural tissue, which disturb free smoothing of lung tissue and conditions the feeling of pain, are created between them.

Dyspnea is considered one of the main symptoms of silicosis. Complaints on it appear already on the early stage of silicosis, at first during the work, what proves secret insufficient breathing. Only at the later stage of the disease, patients have dyspnea even at insignificant physical activity, but it is rarely observed in rest (complicated form of silicosis). Dyspnea is conditioned by many mechanisms: spasms of small bronchi and bronchioles; afterwards, the increase of dyspnea can be explained by progressing fibrosis and emphysema, which limit breathing surface of lungs together with cardio-vascular collapse.

Dry cough or with small mount of mucoid viscous sputum starts causing problems to patients with silicosis on the early stage of the disease as

well. In lungs, disseminated crepitation, sometimes, fine moist or subcrepitated rales on a lower part of lungs can be heard on the background of vesicular or heavy breathing. These auscultated data correspond to the appearing of primary cataract reaction on dust in the bronchial tree, which can expand from trachea to the smallest bronchi and bronchioles.

Then (second and third stages of the disease), dry rales can be ausculated or not.

With the development of process, catarrhal reaction is replaced by atrophic and degenerated changes, which are accompanied by the damage of epithelium and its desquamation. These can explain the lack of clinical symptoms of bronchitis with silicosis.

Emphysema is a usual companion of silicosis. At first, increased lung pneumatosis develops, which can be seen in the change of percussion sound, mostly in lower parts of lungs, in some prolapse of lung ends with keeping their good motion. This state can be evaluated as refectory reaction onto the fibrosis process, which is in the opening of alveoli. Further, a real emphysema develops, though the chest percussion does not show clear box sound above the total area of lungs even then as it happens with other serious forms of emphysema.

Microtraumatization of mucous tunic of the respiratory apparatus by dust particles causes reactive inflammation. Clinically this state is shown mostly in rhinitis, as nasal cavity is affected by dust factor more than the areas, located in lower, like gullet, larynx, and trachea, for which it serves like a filter. Subjectively the sick are concerned with the feeling of “stuffed” nose, sometimes “scratchy” throat, light throat soreness and coughing.

Besides problems with respiratory organs, patients with silicosis have problems with cardio-vascular system as well, though this problem is not obvious clinically for a long time. Patients do not usually have complaints on that. Heart borders are not changed. Tones are clear and rhythmical in the majority of cases. Progressing of silicotic process, and development of lung emphysema causes narrowing of vessel duct with the development of hypertensia in the system of lung artery, increase of stress onto the right ventricle, and its hypertrophy, and then its widening. Arterial pressure is within norms for a long time, but it has tendency to decrease.

Marked changes in periphery blood at non-complicated silicosis are absent. There can be inclination to the increase of the number of erythrocytes and hemoglobin, and moderate leucopenia, mostly conditioned by decrease of lymphocyte content. With progressing of silicon process, amount of leukocytes increases due to appearing of inflammation. ESR increases, what can be caused by the change of protein content of blood serum to the increase of the level of coarse-dispersed fractions, in particular γ-globulins.

Paramount affection of respiratory organs with silicosis conditioned the necessity to research functions of external breathing. The most informative

are such indications as vital capacity of lungs (VCL), its relationship to the corresponding forced VCL, FEV_1 and its correlation with VCL (Tifno test), the data pneumotachometria (in particular, the exhalation power), as well as determination of residual volume of lungs).

VCL of patients with silicosis has the tendency of decrease along the progress of fibrosis process. However, in the primary stages, it can stay at a normal level for a long time. Decrease of VCL is conditioned particularly by the reduction of breathing reserves, particularly of additional air. FVCL indications and pneumotachometria reduce as well, particularly in cases of bronchitis manifestation, what shows the disorder of bronchial permeability.

In the whole, for the silicosis, it is characteristic to have moderate expression of restrictive – obstructive type of disorder of external breathing.

Correspondingly to the existing classification, there are three clinical and radiological stage of silicosis.

Stage I. Patients complain to have dyspnea when having much physical activity, pain in the chest without clear localization, variable dry coughing. Objectively determine indications of basal emphysema, auscultatively – stiff, in some places, vesicular breathing is somewhat weak.

The radiological photograph of lungs at silicosis of Stage I shows double-sided increase and deformation of lung picture, moderate carnification and the change of the structure of lung roots. In case of nodular forms of silicosis on the background of changed lung picture, there is a small amount of punctuate shadows of medium intensiveness, from 1 to 2 mm in size, located mostly in lower and mid lung. Interlobar pleuron to the right is often thickened. On Fig. 1, radiological pictures of normal lungs and lungs with pneumoconiosis of stage I are compared.

As to the function of external breathing, there is moderate compensatory hyperventilation on the background of normal or even some increase of VCL indications.

Stage II. It is characterized by the intensification of dyspnea, pain in the chest and cough. These complaints become permanent. Objective examination shows limitation of the motion of the lower end of lungs, as well as reduction of excursion of the chest.

For silicosis, stage II, more marked intensification and deformation of lung picture. A number of nodular shadows is increased also, size of which achieves from 3 to 10 mm. Sometimes, there is obvious tendency to the joint of nodular shadows. Lung roots are expanded, carnified and start looking “cut”. The pleura can be thickened and deformed (Fig. 2). Radiological picture starts looking like disseminated miliary tuberculosis (“snow storm”).

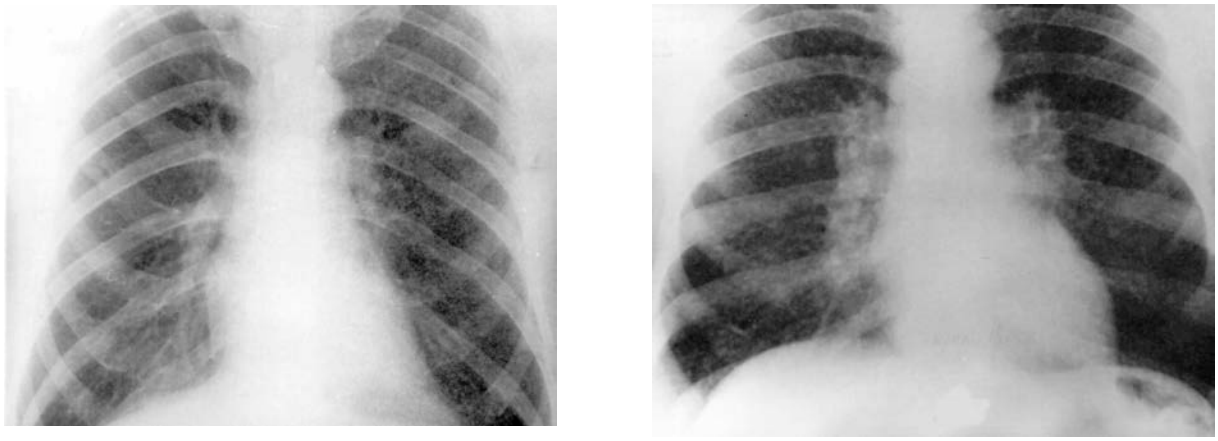
From the point of view of external breathing, lung vital capacity reduction takes place on the background of the increase of minute volume.

Stage III. It is characterized by dyspnea in rest, intensive pain in the chest, coughing with phlegm discharge, and possible fits of asphyxia. Above the lungs,

alternation of sectors of box sound with dull ones can be palpated; and with auscultative methods, it is possible to observe the alternation of breath weakening and rough ones.

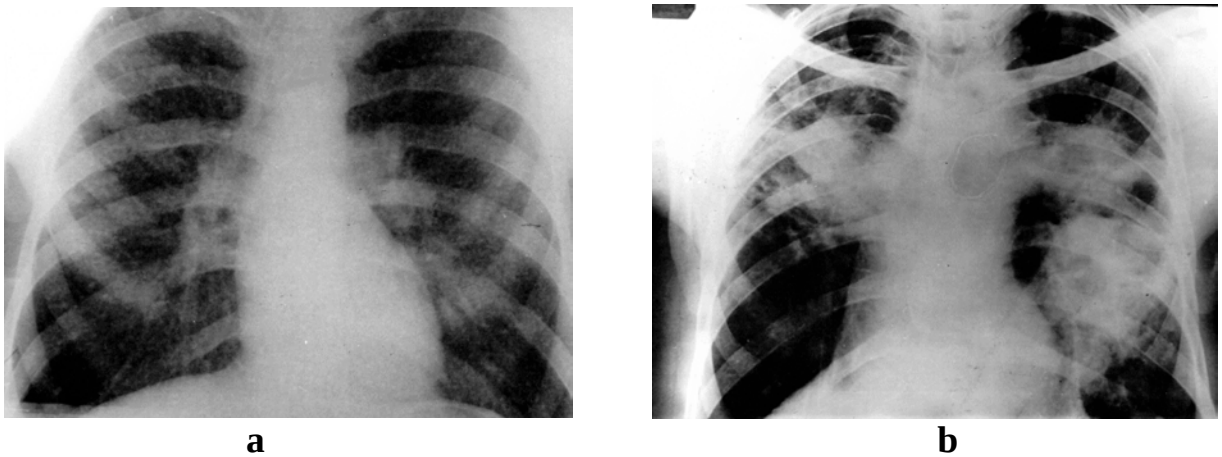
Radiologically, at the silicosis of Stage III, massive shadowings are created on the background of changes, which are characteristic for Stage II. Besides that, marked pleurodiaphragm and pleurocarcial commissures, as well as buliosnic emphysema can be observed (Fig. 2).

Three clinical and radiological forms can be defined: nodular, interstitial and tumor. It is necessary to determine that for the impact of dust with free silicon dioxide onto a human organism can be characterized by the development of the nodular form of the fibrosis process. A tumor form can be observed with patients with silicosis of Stage III, when all possible disease syndromes in connection with the marked functional disorders are present.



a **b**

Fig. 1 Radiological photograph of lungs
***a* – normal, *b* – silicosis, stage I**



a **b**

Fig. 2. Radiological pattern
***a* – silicosis, stage II, *b* – silicosis, stage III**

Clinical pattern of *"acute" silicosis* develops into two phases. The first (latent) phase is characterized by the process of fast developing of dyspnea and cyanosis; patients lose weight and have fit-like coughing; and it is particularly characteristic for "acute" silicosis to have marked dyspnea and cyanosis.

Insignificant emphysema, a big number of rales (mostly in lower parts), acceleration of ESR, quick increase of the amount of β- and α-globulins, and positive tuberculosis sampling determine this form of silicosis.

Radiological pattern of acute silicosis is variable: from nodular ones with small clusters, which are located mostly in lower parts, to tumors due to merging of some elements, in particular in lower parts as well as development of pleural growing.

Late silicosis develops in several years after the termination of contact with dust, which contains quartz. The process of the disease is severe. There are assumptions that this form of silicosis is conditioned by the presence of "depots" of quartz dust, which then is transferred by phagocytes to various portions of lungs.

Differential diagnostics: To differentiate silicosis is possible with many disseminated processes in lungs, which often stimulate dust pathology, and in particular among people, which work in contact with dust. That is why anamnesis of those occupied with dust professions should be taken very carefully, taking into account professional route, work conditions, harmful habits, previous diseases process and development of the found pathology, and radiological changes together with the clinical pattern. Often there is the necessity to involve auxiliary research instruments – tomography, bronchography, bronchoscopy, biopsies, specific sampling, etc. to be able to make a correct diagnosis.

It is necessary to remember that very often it is necessary to differentiate it with the lung tuberculosis, in particular, in cases of hematogenous – disseminated tuberculosis, which can develop in the form of acute miliary and chronic tuberculosis. With lung form of acute miliary tuberculosis, the main criterion is the clinical pattern. Unlike silicosis, the disease starts with the acute form, and is accompanied by the increase of temperature, and in some cases it has a hectic character with profuse sweating. Dyspnea is one of the constant and heavy symptoms. It is so acute that patients cannot sleep, talk, or lie because of it. That is why this form is called "asfiphyxic form of acute tuberculosis". Dry heavy coughing, often in the form of fits, is characteristic for the disease. In the beginning of the disease, objective changes are absent. A patient's face is pale; cyanosis of lips and cheeks is marked. Discrepancy between hard dyspnea and cyanosis, on the one hand, and absence of auscultative changes, on the other hand, can be observed. Then, bubbling subcrepting rales appear, which can go before dry ones.

It is characteristic for it, that the radiological photograph of the chest for the first week of the disease, with marked dyspnea and cyanosis, does not show

any clusters. Only during the second week, leveled rush of miliary clusters on both parts of lungs can take place, which are not bigger than a millet grain in size. Such clusters are most typical, but they can be bigger. Density and intensity of shadows, like with silicosis, are more significant in middle and lower parts of lungs, but mostly near the mean wall; with silicosis – it is in lateral and mean ones. Lung roots are not clear and non-differentiated during the first week; then their change depends on the degree of affection of bronchial glands.

Thus, the total clinical pattern of acute miliary tuberculosis differs from the development of silicosis, and radiological changes are characterized by more common rush, and the speed of development; when treating, positive results can be observed, what is not characteristic for silicosis. But there is not such density, expansion or cut-ends as with silicosis. More often it is necessary to differentiate silicosis with chronic tuberculosis. In these cases, the start of this specific process can look like flu.

When making a diagnosis, disease anamnesis is very significant. It is necessary to clarify, in how many years of working with dust, the first indication of the disease appeared? what were the first work conditions? During the period of tuberculosis getting acute, there are symptoms of intoxication, temperature and changes in the clinical analysis of blood, which are very important for differential diagnostics, and the tuberculosis treatment if started then, can solve it completely.

It is also necessary to know well about the compound of this pathology – silicotuberculosis. Most often, tuberculosis is the secondary disease with silicosis. It is also necessary to take into account that the more marked silicosis is, the more often it is accompanied by tuberculosis. In this case, the progress of tuberculosis is characterized by a number of peculiarities. Very often, extrapulmonary tuberculosis develops, and in the mucus, microbacteria of tuberculosis can be often found. TB diagnostics is not very important here, as with silicosis, there is often increased sensitivity to tuberculine. Conducting differential diagnostics between silicosis and silicoturbeculosis, it is necessary to take clinical and radiological patterns into consideration. On the background of marked silicosis, we have to orient more at the clinical manifestation of tuberculosis: frequent catarrhal diseases in the anamnesis with the increase of temperature, symptoms of intoxication, coughing and dyspnea. Objectively, these are local bubbling rales, which appear or increase, when coughing, in particular, in upper parts of lungs, what can prove TB diagnosis. Noise of pleaura rubbing and hemoptysis take place more often than with distinct silicosis.

Changes in blood sampling assist in diagnosis making: increase of ESR, change of leukocyte formula to the left, lymphopenia and sometimes, monocytosis. There also can be anemia, changes in proteinogram towards increasing of coarse-dispersed fractions, mostly γ-globulines, and positive reaction onto the C-reactive protein. Radiologically, tuberculosis can be found

due to asymmetrical shadow clusters, infiltrates, and small or big nodular shadows, as a rule in upper areas of lungs, which can be distinguished from silicotic nodules by their size.

Much importance to diagnose tuberculosis is given to positive reaction to antibacterial therapy. However, it is necessary to remember, that with silicotuberculosis, this reaction comes slower, than with pure tuberculosis, that is why it is necessary to be taken into account when making differential diagnosis.

Cases of sarcoidosis can be observed much more often lately. The etiology of this disease has not been found yet. The similarity of radiological changes and sometimes lack of clinical pattern of the pulmonary sarcoidosis reminds silicosis much. Both pathologies can be found by chance at radiological examination.

As with silicosis, the process of sarcoidosis development includes three stages. *Stage I* is characterized by clear increase of internal pectoral lymphatic glands, vascular – bronchial pattern at this time is not much changed, whereas at Stage I of silicosis there is already an interstitial fibrosis; and the pattern is intensified and deformed, also some individual shadows can be found.

Lung roots are particularly distinguished, they have polycyclic look at stage I of sarcoidosis, and with solicosis they look cut.

Patients with the disease of *Stage II*, changes in vascular –bronchial pattern remind interstitial form of silicosis, however, they appear mostly in the area near roots, in contrast to silicosis, for which affection of cortical portions of lungs is characteristic.

Stage III of the disease is particularly severe to be diagnosed, as polymorphic fibriosis can take place both with silicosis, and sarcoidosis, and conglomerate shadows, which are created at silicosis, are moved to roots of lungs, what is more characteristic for sarcoidosis.

The main and most reliable indication of sislcosis is certificate in lung roots, and particularly the symptom of “egg shell”. The latter is never observed with sarcoidosis.

It is important for diagnostics of sarcoidosis of extrapulmanory indications of the disease among patients: skin affection, affection of lymphatic nodules, and affection of locomotorium.

Treatment with corticosteroids gives positive results among patients with sarcoidosis in contrast to the patients with silicosis.

Silicosis should be also distinguished from the *syndrome of Haman – Rich*. This disease is also called fibrosing alveolitis, or progressing diffusive fibrosis.

The start of the disease can remind pneumonia, however, antibiotics do not usually help as a rule, but on the contrary, they make the state of the patient even graver. The temperature can be febrile, subfebrile or normal. ESR either grows or stays normal.

Coughing is of unstable character, and can forego dyspnea. In lungs, sound bubbling rales can be heard. On the radiological photograph, interstitial small microcellular fibrosis can be seen. This disease can be differentiated only with chronic development. In contrast to the dust pathology, it is characterized by deterioration of the patient. Dynamic observation of the clinical pattern and radiological changes can show “disproportion”: with rapid grow of pulmonary insufficiency, small dynamics is observed, i.e. stability in radiological pattern is observed.

At any disseminated process in lungs, moreover among those occupied with “dusty” professions, it is necessary to remember about possible cancer as well as the possibility of development of lung cancer (carcinomatosis of lungs).

Among complications of silicosis, there are pulmonary tuberculosis (silicotuberculosis – STB), pneumonia, bronchiatic disease, bronchial asthma, atrophic arthritis, spontaneous pneumothorax, and coniotic cavity.

Tuberculosis of respiratory organs can be linked with dust diseases of lungs- pneumoconiosis, and especially with the most spread one – silicosis.

Tuberculosis can be met particularly often with small and big nodular forms of silicosis, as well as among patients with grave development of the process.

Silicosis with gradual combination with tuberculosis, tuberculosis with further combination with silicosis as well as silicotuberculosis, where the character of the preliminary affection is impossible.

As a rule, tuberculosis with developed silicosis is secondary. The source of tuberculosis process includes old centers, located in upper and cortical portions of lungs. Spreading of the process goes through lymphogenous, bronchigenous and, sometimes, hematogenous ways. It is considered that the peculiarity of tuberculosis spreading process with silicosis is in selective affection of lymphatic system.

Due to significant compensatory possibilities of the organism, silicotuberculosis is not obvious clinically for a long time. After some time, temperature of the body increases, a patient starts coughing, and weight losses can be observed. Along progressing of the disease, symptoms become more distinguished. General condition worsens, intoxication increases, functions of breathing and blood circulation worsen as well. However with silicotuberculosis, intoxication is less clear, than with similar forms of pulmonary tuberculosis, not connected with silicosis, and the discharge of microbacteria does not take place with marked tuberculosis either.

There is no adopted unified classification of silicotuberculosis. In practice, classification of silicosis and tuberculosis (given in Table 3) is usually used.

More often with silicotuberculosis, nuclear form can be met, affection is rarely double-sided with polymorphic nuclears - 1.5 cm in diameter, which are located, as a rule, in under collarbone areas, and in upper portions of lungs. Diagnosis of nuclear tuberculosis on the background of marked silicosis is

difficult to be made, because tuberculosis nucleons are difficult to be distinguished from merged silicotic nodules.

Table 3

Clinical Characteristics of Silicotuberculosis (A.Goldelman and D.Zislin)

Clinical forms of tuberculosis, complicated with silicosis	Characteristics of the STB Process	
	Process phase	Site
Silicotuberculous bronchadenitis	Infiltration Insemination Carnification	Bronchopulmonary lymphatic glands
Nuclear	Infiltration Carnification Calcination	Mostly segments I and II
Infiltrated	Rarely, decay phase	Mostly segments I and II
Disseminated	Infiltration Carnification Decay	Restricted in upper portions and spread
Silicotuberculoma, isolated	Decay or without it	Mostly segments II and III of upper portion, mostly to the right
numerous	Same	Segments of II and III upper portions, and IV and V mean portions
Fibrous – cavity	Insemination Infiltration Cirrhosis	Mostly segment of II and mean portions
Conglomerate	Stabilization Infiltration Decay	Mostly in upper portions, but other localization can also take place

With all forms of silicotuberculosis, changes of lung roots occur, conditioned by the increase or, sometimes, calciniosis of lymphatic nodules, fibrosis of lungs and hypertensia in small circle of blood circulation. Classification of tuberculosis is incapable to unite all forms of tuberculosis development at silicosis. Thus, the clinical and radiological classification was suggested, where four main groups of atypical forms of silicotuberculosis are given:

1) Silicotuberculous bronchadenitis with major localization of tuberculosis process in innerpectoral lymphatic glands;
2) small nodular form of silicotuberculosis with appearing of individual shadows, up to 3 cm in diameter;
3) big nodular silicotuberculosis with single or many shadows, with the diameter from 3 to 8 cm, rounded forms – silicotuberculoma.
4) massive silicotuberculosis, where the clinical form of tuberculous form is impossible to state.

Among complications of silicosis, it is necessary to mention spontaneous pneumothorax, which is mostly limited, and thus it develops as non-malignant; however in some cases, it can develop into a total or even double-sided

pneuomothorax with air spreading to the mean wall portion. Valve pneumothorax can be observed as well.

Interstitial pneumonia, as a silicosis complication, changes the degree of compensation of the organism. Bronchoectatic disease with silicosis is a rather rare complication. Mostly, combination of silicosis with bronchial asthma can be observed. Cancer with silicosis is observed as often as with pneumosclerosis of non-occupational etiology. Exceptions are the cases when patients are within the work zone, where ores contain radioactive elements. Then the development of silicosis is more often combined with new formations in lungs.

Cases of combination of silicosis with diseases of conjunctive tissues can be observed more seldom, like atrophic arthritis, sclerodermatitis, and system lupus erythematosus. Combination of silicosis with affection of joints like atrophic arthritis is known in the literature as the Syndrome of Coline – Caplan. Whereas, X-ray can show the presence of rounded shadows, located mostly along the periphery of both lungs and consisting of atrophic granulomas and silicotic nodules.

Atrophic arthritis on the background of silicosis can develop without particular indications of visceral affections. Sometimes, in the clinical pattern of silicoarthritis, affection of internal organs is the main. Lethal cases of patients with silicoarthritis from uraemia are known, caused by atrophic affection of kidneys.

Combination of silicosis with sclerodermatitis is not a rare case, especially among miners.

Treatment. Main approaches in treatment of patients with silicosis are based on the understanding of the mechanism of the disease development, character of morphological and functional changes, especially of the progress and complications.

First of all, it is necessary to remember that patients with silicosis of Stage I, who do not have external respiratory function disorder, should work rationally. Strengthening of general state of organism and increase of its protective forces, in particular by training and tempering, are very important. To do so patients should be recommended to do hygienic physical exercises and walking in fresh air. Significant role is played by rational meals (special diet, enriched with protein – 100 – 150 g of cheese, with adding pancreatin or methionine in the dosage of 0.5 to 1 g a day).

Among means of impact onto the pneumoconiotic process, it is very important to withdraw dust from lungs by inhalation of mineral waters of various composition. It has positive impact onto the mucous tunic of trachea and big bronchi.

Such gradual development of fibrosis process with silicosis served as a basis for clinical utilization of glucocorticoids. But then it was found out that patients with non-complicated silicosis should not take in glucocorticoids. They can be prescribed only to patients with combination of marked stages of silicosis

and atrophic arthritis, as well as in case of fast progressing silicosis. The following treatment regimen is suggested: prednisolone in the dosage of 20 to 25 mg a day. Maximum amount is prescribed for 10 to 12 days, then it should be gradually decreased by 2.5 mg every 5 to 10 days.

Treatment with corticosteroids should be conducted only under protection of anti-tuberculous drugs, among which phthivazide and para-aminosalicylic acid, PAS(A) are the best, whereas these drugs should be prescribed for 1 to 2 months after hormones are cancelled.

Among drugs, which have antifibrous action, it is possible to use 2 % solution of poly-2- vinylpyridine-N-oxide, which is introduced intravenously together with the isotonic solution of sodium chloride (150 – 200 ml) in the drop form every other day. One-time dose of 0.1 – 1 g, and the course is 15 to 20 infusion.

However until now, there is no unified thought as to the efficiency of this drug; there is data on side effects of poly-2- vinylpyridine-N-oxide (gonadotropic and cancerogenic). Besides, some clinical testing has not established treatment effect of the drug. Thus, search for efficient polymers, capable to restrain the development of fibrous process in lungs, continues.

There is positive data regarding utilization of glutamic in the dosage of 0.25 – 0.5 g three times a day for patients with silicosis. It restrains the development of silicosis and asbestosis in the experiment, decreases the level of progressing and causes reverse development of dust fibrosis in lungs.

The main pathophysicological disorder, which appears in the very first indications of silicosis, is oxygen insufficiency. Thus, in the therapy of patients with silicosis, it is necessary to use oxygen therapy, drugs, which stimulate activity of the respiratory center (Cordiamin – 25 to 30 drops or 1 – 2 ml subcutaneously). Besides pathogenetic methods of treatment, significant place in therapy of patients with silicosis is taken by symptomatic therapy.

If patients cough, they are recommended to take in expectoration drugs: 3 % of potassium iodide solution or 0.5 % of the tincture of termopsis herb. For bigger effectiveness, this drugs should be taken in with much water.

Among physiotherapeutic methods of treatment, the following have been proved to be good: ultrasound, particularly among patients with uncomplicated silicosis of Stage I with the presence of pain syndrome, coughing, disorder of drainage function of bronchi, bronchial permeability, as well as electrophoresis with various medicinal drugs, depending on one or another clinical syndrome.

Particular place in treatment of patients with silicosis is taken by drugs, which increase total reaction of the organism. They include alcohol extract of eleuteroke, which is taken in by 30 to 40 drops 30 min before meal each day for 30 days.

Among medicinal drugs, which create non-specifical stimulation of the patient’s organism, it is possible to name prodigiosan, introduced

intramuscularly in the dose of 25 to 50 mg of 0.005 % solution once a day every 4 to 5 days, the course is from 3 to 4 injections.

Treatment measures should be directed at the fight against complications as well (development of cor pulmonale, cardio-vascular insufficiency, or pneumonia). To do so, cardiac glycosides are taken (strophanthin 0.5 ml of 0.05 % solution), aminophylline (5 – 7 ml of 2.4 % solution), diuretic (furosemide and hydrochlorothiazide). Treatment of pneumonia should be purposeful, with the consideration of the character of microflora (ampicillin, 0.5 g, 4 times a day; sulphalen – the first day – 1 g and then 0.2 g a day). Protein synthesis in the organism can be stimulated by anabolic steroids (metanedrostenolon – 0.005 g 1 to 2 times a day before meals). Treatment course should be from 3 to 4 weeks, retabolil – 1 ml of 5 % oil solution intramuscularly, total 8 – 10 injections.

Main method of treatment of silicotuberculosis is chemotherapy with modern anti-tuberculous drugs: isoniazid (dosage – 0.6 – 0.9 g a day), rifampicin (average dose – 0.45 g a day); ethambutol (15 – 25 mg per 1 kg of body mass a day); etionamid (inside, 20 min after meals, once a day 0.5-0.75 g); streptomycin (intramuscularly, in the dosage of 0.5 – 1 g a day).

Total duration of the treatment of active forms of silicotuberculosis – not less than 1 – 1.5 years. Besides, the whole arsenal of medicinal drugs against silicosis can be used (generally sanative, physiotherapeutical therapy) with the consideration of the development and expression of coniotic process.

Verification of work ability: Silicosis of stage I of the stage is a contraindication of industrial dust impact. Patients should be transferred to another job beyond the conditions with dust, irritating gases and unfavorable metrological factors. Hard physical work is contradicted also. If rational job is connected with the decrease of qualification, the patient should be assigned to attend doctor – labor commission to receive Disablement Group III on occupational disease.

At stage II, the patients always have the right to receive Invalidism Group (mostly Group III, and in case of its combination with tuberculosis, and respiratory insufficiency – Group II).

At stage III of the disease, there is marked respiratory and cardio–vascular insufficiency, and sometimes the need of assistance from others (occupational disablement of group II or I).

Preventive measures of silicosis envisage the conduct of the following:

- complex mechanization of production processes;
- sealing-in of the machines;
- organization of efficient production ventilation;
- hygienic norming of professional hazards;
- record-keeping and research of specific cases of occupational diseases;
- biological methods of prevention methods:

a) general sanative ones (rational organization of the work and leisure, rational meals, and physical exercises);

b) special (respiratory exercises, inhalation of aerosols, and rational meals with vitamins);

- preliminary and periodical medicinal examination of people, who work under conditions of professional hazards;
- utilization of individual means of protection.

Silicatosis

Silicatoses are pneumoconiosis, which develop in the result of inhaling of silicate dust.

Besides, free silicon dioxide, in the nature there is a number of complex mineral compounds, which include silicon dioxide, which is not in a free mode, but in connection with other elements (silicates). Silicate ores can be met in mountain mines: in asbestos – talc mining industry. Silicates are used in fire clay and dinas production, when manufacturing rubber products, perfume and many other industries.

Work processes, connected with extraction and utilization of silicates, are often accompanied by inhaling silicate dust. Long inhaling of this dust can cause the development of pneumoconiosis – silicatosis. The type of dust, which caused it, determines type of silicatosis.

Asbestosis is silicatosis, which is caused by inhaling asbestos dust.

Asbestos is a mineral with characteristic fibrous structure, which is widely used to produce thermal isolation materials. Creation of asbestos dust takes place in mining asbestos, as well as during its sorting and processing. The dust degree of the environment directly depends on the character of production process and is very high when crushing asbestos. Boundary permitted concentration in the work zone for aerosols of natural asbestos, as well as mixed asbestos-natural dust with the concentration of asbestos in them over 10 % is 2 mg/m^3; for asbestos bakelite – 8 mg/m^3 and for asbestos – cement – 6 mg/m^3.

Clinically, asbestos is apparent in a number of complexes of chronic bronchitis symptoms, lung emphysema and pneumosclerosis, which are accompanied by dyspnea and coughing.

Dyspnea is one of the first symptoms of the disease. At first, it appears at physical activity, and in more serious conditions, it can be observed in rest as well. Together with dyspnea, dry rales can be observed, which later develop into viscous expectorating mucus; sometimes, coughing is in the form of fits. Rarely, astmatic phenomena can take place as well.

Characteristic complaints onto dyspnea and coughing are often accompanied by pain in the chest, and in particular when making deep inhales. Patients with marked asbestosis have disorders of the general conditions: headaches, general weakness, and undue fatiguability. Sometimes, dyspeptic phenomena take pace as well. Appearance of patients can have peculiar gray-

ashy color with light cyanosis of lip mucous tunic. Patients can start losing weight.

The chest often has a usual form. When examining, emphysema can be found in upper portions. Breathing is rough with prolonged exhaling, in the lower portions – weakening is observed, often with dry disseminated crepitations; in lower parts, fine and medium moist rales can be observed. Cor pulmonale develops late. At first, there is accent of tone II on the lung artery. Labial pulse develops with time, as well as tachycardia and phenomena of decompensation on the bigger circle with characteristics changes for that time on the electric cardiogram (tall waves P_2 and P_3, reduction of interval S – T in II and III sectors, dextogram).

Sometimes in the mucus, asbestos corpuscles can be found, which are light yellow lumps of prolonged form with clavate or circular endings.

Asbestosis appears among those who working contact with asbestos dust for about 5 years or so, whereas in contrast to silicosis, clinical indications are usually ahead of radiological.

In compliance with the clinical and radiological indications, usually there are three stages of asbestosis: I, II and III; stage III can be observed in single cases – in particularly unfavorable work conditions or with complication of chronic pneumonia and bronchoectatic disease.

Patients with the disease of stage I have marked diffusive emphysema of lungs, diffusive intensification of vascular- bronchial pattern, more intensified in lower portions of lungs (bronchitis and bronchiectases) and fine cellular pattern in mean portions, found after radiological examination.

Lung roots are slightly widened, shadows are dense, and their structure is rough.

Patients with asbestosis of stage II have the same indications, only in more marked form based on radiological examination. Vascular – bronchial pattern has more coarse cellular structure. Sometimes, there are numerous punctuate shadows of nodular character. Lung roots are denser and widened. There can be indications of the beginning of cor pulmonale.

With the disease of stage III, there are marked phenomena of pneumosclerosis and emphysema. Often there are significant changes of pleura and characteristic indications of cor pulmonale. In contrast to silicosis of stage III, with asbestosis of stage III, there is not many pneumosclerotic fields.

As it is, pneumosclerotic process from asbestos dust action does not have a tendency of fast progressing. The severity of the condition of patients with asbestosis depends on the degree of the expression of bronchitis, emphysema of lungs, development of bronchoectasis, and infection joining. The given complications are the main reason of significant disorders of respiratory function and disorders of hemodynamics of small blood circulation circle.

Talcosis is silicatosis, which appears due to the talc dust action. Talc is magnesia silicate, which does not dissolve in water, and slightly dissolve in

acids and alkali. In the production, talc is used in rubber, textile, and paper industries. Highest sorts of crushed talk are used in perfume industry.

Pneumoconiosis caused by pure talc, when it is mined and used, develops usually not earlier than after 10 years of work. The process of the disease is moderate. Complaints: dyspnea at physical activity, pain with unstable character in the chest, coughing, mostly dry with some mucus. Weight losses are observed also. With percussion, box sound is heard in lower lateral portions of the chest. With auscultation, coarse breathing can be heard.

Radiologically, fine cellular pattern of lungs and single shadow spots are observed, which cover the lung area levelly. Lung roots are somewhat widened.

However, the clinical and radiological pattern of the talc pneumoconiosis can be more marked as well. Workers, engaged in talk mining and primary processing of ores, can have diffusive fibrous changes in lungs, which on the x-ray remind a pattern of silicosis of groups I and II. It is necessary to consider that the bigger degree of affection in this case can be explained by the additive of talc to free silicon dioxide, i.e. silicotalcosis takes place here.

There are three stages of talcosis:

Stage I. Patients complain to have dyspnea when having physical activity, unstable coughing, and pain in chest. With percussion, it is possible to determine indications of basal emphysema, and with auscultation – coarse, and in lower lateral portions – slightly weakened respiration, and dry tales. After radiological examination, it is possible to observe the increase and deformation of vascular pattern due to the development of fibrous process, as well as deformation of lung roots.

Stage II is characterized by the increase of dyspnea, coughing and mucoid sputum. Objectively, complex of symptoms of bronchitis with emphysema indications. Radiological examination shows a marked intensification of vascular – bronchial pattern and emphysema. Lung roots are widened and deformed.

Stage III. Dyspnea in rest, coughing and intensive pain in chest, as well as presence of cyanosis. After percussion of lungs, box sound is observed, quick restriction of movements of the lower portion of lungs. Dry and moist rales can be heard. As to the cardiac-vascular system: widening of the right border of heart, cardiac tones are muffled, accent of tone II is over the pulmonary trunk. Radiological examination shows pneumosclerosis. Lung roots are widened.

Cement pneumoconiosis is silicatosis, caused by the action of cement. Cement is silicatosis, but it includes free silicon dioxide as well. In the production dust of Portland cement, there are from 3 to 7 % of free silicon dioxide, and in the production dust of acid-proof cement – up to 67 %. The permitted concentration for cement dust is 6 mg/m^3.

Cement pneumoconiosis is characterized by complaints on coughing, mild pain in chest and dyspnea at physical activity. The progress of pneumoconiosis depends on the type of cement, which cased the disease. Thus, with prolonged

inhaling of Portland cement dust, interstitial dust fibrosis gradually develops among workers. Workers, producing acid-proof cement, can have the disease with comparatively low term of work (from 7 to 9 years). During radiological examination in this case, besides interstitial fibrosis, there can be fine-nuclear formations in mean portions on both sides, which remind silicosis. Complications with tuberculosis are rare. Besides, pneumoconiosis, there is development of bronchial asthma, skin irritation, and “cement rash” and conjunctivitis.

Treatment. To treat silicatosis, means are utilized which stimulate protection forces of the body (solux, ultra-violet radiation, oxygen therapy, and respiratory exercises). Bronchological, antihistamine and inflammatory drugs, as well as vitamins (P, ascorbic acid and nicotinic acid)In case of complications of silicatosis, the following can be prescribed: antibiotics and sulfanamides (pneumonia), cardiac glucosides – strophanthin, corglucon, and diuretics – lasix, and hydrochlorothiazide (cor pulmonale).

For further rehabilitation, it is advised to take resort treatment (Crimea) under conditions of marked cardio-vascular insufficiency and exacerbation of the inflammatory process.

Verification on work ability. Patients with silicatosis of stage I are subjects to rational work, in particular if they have bronchitis, pneumonia or signs of further development of fibrous process (asbestosis, rarely talcosis, or olivinosis).

If the stage of silicatosis is II or III, patients should get disablement group II or I of occupational character (first of all, if there is chronic obstructive bronchitis, cor pulmonlae, marked decrease of external breathing function).

Preventive measures. To prevent appearing of silicatosis, it is important to take sanitary – technical measures (sealing in and mechanization of production processes, ventilation, utilization of individual respiratory organ protection means) as well as conduct of preliminary and periodical medical examinations.

Carboconiosis

Carboconiosis is pneumoconiosis, caused by the action of dust, which contains carbon (coal, graphite, or coke).

With carboconiosis, moderately marked mostly fine nuclear and interstitial fibrosis is observed. Anthracosis is one of the most spread and practically the most important disease in this subgroup of pneuconioses, which usually develops among miners, engaged in mining coal, as well as workers of ore-dressing plants and some other manufactories.

Among workers of coalmines, depending on labor conditions and domination of some type of dust, there are three types of pneumoconiosis: anthracosis, silicosis and anthrasilicosis.

Anthracosis. It appears and develops in case of long work period in mines (from 15 to 20 years and inhaling the air with high concentration of coal dust. The permitted concentration of dust of coal, which contains less than 2 % of silicon dioxide, is 10 mg/m^3.

Pathological and anatomical pattern. Coal dust depositing in lungs is primarily characteristic for anthracosis. Lungs become of gray – black color. In marked stages of anthracosis, conglomerations of coal dust pigment is levelly spread along the total area of lungs.

In alveolar septum, around vessels and bronchi there is union of conjunctive tissue, in some places clusters of cells with particles of coal dust are observed which got their name from anthracotic nucleuses. In contrast to silicotic nodules, they do not have concentrically placed beams of conjunctive tissue. Pneumosclerotic changes are joined with emphysema of lungs and chronic bronchitis.

With anthracosis, silicotic nodules can be observed in lungs.

Clinics. For coal pneumosclerosis, more marked bronchitis and lung emphysema are characteristic, what distinguishes it from silicosis. More marked and functional disorders of the external respiratory apparatus can be observed. Radiological changes contain diffusive interstitial fibrosis and numerous small nuclear formations on the background of emphysema of lungs. There are three stages of the disease.

Stage I. Patients complain on fast fatiguability, dyspnea at physical activity, coughing and pain in the chest. Sometimes, complaints are absent, though radiological photograph register clear changes. Radiological pattern is characterized by the appearance of small nuclear shadows on the background of cellular deformed lung pattern in mean portions (mostly to the right) with the presence of bigger shadows of lung roots. The diameter of clusters is between 1 to 3 mm, and sometimes 1 to 5 mm.

Stage II. Complaints on dyspnea, and sometimes in rest. fast fatiguability, and pain in the chest. Objectively, emphysema and bronchitis are observed. Radiological pattern is marked with the increase of the number and sizes of fine nuclear shadows, located not only in the mean, but also in subcollar bone portions. Shadows of lung roots are widened; their intensity is increased. Pleural changes can be changed often in the upper portion and emphysema is marked.

Stage III (is rare in the conditions of coal dust only). Complaints on general weakness, dyspnea even in rest and at light physical activity, coughing, often with mucus, and pain in the chest. During radiological examination, massive homogenous shadows of irregular form with clear outlines can be observed, which are located symmetrically or on different heights in both or one lung among fine nuclear and porous formations.

As to clinical and radiological development, anthracosis is comparatively a non-malignant, moderately progressing chronic disease. Combination with

tuberculosis worsens the forecast much. However with anthracosis, the danger of complication with tuberculosis is much less than with silicosis.

Coal miners can have both anthracosis and silicosis. Anthracosis is usually observed among miners, who work full-time on coal extracting and face lavas. Tunnellers, who conduct preparatory works and inhale dust with silicon dioxide, have anthracosis or silicosis developing. That is why to solve the problem as to the character of pneumoconiotic process, it is necessary to study occupational anamnesis and work conditions well.

Graphite pneumoconiosis. It is carboconiosis, caused by graphite dust. Graphite is a dark gray substance with fair chemical activity, one of the variations of coal. It is used to produce cast iron, stainless steel, electrodes, in electrical devices, as well as to make pencils and paints.

Pneumoconiosis caused by graphite dust, develops slowly, after the work period of over 10 years, and has non-malignant character. Patients with graphite pneumoconiosis complain to have pain in the chest, dyspnea and fast fatiguability. After objective examination, chronic bronchitis and lung emphysema are observed. Harmful impact of graphite dust onto the ENT-organs is observed (atrophic pharyngitis and rhinitis).

Radiological examination shows cellular fibrous process, which correspond to stages I or II of the disease. However, in rare cases with graphitosis, graver changes in lungs can be observed, i.e. large fibrous fields with portions of necrosis, which can be explained by particularly unfavorable conditions.

Verification of work ability. In case of appearing of pneumoconiosis of Stage I, without complications of disorders of external respiration, a patient can work without preliminary attending the Expert Commission. The obligatory condition is dynamic control of the health state by doctors and work conditions normalization. If pneumoconiosis of stage I is combined with bronchitis, and moreover in case of appearing of pneumoconiosis of stage II or III, further work in dust conditions is contra-indicated.

Preventive measures: prevention of dust creation (wet drilling and mechanization of work processes, as well as medical examination of workers.

Metal-coniosis

Metal-coniosis is characterized by depositing of radiological contrasting dust in lungs with moderate fibrosis reaction (siderosis, baritosis, etc). These pneumocioses are distinguished by non-malignant development. According to the current classification, berylliosis, aluminosis, pneumoconiosis caused by cobalt dust and toxic air with repeated reaction of pulmonary tissues are included to the group of metal-coniosis.

Aluminosis is metal-coniosis, caused by aluminum dust action.

Aluminum is white silvery light metal. Metal aluminum and its compounds are widely used in the industry. Contact of workers with aluminum

dust or vapors takes place when producing metal dyes, artificial abrasive elements, pyrotechnic aluminum powder, etc. The permitted concentration for the aluminum and its compounds is 2 mg/m^3.

Aluminum gets into the organism when inhaling its vapor and dust. The term of development of aluminosis varies from 6 months to many years.

With such concentration of aluminum dust, changes in lungs can take place within the first year of work in the result of probably allergic reaction of the organism.

Mechanism of the action of aluminum dust onto the pulmonary tissue has not been completely understood yet. Obviously, under the impact of the tissue fluids, aluminum ions, starting reaction with proteins in the organism, create strong complex compounds, which violate normal life activity of cells. With time, interstitial regenerative union of collagen- hyaline tissue takes place. Around of some particles of aluminum, non-dyed membranes are observed, which are called “aluminum corpuscles”.

Clinics. Patients often complain to often have catarrhal diseases, fits of coughing with mucus or without it, tension in the chest, flabbiness, dyspnea, absence of appetite and pain in stomach. Then dyspnea at physical activity is observed, in some grave cases – clear dyspnea and strong suffocating coughing even with insignificant physical activity.

Objectively, clinical data is not very characteristic in the beginning. Breathing is hard, dry rales can be heard. With the development of the process, cyanosis, degrease of vital capacity of lungs, and sometimes up to 1000 ml or less are observed.

In spite of significant disorder of external breathing, radiologically it is impossible to find any changes at this stage. On the radiological photograph, first changes appear in the form of intensification of vascular pattern of lungs, especially in mean portions. Sometimes, there can be single, fine and average sized mottled formations. These changes are located symmetrically. Lung tops in the majority of cases are free. In the future, merging of spotted shadows takes place with the creation of homogeneous carnifications. In severe cases, lung roots are widened and carnified. In contrast to silicosis, there are no nodular formations and tumor-like carnifications at aluminosis.

As a rule, even after the termination of the contact with aluminum dust the dust, accumulated during the production period in lungs, continue acting, and this process progresses inevitably. In blood, limphocytosis and eosinophilia are observed.

Siderosis is metaloconiosis caused by inhaling metallic ferruginous dust.

Mostly it is observed among workers of blast-furnaces and agglomeration factories.

Pathologic and anatomic pattern. Volume of lungs is increased. On their sections, nodules are observed, the diameter of which is up to 6 mm. Lung coloration is black (if impacted by ferrous oxide) or yellowish (if impacted by

the dust with ferrioxide). Lymphatic glands are increased, and are or red color in the section.

At histological examination, excrescence of conjunctive tissue and dust deposits with iron are observed in alveolar septum, as well as around bronchi and vessels. Also, fine nodules (dust pictures, pieces of conjunctive tissue, histiocytes and lymphoid tissues are observed.

Clinics. Clinical pattern of siderosis is very poor. Patients have no complaints during long times, which would prove the affection of lungs. Function of breathing stays unchangeable. Only on the radiological photograph of lungs, lightly marked interstitial fibrosis and disseminated fine nodular shadows with clear outlines, where dust accumulated, are observed. Thus, the diagnosis of siderois is sometimes determined only based on results of radiological examination with the consideration of dust composition, present at the production where the patient works.

Practically, there are no complications with siderosis. Patients with uncomplicated forms of siderosis do not require treatment in the majority of cases. They are completely capable to work.

Berylliosis is metaloconiosis, caused by inhaling beryllium.

Beryllium is silvery-gray light metal. In production, beryllium compounds are used (beryllium oxide, beryllium sulfate, beryllium chloride, and beryllium fluoride) to produce X-ray tubes, luminescent lamps and to receive atomic energy. Beryllium metal is used to receive alloys of strong and sparkle tools, beryllium steel, as well as in ceramic production. The boundary permitted concentration of beryllium is 0.001 mg/m^3.

Beryllium gets to the body through lungs in the form of smoke and vapors. It is mostly deposited in bones, liver and kidneys. It permeates through placenta, and can be found in urine many years after termination of contact with it.

Both beryllium and its alloys are toxic (the most toxic are compounds, especially beryllium oxyfluoride). Beryllium and its compounds have local (onto respiratory tracts and skin) and resorptive (onto the central nervous system and parenchymatous organs) impact and is a carcinogenic matter.

There are two forms of berylliosis: acute and chronic. Dissolved beryllium compounds mostly cause acute intoxications, and non-dissolved compounds – chronic ones.

In **pathogenesis** of berylliosis, an important role is played by autoimmune processes, conditioned by sensitization the proteins of the body itself, what is significantly changed under the influence of beryllium.

According to contemporary understanding, berylliosis is close to colagenoses. Beryllium interrupts the activity of a number of ferments (alkali phosphotasis, and magnesium interaction).

Pathologic-anatomic pattern. With acute poisoning with beryllium, quick plethora and swelling of mucous tunic of trachea and bronchi are

observed. In gaps between bronchi and around vessels there are some erythrocytes and lymphoid cells. Interalveolar septums are thickened due to their infiltration with lymphocytes. In alveolar cavities, there are accumulations of exudation with a big amount of fibrins, separate gigantic cells, Langhasen' s type, as well as peeling of epithelium. Blood vessels are expanded; in the parenthem there are separate hemorrhage focuses. In later stages, industrial alveolitis rakes place in the form of carnificating pneumonia.

At chronic berylliosis, lungs are much enlarged on the macroscopical stage; they are dense of gray red color. The surface of lungs is small-grained. There are numerous fine nodules, which are dense when touched. Morphological pattern with chronic berylliosis consist of epithelial cells, and a small number of lymphoid, plasmatic and multinuclear gigantic patterns of Langhans, located mostly along the periphery. The evolution of granulomatosic process with berylliosis is characterized by formation of fine sclerotic nodules, which form large granulomatosic nodules after merging.

Clinics. Acute form of the disease develops in the following forms:

- in the form of acute affection of conjunctiva and upper respiratory tracts; transition into a sever form with lung affection is possible (disease duration: several days or weeks);
- in the form of "beryllium fever";
- the most sever case – acute brochobronchiolitis or the so-called pneumonitis, the progress of which can develop in two phases: in these cases, the disease starts with symptoms of metal fever, after which a non-symptom stage starts (4 – 6 days), and after that, bronchobronchiolitis takes place.

Patients often complain to have pain in the chest as well as very strong coughing (dry or with heavy expectoration of mucus, often with a mix of blood, asphyxia, committing and clear cyanosis. Tachycardia and hypotensia are well observed as well. Lungs are emphyzematosic; dry and sometimes bubbling rales can be heard. Liver is often bigger in size, and painful. The temperature is increased up to 38 to 39 ^{0}C. In the blood there are neutrophilic leukocytosis with stab changes, ESR is increased. In urine, beryllium is found. The progress is wavelike with periods of worsening, long – up to 2 – 3 months. There are possible relapses in case of returning back to work, as well as beyond contact with beryllium under the impact of intercurrent diseases or without any obvious reason. Radiologically, it is possible to find indications of confluent pneumonia large in size, which covers one or portions of lung at a time and develops with acute reaction of lung roots. With the second version of this affection, together with diffusive decrease of transparency of lung, on the bigger or smaller portion of lungs there are small nuclear shadows from 1 to 2 mm in diameter. Lung pattern is changed on a large scale or diffusive, unclear and of small porous character. Roots are widened and non-differentiated. The described pattern stays in place for 2 – 6 – 8 weeks. Scars can stay forever,

Chronic berylliosis. It develops gradually. At first there are asthenic complaints: weakness, increased fatiguability, then breath catching during walks and later in rest as well, fit-like coughing, pain in chest. Some patients lose weight fast: within a short period of time patients lose from 10 to 20 kilos. At fast progressing transition, the body temperature can increase up to 39 – 40 ^{0}C with chill, grave general feeling and explicit asphyxia. Cyanosis becomes much clearer now, which gets diffusive character with time. Lymphadenopatia is observed.

During pulmonary percussion, box sound can be heard, which proves the presence of emphysema, restriction in the movements of lung end though union process in pleural cavity. In lower parts of lungs, there are small bubbling rales, rarely, they are dry and scattered; pleura friction noise is observed also.

During the research of functions of the external breathing, hypoxemia can be heard, the degree of which grows in parallel tot the severity of the case. The most characteristic is the change of diffusion ability of lungs, connected with clinical infiltration of interalveolar septums and development of alverolar and cailar block. Deficit of saturation of the arterial blood with oxygen, increase of the content of reduced hemoglobine are caused by early development of cyanosis.

In case of further development of the disease, lung hypertensia takes place with further development of cor pulmonale: tachycardia takes place, right sections of hears increase, myocardium tonic reduces. ECG shows tall wave P in II and III standard ducts, relative increase of P_{V1} wave.

Granulomatosis process can be determined in parenchimatoous organs – liver, spleen, as well as in lymphatic nodules. Often, increase and pain in liver is observed with disorder of its functioning as well as spleen expansion.

In the blood, increase of erythrocytes is observed, as well as increase of the level of total protein of blood serum, mostly due to hypergammaglobulinemai (20 – 30 g/l), as well as increase of IgC, in case of acute disease – IgA.

The listed above clinic indications are characterized by the acute berylliosis, in the period of remission, the disease does not have many syndromes. Depending on the character of radiological changes in lungs, there are two forms: interstitial and granulomatous; depending on the explicitness of the latter – stages are I,II and III.

The interstitial form is characterized by diffusive changes of lung pattern, clinically it s more non-malignant and as a rule it is restricted by stage I. The grave form is granulomatous, which is characterized by the presence of fine or large nuclear shadows (granulomas), widening of roots due to hyperplasia of lymphatic nodules, early development of pulmonary and cardiac insufficiency in the result of alverolar-capilar block, which causes the violation of oxygen diffusion.

The disease in *Stage I* is characterized by relative diffusion increase and deformation of lung pattern. The increase of radiological photograph enables to find spot shadows opf granulomas.

Patients with S*tage II* have marked deformation of lung pattern, numerous fine spotted shadows of nodules. Changes are located mostly in mean and lower portions.

In *Stage III*, further increase of the number and sizes of nodules, diffusion fibrosis, as well as emphysematous changes are observed.

Berylliosis often develops after a short period of work in contact with beryllium or after many years of the termination of contact with it.

Berylliosis can be observed among those, who are not in contact with beryllium, but live not far from beryllium production, and sometimes even at a bigger distance from it. Thus, to develop a sever form of the disease; sometimes it is enough to have a short-term contact with beryllium under conditions of its small concentrations.

Skin manifestations appear at direct action of berillium action, its vapors and aerosols. There are dermatitis with such types as contact and allergic; on the place of former microtrauma, there can be ulcers, which gradually heal. In case of permeation of undissolved compounds of beryllium into the skin, underskin granulomas appear, also fistulas are possible; sometimes it can develop for a long time (for months). Bone affection with thickening of periostis of ribs and long cortical bones

Diagnostics of berylliosis is based on the contact with beryllium, as well as characteristic clinic and radiological pattern, disproteinemia; beryllium can be found in bioenvironmental (urine); magnesium content in blood plasma is reduced, and its increased excretion with urine.

Significant diagnostic criterium, especially with granulomatous form, is positive allergic skin sampling with beryllium. Compress of 0.25 – 0.5 % water solution of $BeCl_2$ is put onto a healthy patch of shoulder skin. At positive result, scattered follicular papules appear in 8-12-20-24 hours, sometimes there are erythema or swelling (they stay from 5 to 12 days and then pigmentation is left).

Differential diagnostics of berylliosis (chronic form) is a very serious problem, as its clinic – radiological form has many similar moments. First of all, it concerns sarcoidosis, miliary tuberculosis, syndrome of Haman – Rich, silicosis and other pneumoconiosis.

In spire the fact that berylliosis is referred to metalconiosis, in the action of beryllium there are moments, which are not chatracteristics to the impact of other types of dust. It proves to be more like poison with allergic action mostly affection respiratory organs. It is considered that silicosis is a chronic disease, referred to real occupational nosologies, then the development of berylliosisis possible among people, who do not have direct contact with beryllium, and the severity of intoxication is often unadequate to the amount of poison, which permeated into the organism.

Sever cases of diseases (often lethal) can be among people, who lived not far from beryllium production (1 to 2 km away). Some cases have been described when the disease developed after a short-term contact with beryllium (20 min contact). The beginning of chronic berylliosis differs from silicosis. Patients lose weight fast, they become weak, fatigued, often do not stand any medicinal drugs, antibiotics in particular. When conduction pulmonary percussion, box sound can be heard. With auscultative methods, scattered dry and fine moist rale can be heard; physical pattern is more vivid, usually a patient has well marked asphyxia. A patient can also have fever. Good results can be observed after glucocorticosteroid therapy. In contrast to other pneumoconiosis, berylliosis can affect not only respiratory organs with various clinical manifestation, but also skin, and lymphatic apparatus; it can produce marked hepatolienal syndrome, and affect joints, what differ it much from silicosis and brings closer to sarcoidosis. There is also such a definition that berylliosis is sarcoidosis with known etiology, though its progress is milder.

Indications of berylliosis can appear in about several months, and sometimes in many years (15 and more) after the termination of any contact with beryllium. That is why it is always necessary to remember about the possibility of berylliosis development under conditions of unclear diagnosis, as well as well remember about the existence of acute berylliosis, where syndromes of rhinitis, pharyngitis, tracheitis can develop, as well as the development of bronchiolitis, and toxic pneumonia can be observed which develop for a long period of time and in severe forms with its episodes and relapses. Acute form can remind the syndrome of Haman – Rich.

To make a final decision as to the diagnostics of berylliosis can be done with the help of skin sampling of Curtis with the solution of sulfate or beryllium nitrate.

When conducting differential diagnosis of berylliosis with sarcoidosis, it is necessary to remember that for the latter, it is characteristic to have simple progressing, absence of alveolar-capillary block, large polymorphism of clinical manifestations (affection of skin, lymphatic nodules, locomotor apparatus, eyes, nervous system, heart, liver, and kidneys). Positive reaction of Quame is also very important.

As to differentiating berylliosis with the syndrome of Haman-Rich, it is necessary to take into consideration the presence of contact with beryllium, positive skin sampling, as well as results of puncture biopsy of lungs.

Exception of the diagnosis of tuberculosis is based on absence of clear symptoms of tuberculosis intoxication, negative tuberculosis testing (based on the positive Curtis testing) as well as on the results of specific test-therapy.

Numerous research of mucus to have atypical cells, results on broncioscopic research, conduct of transbronchial puncture of lung tissue, definition of the state of function of external breathing permit exclude a possible diagnosis of miliary tuberculosis.

Treatment. Treatment measures for patients with berylliosis differ much from the therapy of other types of pneumoconiosis. With acute forms of the disease, it is important to terminate any contacts with beryllium compounds. If upper respiratory tracts are affected, it is necessary to use inhalations: warm alkaline or oil ones with menthol. With acute bronciolitis and pneumonia, treatment should be complex and include drugs, which are directed at the treatment of pulmonary and cardio-vascular insufficiency (oxygen and cardiac glycosides), anti-infection means (antibiotics and sulfonamides), as well as desensitizing drugs (Dimedrol and Suprastin). In grave cases, it is necessary to use dexamethasone and prednisolone.

Autoaggressive character of changes with chronic berylliosis is the basis to use glucocorticoid drugs. When choosing the treatment scheme for patients with berylliosis, it is necessary to consider their age, concomitant diseases, as well as the disease stage. Usually, the treatment is conducted in courses, 30 to 50 days each, with 30 to 40 mg of prednisolone to start with. The dosage reduction should be done gradually. It is necessary to remember about the possibility of complications due to glucocorticoid therapy (metabolism disorder, worsening of diseases of alimentary canal, increase of arterial blood pressure, and reduction of resistance to infections.

Verification of the ability to work. Considering the quickness and fast development of berylliosis already within first days and weeks of work, as well as ability to relapses, severity of development and complications, relapsing character of skin affections and tendency to development of pulmonary pathology among these people, and with chronic or marked acute poisoning, it is necessary to restrict person from working in the conditions with beryllium and its compounds, and to provide full-time rational job.

Contraindications to continue work with beryllium can become positive skin sampling. With full-time work, all patients with marked form of berylliosis are assigned to visit doctor-expert commission (full restriction of work), as well as with mild forms – those who need to be re-qualified. Issues temporary termination of contact with berylliumcan be raised in case of isolated affection of eyes (conjunctivitis) or light affection of upper respiratory tracts.

Preventive measures. Among the measures to prevent intoxication with beryllium, it is necessary to do the following:

- to utilize protective measures and first of all to use uniform with its further treatment;
- to utilize technical devices and equipment which would reduce the risk of beryllium impact of the worker;
- to utilize effective general ventilation;
- to conduct preliminary and periodical medical examination of workers.

Pneumoconioses Caused by Mixed Dust

Pneumoconioses of this type develop in case of combined action of various types of dust. Clinic and radiological manifestations within this group are various, what depends on the composition and physical and medical properties of the dust, especially from the mixture of free silicon dioxide.

Peumoconiosis, caused by the action of mixed dust, which large amount of free silicon dioxide, are well spread and are close to silicosis on their manifestations. It can be met among workers of coalmines, iron ores, as well as ceramic and china – faience industry. Depending on the character of the mixture, the following forms are distinguished: anthracosilicosis, sidersilicosis, and silicosilicatosis.

Among pneumoconioses from the action of mixed dust with insignificant mixture of silicon dioxide, there are pneumoconiosis of electric welders, gas welders, and grinders, when radiological dust of metal depositing takes place.

Pneumoconiosis of grinders. This pneumoconiosis can be met in 7 – 14 % of cases among workers of metalworking industry among those, who work on grinding – and – polishing operations. It appears in the term from 15 to 30 years from the start of working on the profession, connected with dust impact. The disease usually progress gradually, and very rarely it get to stage II.

Clinics. The clinical pattern is characterized by symptoms of bronchitis and emphysema of lungs. Patients complain to have dyspnea, coughing, and pain in the chest. In contrast to the silicosis, coughing is observed more often with pneumoconiosis of grinders. Often, this is coughing with expectoration. At objective examination, as a rule, lung emphysema is observed (primary moderately expressed) and more often than with silicosis, dry and sometimes bubbling rales can be heard.

Thus, pneumoconiosis of grinders is more often manifested with bronchitis (and perhaps in some cases it goes before pneumoconiosis or accompanies it). Workers on damp grinding often have the diagnosis of inflammatory disease of respiratory tracts, caused by inhaling small drops of liquid, used when grinding (petroleum oils and their emulsions, alkaline solutions, kerosene, etc). But the dust concentration with damp grinding is smaller, thus pneumoconiosis develops more rarely.

Radiological and morphological pattern among patients with pneumoconiosis is characterized by diffusive interstitial fibrosis with main localization in lower and mean zones of lugs. Fine nodular forms of fibrosis can be met much more rarely.

Pneumoconiosis of grinders can be rarely complicated with tuberculosis (3.1 %) and generally it is characterized by non-malignancy of the progress.
Forecast of this pneumoconiosis is mostly connected with bronchitis development, how marked emphysema of lungs is and the complications of a non-specific infection.

Pneumoconiosis of electric welders. Prolonged inhaling of multi-component of electric welding aerosol can condition the development of pneumoconiosis, which refers to pneumoconiosis from inhaling mixed dust with insignificant among of free silicon dioxide and iron and has some clinical and radiological peculiarities.

Pathologic and anatomic pattern. For pneumoconiosis of electric welders, a morphological substrate is interstitial fibrosis with relatively small accumulation of dust. In lungs, sclerosis is present with dust depositing, and thickening of alveolar septums; in lymphatic glands – dust, and development of conjunctive tissue, which in contrast to silicosis, does not cover the total territory of a lymphatic nodule. It is not characteristic for it to have nodules like with silicosis.

Clinics. The disease appears after a long period of work as an electric welder (in average 15 – 16 years). When working in closed premises, pneumoconiosis can develop within shorter period of time (up to 5 years). On early stages of the disease in most cases, complaints are absent or unclear (some dyspnea after physical activity and rare dry rale). The results of physical examination are rather insignificant too. General state is rather satisfactory as a rule; cyanosis is not cleat; chest form is not changed. In some cases, there is percussion sound with moderate box sounds in lower lateral portions of the chest. Some dry rales are non-permanent. When doing physical examination, the majority of ventilation indications are not changed: in some cases there is some increase of the amount of residual air in compliance with moderate emphysema of lungs.

On the radiological pattern of lungs. the primary period of the disease shows diffusive intensification and deformation of lung pattern. Then, there is spread porous looping, more marked in mean and lower portions of lung fields. On the background there are round formations, which differ by fine sizes, sharp outline and increased intensity of shadows. Absence of the tendency to merge these shadow formations is characteristic. as can be observed with nodular form of silicosis, mostly clusters of radiologically contrasted dust, which contains iron, and in a less amount, it is conditioned by finely popular shadows, and by the development of conjunctive tissue. Dynamic observations prove non-malignant progress of pneumoconiosis of electric welders. As a rule, progressing of the disease to stage III is not observed.

Complication of pneumoconiosis with pulmonary tuberculosis can be met much more seldom, than with silicosis and mostly nuclear forms of tuberculosis are observed. In some cases there is regressing development of the process, connected with graduate emptying of lungs from radiologically contrast dust. Combination of pneumoconiosis of electric welders with chronic bronchitis worsens significantly the disease development. In such cases, clinical pattern of the disease depends on the activity of non-specific infection, as well as clear obstruction of bronchi and lung emphysema.

Treatment. Patients with stage I of pneumoconiosis, especially if it is not complicated, does not need specific treatment. They are recommended to have general means of to increase the resistance of the body to the infection, balanced meal, physical exercises, as well as keeping to a work/leisure regime. In case of complication of pneumoconiosis with bronchitis, lung insufficiency, and possible bronchial asthma, a corresponding treatment is carried out.

Verification of the ability to work. Electric welders with pneumoconiosis of stage I with the absence of clinical symptoms can continue working under attentive observation of doctors, but it is necessary to restrict their work in narrow restricted premises. At stage II of pneumoconiosis, in case of its combination with chronic bronchitis, emphysema or tuberculosis, the work as an electric welder is contra-indicated.

Preventive measures. Prevention of pneumoconiosis of electric welders is provided by the improvement of technological process (replacing arc welding with contact one and utilization of special welding machines); provision of effective ventilation; utilization of means of individual protection of respiratory tracts and eyes; as well as medical examination of workers.

Pneumoconiosis Caused by Organic Dust

Occupational diseases of lungs, conditioned by the impact of organic dust, can be referred to pneumoconiosis conditionally, as not all of them progress with the development of diffusive pneumofibrosis. Thus, with biocenosis, which is caused by the action of dust of herbal fibers (cotton, flax and hemp), there are mostly functional disorders of bronchial permeability, sometimes joined with bronchiatic syndrome.

In the result of dust action of grain, flour, tobacco and some types of plastics, there can be changes in lungs with moderate diffusive fibrosis, accompanied by general or allergic reaction.

Biosinosis is an occupational disease among people, who were under the impact of organic fibrous materials for a long time (cotton, flax and hemp).

The main hazard at ginneries and cotton-spinning factories, as well as flax mills is the dust of complex composition, which contains organic and mineral fractions.

With preparatory operation on treating and processing cotton, flax and other fibrous materials, especially when processing coarse low quality raw materials, the dust can contain 20 % and more of free silicon dioxide at the expense of ground contamination. Such dust is silicon hazardous. However, at the majority of textile mills, dust includes, as a rule, only matters of organic origin. It can be contaminated with bacteria and fungi (mostly mold). The permitted concentration for dust of herbal and animal origin (grain, cotton, wool, down, etc) with a mixtures of silicon dioxide is as follows: a) over 10 % - 2 mg/m^3; b) from 2 to 10 % - 4 mg/m^3; and c) less than 2 % - 6 mg/m^3.

Pathogenesis. In the basis of clinical pattern of biocenosis, there are disorders of bronchial permeability, which obviously have double origin. On the one hand, they are the results in direct action of agents, which narrow bronchi. These agents are contained in the dust of flax and cotton. Besides, under prolonged impact of organic dust, another mechanism turns on: in bronchial wall, matters are deposited, which are withdrawn during the next contact with dust, and also create bronchial and obstructive, but slower action. Subjectively, this is accepted as breathing obstruction, feeling of pressure and pain in the chest. Specific role in forming biocenosis is played by matters of protein origin, which are capable to sensibilize the body of workers: histamine, etc, contained in organic dust, as well as fungi and bacteria, which contaminate it.

Clinic. Biocenosis symptoms, which appear in several years under work conditions of high concentration of dust, are very characteristics. Patients complain that they feel squeezing, pressure and pain in chest, obstructed breathing, and dyspnea at physical pressure, dray rales and weakness. At first the mentioned disorders appear only when renewing work after a break ("Monday symptom"). With time, they do not stop during workdays too, and then can become permanent. In the basis of subjective symptoms of biocenosis was the peculiar dynamics of disorders of bronchial permeability; it is mostly expressive on those days when after a break, contact with industrial dust takes place again. Repeating themselves, these disorders cause the development of cardiac pathology of bronchi and pulmonary apparatus and cardiopulmonary decompensation, combine with other diseases.

Biocenosis progress is divided into the following stages:

Stage I. When renewing work after a break, complicated breathing, squeezing in chest, coughing and weakness are observed. In the majority of patients, auscultative symptoms are absent; sometimes there are physical indications of initial emphysema and bronchitis.

Stage II. Dyspnea and coughing become more expressive and take place during work, but Mondays are still "one of the hardest days". Expectoration, either mucous or pus-mucous, appears. Dyspnea can be significant, sometimes becoming characteristic asthmatic fits. As a rule, there are clinical and radiological indications of bronchitis and emphysema of obstructive and restrictive type.

Stage III. Light gaps disappear; subjective disorders take place throughout the whole week, beyond work place as well. Objective symptoms include chronic bronchitis, lung emphysema, and sometimes bronchial asthma. Ventilation disorders take place; pulmonary-vascular decompensation is observed. Radiologically, indications of emphysema, Carnification of lung roots and intensification of lung pattern are observed.

There are a number of changes in the organism besides respiratory system, like arterial hypertensia, dyspeptic phenomena, scent depression (among

workers of flax mills), atrophic rhinitis, laryngitis and dermatitis. Skin tests with the extract of production dust are positive.

Treatment. It is done in compliance with the general principles of pneumoconiosis treatment. In case of complication of biocenosis with inflammatory process, antibiotics are prescribed; and with the development of cor pulmonale – cardiac glycosides and diuretic are prescribed.

Preventive measures. Measures to prevent biocenosis development are similar to those, which are carried out to prevent silicosis.

DUST BRONCHITIS

Dust bronchitis is one of the occupational diseases, caused by prolonged impact of production dust. It is characterized by diffusive inflammation of bronchi and is an initially chronic diffusive endobronchitis.

As a nosological form, dust bronchitis was enlisted in the list of occupational diseases in 1970.

Under modern conditions, dust bronchitis develop rather slowly, in 8 to 10 years of work under dust impact, and can be met in various industries in Ukraine (table 4).

Table 4

List of industries, potentially hazard for the development of dust bronchitis (Yu.Kundiev and O.Krasnyuk)

Production, Occupation	Factors, which cause bronchitis development	Other Possible Forms of Dust Pathology
1	**2**	**3**
Coal industry (workers of face factories, tunnellers, drivers of coal coal-plough machines)	Dust, containing quartz and coal; irritating gases (methane); hot microclimate and high humidity	Pneumoconiosis
Mining industry (borers, tunnellers and shot-firer)	Dust, containing quartz	Pneumoconiosis, bronchial asthma
Machine engineering industry		
foundry (founders, cutters and formers)	Dust, containing free silicon dioxide, metal aerosol, irritating gases (sulfuric anhydride); hot microclimate; and temperature difference	Pneumoconiosis
welding production (electric and oxy-acetylene welding, cutter	Aerosols of metals, gases (nitrogen oxide and anhydrous hydrogen fluoride)	Pneumoconiosis, bronchial asthma, exogenic allergic alveolitis
Construction materials and construct industry (concrete mixers, borers and shot-firers)	Abrasive dust, metal dust	Pneumoconiosis, bronchial asthma
Textile industry and	Dust with free and bound	Biocenosis, bronchial

preliminary processing of fibrous technical cultures (operators of hackling machines and units, raw material sorters)	silicon dioxide	asthma
Plant growing (tractor and combine drivers)	Fibrous herbal dust, fungi and bacteria insemination	Pneumoconiosis, bronchial asthma
Cattle breeding (operators of poultry farms and animal farms)	Ground, plant gases, mineral fertilizers, and pesticides.	Bronchial asthma, exogenic allergic alveolitis
Feed production (operators and shift men)	Dust of herbal origin, biologically active matters (microelelents), fungi and bacterial insemination	Bronchial asthma, exogenic allergic alveolitis
Bakery (elevator operators, millers and bakers)	Grain and flour dust, fungi insemination	Bronchial asthma, exogenic allergic alveolitis
Tobacco production (sorters of leaf tobacco, operators of cigar and cigarette machines)	Tobacco dust	Bronchial asthma

Etiology. The development of dust bronchitis depends in prolonged inhaling of much dispersed dust with small amount of quartz or even without it (cement, herbal, or wooden dust).

The appearance of disease is influenced by the presence of unfavorable conditions of production components: work conditions (microclimate, heavy work, or noise); a number of unprofessional factors (sex, age, smoking, infection in the past, or diseases of upper respiratory tracts).

Pathogenesis. In case of action of dust onto the body, disorder of some systems of protection of bronchi-pulmonary apparatus is observed, like mucociliary transportation, local immunity, and surfactant system. There are disorders of evacuation of dust portions and secretion function of bronchi on the background of structural changes of ciliary epithelia.

Dust bronchitis is characterized by atrophic and sclerotic changes in all the structures of bronchial tree, which form on the initial basis of the disease already, also by changes of bronchi motor activity, and hypersecretion.

In the pathogenesis of the disease, bronchospasm is very important. It appears in the result of reflector reaction of bronchial muscles onto dust particles or sensitization to allergens, contained in the industrial aerosol (chromium, manganese, nickel, phenol-formaldehyde resins, etc). Pathogenic microflora of respiratory tracts influences the development of the inflammatory process in bronchi and allergization of the patient.

At this disease, decrease of cell and humoral immunity decreases also, and significant meaning is also possessed by some genetic factors, in particular deficit of β_1-inhibitor of the protease.

Pathologic and anatomic pattern. At dust bronchitis, bronchi, bronchial tubes and alveoli are affected. The action of dust first causes relevant reaction from the side of mucous tunic in the form of bronchi hypersection. Number of goblet cells increases. Reological properties of mucus change, its viscosity are increased. Then cells of ciliary epithelia die, basal membrane, infiltration with lymphoid cells starts. This period is clinically determined as endobronchitis, or dust catarrh.

With time, endobronchitis transits into panbronchitis, and then into peribronchitis. Centers of infections in perobroncial cell are accompanied by perineal sclerosis and transition of inflammatory changes into parenchyma of lungs. This phase of reamed inflammation, which is along formation of various grades of sclerosis expression and obliteration of fine bronchi transits into the third stage – reconstruction.

Thus, evolution of chronic bronchitis can be presented by consecutive pattern of hyperthrophic changes of bronchi with atrophic ones with further development of catarrhal mural deforming bronchitis. Spreading of inflammatory changes in distal sections of bronchial tree is accompanied by the violation of production of surface active matter – surfactant, which causes the development of bronchospasm and assists the appearance of sever complication – obstructive emphysema of lungs.

Clinics. Modern classification of dust bronchitis envisages the evaluation of the stage, period (phase) of the disease, as well as presence of complications. There are three stages of dust bronchitis:

Stage I: irritation is weakly marked bronchitis. After many years of work in contact with industrial dust, there is dry rales with some mucus appears. Gradually with years, it becomes stronger; dyspnea appears, as well as general worsening of health state. Worsening of the disease is rare and does not last long.

During objective examination of the patient, there is clear pulmonary sound with box hint, mostly ion lower portions of the chest. According to auscultative examination, breathing is coarse, dry and, sometimes, bubbling rales can be heard. No significant radiological changes are observed. Pulmonary insufficiency is absent, and within the period of acuteness corresponds to 0-1 degree. Changes in periphery blood are absent.

Bronchitis at this stage has clear phases: relapses or exacerbation. Under conditions of timely treatment and normalization of work conditions and the life method, this process is rarely reverse.

Stage II: inflammatory. Clinic manifestations of dust bronchitis in this stage of the disease are conditioned by a variant of bronchitis progress: obstructive, asthmatic, and inflammatory, which mostly depends on etiological factor. Thus, under the action of mostly quartz dust, as a rule obstructive bronchitis with lightly marked inflammation develops with fast developing emphysema of lung with obstructive genesis. Miners, electric welders, workers

who are in contact with organic dust; mostly asthmatic variant of dust bronchitis develop. Presence of toxic components (metal oxide, formaldehyde, or sulphuric compounds) assists the development of bronchitis with manifestation of infectious process in bronchi, which reminds chronic toxic bronchi with the development of bronhcoectases and pneumosclerosis. In such cases, frequent exacerbations are observed, with secretion of mucopurulent or purulent sputum, as well as corresponding changes of indications of clinical and biochemical blood analysis.

After the radiological examination, slow intensification of lung pattern, which at the period of exacerbation becomes more marked. Pulmonary decompensation of I or II degree. Initial symptoms of cor pulmonale can be observed. Exacerbations are longer (2 to 3 weeks), and their frequency is up to three times a year.

Thus, bronchitis of II degree enables to determine one variant of progress or another, as a rule, it is complicated bronchitis, which is not reverse in full, in spite of rather intensive treatment.

Stage III – deep bronchitis. Clinical manifestations in this stage are characterized by stable coughing with mucus, dyspnea in rest and pain in chest. During the examination, it is observed that patients are cyanotic, and have hydropic face. Percussion examination shows box sound above lungs, and after auscultative examination – big number of scattered dry and bubbling mixed crepitations can be heard.

Radiological changes of II degree are characterized by marked intensification and deformation of lung pattern (as an indication of dust impact), as well as indications of emphysema. Pulmonary decompensation and cor pulmonale are well marked. Exacerbations are frequent and relapses are rather short, and sometimes are even absent.

Thus, dust bronchitis in this stage is a complicated bronchitis with rather grave progress, for which it is characteristic to combine several syndromes (inflammatory and obstructive or inflammatory and emphysema). The most frequent and grave complication is formation of cor pulmonale with the development of cardiopulmonary decompensation, which leads to the loss of work ability by the patient.

Depending on clinical peculiarities of the disease, there are the following forms of dust bronchitis: emphysematous, bronchospasmodic and inflammatory forms of dust bronchitis.

Emphysematous form is characterized by dyspnea of various degrees, coughing with insignificant mucus, which expectorates hard. Objectively, indications of emphysema, coarse breathing and dry crepitations. Radiologically, it is possible to determine increased transparence of lung fields, restriction of movement and low condition of domes of the diaphragm, as well as increase of bronchial vascular pattern. The function of external breathing is changed after the restrictive type within I-II degrees. On the side of cardio-

vascular system, presence of cor pulmonale in the stage of compensation, and more seldom – with the insufficiency of blood circulation of I-II degree are possible.

Stage II of bronchitis is particularly marked. Patients complain to have dyspnea in rest, discharge of small among of mostly phlegm. Objectively, acrocyanosis and emphysematous form of chest are found, as well as lower portions of lungs is lowered. There are symptoms of cor pulmonale with insufficient blood circulation of II and III stages with obstructive restrictive type. In the peripheral blood, there might be inflammatory changes.

In case of overbalance of asthmoid syndrome, in stage I of bronchitis, some breathing obstruction is observed, which appears periodically during the contact with the dust factor and changes of weather conditions. Physical changes are insignificant in this case: during percussion examination, lung sound can be heard with some dry crepitations, in particular during forced breathing when the patient is in horizontal position. In the peripheral blood, eosinophile is found. As to the functions of external breathing there can be I^{st} degree disorders of obstructive type. During stage II– periodical fits of dyspnea, as well as asphyxia increase. Over the lungs, there is box sound in basal portions, as well as coarse breathing, exhaling is prolonged, and crepitations are dry and whistling. In the blood, eosinophile is increased, and in the prhlegm, there are elements, characteristic for bronchial asthma. As to the cardio-vascular system, there are indications of cor pulmonale in the stage of compensation or with insufficient blood circulation of I^{st} degree; the function of external respiration – the reduction of within I-II degrees of obstructive type. Clinical pattern of stage III reminds the secondary bronchial asthma. Present frequent fits of asphyxia, and dyspnea in rest, coughing, expectoration is difficult to come out. There is emphysema of lungs, big number of dry crepitations. In the blood and phlegm testing – sings of allergic component. Cor pulmonale with insufficient blood circulation diagnosis is often to be made.

In case of overbalancing of inflammatory process in bronchi – clinical pattern of bronchi reminds common infectious bronchitis. Characteristic indications of the version are coughing with phlegm. At the initial stage, the latter has mucous, then mucopurulent and purulent character, increased temperature, fatiguability and increased hyperhidrosis. In lungs on the background of box shade of percussion sound, there are dry, and further crepitations. There might be areas of muted percussion sound. In the periphery blood, there is neutrophyl leukocytosis and ESR increase. Function of external respiration is decreased.

Diagnostics. Diagnostics of dust bronchitis is carried out in two stages: at first, it is envisaged to make a diagnosis of the chronic bronchitis as nosological form and definition of the degree if its severity, then dust etiology of bronchitis and thus occupational category of the disease.

The first stage of the diagnostic process is based n the record of clinical manifestations of the disease (anamnestic data, patient's complaints, results on physical examination) and auxiliary methods of research (functional, radiological and endoscopic ones).

When deciding on referring chronic bronchitis to an occupational disease, it is necessary to follow the following criteria:

1. Presence of sufficient stage of work under conditions of dust action (7 to 10 years and more). Work period under dust conditions should be supported by a corresponding written in a work record book of the patient.
2. Unfavorable conditions of work, supported by sanitary and hygienic characteristic (presence of dust on a work place with mentioning its concentration and composition), irritating gases, unfavorable microclimatic factors – changes of temperature, humidity and hard physical work).
3. Peculiarities of the development of chronic bronchitis – beginning and character of the disease development, presence of carried disease, especially pneumonia, described and proved by the extract from the out-patient record book of the patient.

Dust etiology of bronchitis is rather easily stated in the case when there is explicit development of the disease of bronchial and pulmonary system under conditions of the dust factor. Anamnesis indications of frequent diseases of bronchial and pulmonary system and much smoking can make the process of stating the professional character of the disease more complicated. But it is necessary to remember that in case of long work period, connected with the action of the production dust, in spite of the previous acute infections of respiratory breathing, it is difficult to exclude the impact of dust onto the development of chronic bronchitis. In case when a worker, whose past has an indication of acute bronchitis and pneumonia, but at the time of getting a job, which deals with dust factor, he/she was considered as healthy (what is indicated by a corresponding record), then was considered healthy and only in some time, he/she got chronic bronchitis, then this disease should be considered occupational as well.

When chronic bronchitis is a direct outcome of acute infection disease of respiratory organs, the issue on the connection of chronic bronchitis with the work conditions is solved individually first of all, with the consideration of work conditions and work period of the patient. Very often it is necessary to exclude unfavorable impact of production factors onto the development of the disease, which will enable to say about a joint genesis of hazard factors, first of all, dust and infections. In this case, they say about chronic bronchitis of the joint genesis (dust infection).

Based on complaints of the patient, changes, found during clinical examination, as well as presence of data from the listed above official

documents, and differential approach diagnosis of dust bronchitis can be made. In compliance with the traditional clinical classification of dust bronchitis in making the diagnosis after nosologic form of “dust bronchitis” in compliance with the stage (I, II and III) show clinical syndrome, which prevails in the clinical pattern (inflammatory, asthmatic, and lung emphysema) and its explicitness, as well as the degree of luminary and heart decompensation (table 5).

Table 5

Classification of dust bronchitis

Disease stages	Clinical syndromes			Functional disorders	
	Asthmatic	Inflammatory	Lung emphysema	Pulmonary decompen-sation, degree	Cardiac decompen-sation, phase
I	Unclearly manifested	Exacerbation frequency (frequent, rare)	Initial manifestation (degree I)	I	Compensations
II	Moderately manifested	Bronchio-extases	Moderately manifested (degree II)	II	Subcompensations
III	Manifested	Periphocal pneumonia	Manifested (degree III)	III	Decompensations

To clarify the degree of lung decompensation by following the traditions of deviation of indications of external respiration from the norm (Table 6).

Table 6

Limits of norms and gradation of deprivation of indications of external respiration from the norm (M.M.Kanayev)

Indicator	Norm	Conditional norm	Derivation		
			moderate	significant	acute
GEL, %	> 90	90-85	84-70	69-50	< 50
Proper MVL, %	> 85	85-75	74-55	54-35	< 35
Proper OFV_1, %	> 85	85-75	74-55	54-35	< 35
Proper OFV_1/ GEL	> 70	70-65	64-55	54-40	< 40
MVL/GEL	< 28	28-30	31-33	34-40	> 40
	> 22	22-20	19-16	15-10	< 10
ZEL, %	< 100	110-115	118-125	126-140	> 140
Proper ZOL, %	> 90	90-85	84-75	74-60	< 60
	< 125	125-140	141-175	176-225	> 225
Proper ZOL/ ZEL, %	+ 5	(+5) — (+8)	(+9) — (+15)	(+16) — (+25)	> (+25)

Note: GEL – lung vital capacity, MVL – maximal ventilation of lungs, OFV_1 – volume of forced echalation per second; OFV1/GEL – Tiffano testing; ZEL – total capacity of lungs; and ZOL – residual capacity of lungs

When diagnosing dust bronchitis, it is necessary to determine the process activity. Dust bronchitis develops with periodic exacerbations, with which its progressing is connected, but these exacerbations do not manifest with clear

indications usually, what shows the activity of pathological process. Such generally accepted indicators f the activity of the inflammatory process, as body temperature, ESR, number of leukocytes, leukocytic blood formula, biochemical indicators (C-reactive protein, sialic acids, sulfur-mucoid, as well as haptoglobin), and during acute condition of bronchitis, they can be very unclear. That is why during the period of exacerbation, it is necessary to pay particular attention to the changes of clinical manifestation of the sick, reduction of their ability to work, sign of brocnhospasms, weakness, increased hidrosis, increased coughing, signs of bronchospasms, and appearing of mucopurulent sputum show the exacerbation of dust bronchitis.

With dust bronchitis, there is not always an opportunity to consider the dynamics of the process in two opposite phases – exacerbation and remission. Often after massive course of treatment in hospital, patients are released with some indications of delayed exacerbation. This condition should be considered as the stage of fading exacerbation, which envisages corresponding recommendations regarding the following outpatient treatment and regime. Sometimes, patients have clear clinical exacerbations, however some objective sings of bronchitis are increased (a threat of exacerbation). Corresponding job, and out-patient treatment) can prevent the appearance of exacerbations and loss of ability to work.

Thus, diagnostics of dust bronchitis is based on thorough recording of results of clinical and assisting research, career data and data on labor conditions.

Treatment. Tactics of treatment of dust bronchitis is based on the results of examination of patients and is conditioned by mostly symptoms, functional state of the external respiration, blood circulation, nervous and other systems, presence and explicitness of an allergic component, as well as the state of immune reaction.

Considering that at dust bronchitis, there is the development of atrophic processes in mucous tunic of the bronchial tree on the first stages of the disease; main treatment should be directed at the increase of general reactivity of the organism, stimulation of general regenerative processes in the mucous tunic of bronchi, as well as liquidation of bronchospasms.

First of all, it is necessary to tell about the utilization of means, which stimulate processes of epitelization. Such properties are possessed by methyl-uracyl, which is given in the dose of 1 g 3-4 times a day after meals. It is also possible to prescribe 4 % solution of calcium pantothenate, which is given in the form of 4 % aerosol inhalation – 10 ml every day. The course consists of 10 to 12 inhalations.

Patients, who mostly have bronchospasms, are prescribed sympathomimetic agents: isadrin and novodrin, which are taken in the form of aerosols. Of some advantage are medicinal drugs of the same group in small dosated tanks: asthmopent, alupent, and berotek.

Optimal dose for inhalation is two inhaling, which are repeated 4 to 5 times a day. When treating patients with dust bronchitis with disorders of bronchial permeability, aminophylline is widely used – 2.4 % IV solution 10 ml dose.

When treating patients with dust bronchitis, medicinal drugs are used which improve phlegm discharge, like althaea root, termopsis herb, and potassium iodide, as well as means which have mucous solving action, like mucous solving inhalations and sodium chloride.

Considering the important role of an allergic component in the development of bronchitis, patients with this pathology are prescribed to use antihistamine products, like Dimedrol, diazoline, and phencarol.

When treating patients with inflammatory version of dust bronchitis, main place is occupied by medicinal drug therapy, aimed at liquidation of inflammatory process and prevention of the process transfer into a chronic form. Infectious etiology of the given form of the disease is conditioned by utilization of a corresponding therapy (antibiotics, sulfanilamide, etc) with simultaneous conduct of measures to increase protective immunity of the body.

The most well spread medicinal drug to treat patients with inflammation, caused by pneumoconiosis and streptococcus is penicillin. One-time dose is 300 000 – 600 000 OD every 3 to 4 hours, or 2 000 000 – 5 000 000 OD a day. Medicinal drugs of the group of semisynthetic penicillins, like ampicillin and oxacillin are widely used (daily dose is up to 4 g, 0.5 g – 4 to 6 times a day, one or up to three hours before meals).

Out of sulfanamide medicinal drugs, sulfalen and sulfadoetoxia are prescribed (the first day – up to 2 g, and then 1 g for 8 to 10 days).

As preventive measures against dysbacteriosis, Nystatin is prescribed: 2 000 000 – 4 000 000 OD a day.

An important role in treating diseases of lungs are played by endobronchial sanitations with introduction of necessary medicinal drugs.

Verification of the ability to work. Issues of the verification of the ability to work for those, who have dust bronchitis, are solved individually, with the consideration of the severity of the disease, age, work period, occupation of the sick and work conditions.

The patient with dust bronchitis of stage I can continue working in the competence of his/her occupation with obligatory dynamic medical examinations (not less than twice a year).

Patients with bronchitis of stage II are subject to rational employment, which is not connected with the influence of dust, irritating matters, and unfavorable weather factors as well as without significant pressure. The decrease of qualification at transfer to another job is the basis to send the patient to the Treatment and technical verification commission to get disablement status (as a rule, it is group III) due to the occupational disease.

Patients with stage III of bronchitis due to frequent cases of exacerbation of inflammatory process and the development of cardiac and pulmonary decompensation lose the workability completely and often need external help, what conditions the need for them to get disablement group II or I in the result of the occupational disease.

Preventive measures. Main preventive measures of bronchitis is the conduct of technical, sanitary and hygienic measures, aimed at further improvement of the work environment for workers of dusty professions.

Measures of medical preventive measures, first of all, a quality conduct of medical examinations, both preventive and periodical, are very important. Another important preventive measure for dust bronchitis is timely and rather long treatment of acute inflammatory diseases of respiratory organs, active anti-smoking campaign.

OCCUPATIONAL BRONCHIAL ASTHMA

Occupational bronchial asthma is the disease, main manifestation of which include fits of asphyxia, conditioned by bronchospasms, hypersecretion of bronchial glands, swelling of mucous tonic of bronchi, and which is etiologically connected with the action onto the bronchial apparatus by the agents on the workplace of a worker. Thus, occupational bronchial asthma, which is observed under various production conditions, is etiologically connected with the impact of occupational factors.

At the meeting of the WHO in Geneva in 1980, bronchial asthma was listed among other occupational diseases. Whereas it was stressed that the main criterion to recognize occupational etiology of bronchial asthma is the presence of connection of its appearance with the work conducted.

Some epidemiological research showed that 2 to 14 % of all the patients with asthma suffer from professional bronchial asthma. The frequency of professional bronchial asthma much varies in various occupational groups. Thus, it is considered that among those farmers who contact with animals and birds, about 6 % of the people have asthma, and as to those who work in bakeries – about 10 % have asthma.

Etiology. In the etiology of occupation bronchial asthma, an important role is played by the following matters: allergic agent of animal (wool, silk, hair, feather, pieces of epidermis, bees and helminthes) and plant (pollen of herbs, bushes, trees, flowers, wooden, grain and flour dust, volatile oil, flax and tobacco) origin; a large number of chemical matters (Ursol, metal compounds – chromium, nickel, cobalt, manganese; formalin synthetic polymers, dyes, and pesticides); medicinal drugs (hormones, vaccines, ferments, protein and vitamin concentrates, as well as forage antibiotics). Among medicinal drugs, the most important are antibiotics (especially penicillin, more seldom – streptomycin, biomycin, and tetracycline), as well as vitamins, sulfanilamide, analgetics, hormonal drugs and aminazine.

In principle, etiological factors can be divided into the two following groups: allergic agents and asthmogenic agents. The former include flour, especially, wheat flour, natural silk, epidermis of animal fur and skin, castor oil; dust of green coffee beans; detergents; and various medicinal drugs. The latter one includes wooden dust, cotton, PVC, lacquers, pesticides and phenol.

Pathogenesis. In case of contacts of a worker with occupational allergic agents, int the body there is the increase production of antibodies of class IgE. the latter are fixed on mast cells (immune stage), after what degranulation of mast cells with the discharge of a great number of bronchospastic and vasoactive matters - histamine and serotonin (pathochemical stage) takes place. Under the impact of biologically active matters, permeability of microcirculatory flow is increased; swellings, severe inflammation and bronchospasm (pathophysisological stage) develop. Clinically, this is manifested by the disorder of bronchial permeability as well as the development of fits of bronchial asthma. This is a so-called atopic occupational bronchial asthma, in the genesis of which reagent type of immediate hybersensitivity takes place.

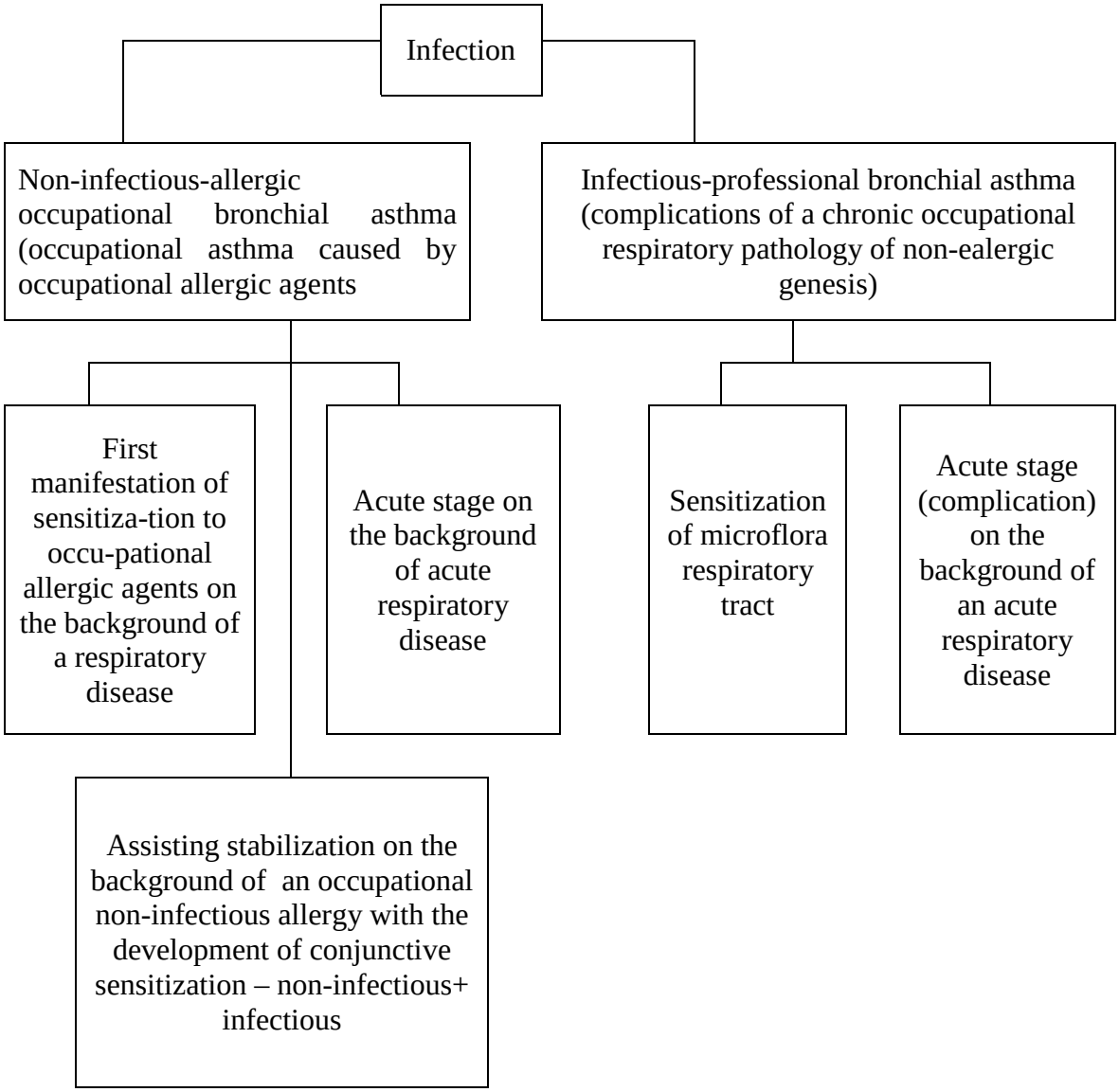
Prolonged impact of astmogenic agents causes changes in the reactivity of target cells (first of all, mast cells, located along the respiratory tract). Change of reactivity of these cells is first of all accompanied by excessive production of biologically active matters (histamine and leukotriene). In the response, bronchial spasms, swelling of mucous tunic, and hypersecretion of bronchial glands develop. All these change the permeability of bronchi much and cause asphyxia fit.

An important meaning in the development of professional bronchial asthma is also caused by heredity and genetics, as well hormonal disorders, misbalance of vegetative nervous system, and respiratory infections. As to the last factor, it is considered that there are several variants of the interaction of allergy and infection: as to the first – infection in he bronchial tree causes formation of bacterial allergy, which causes asphyxia; as to the other one – infection improves the permeability in the tissue of infectious allergic agents, and as to the third one in the opposite– sensitization of the organism is an infection “conductor”. The development of infectious – inflammatory process in bronchial tree of those who have occupational bronchial asthma are assisted by atrophic processes in the mucous tunic (result of the contamination of the production environment with matters of irritating action: solvents, acids, alkaline, vapors and gases of various toxic matters). This is manifested by intensified hemorrhage of tissue metabolites and stimulates the production of autoantibodies. The same is caused by sensitization of organism to agents of infectious processes in the bronchial tree.

Along the intensification of infectious and allergic component on the background of the occupational allergy in the pathogenetic process, besides the reagent type of allergy, there are other types of allergic reactions. In the blood, the number of circulating immune complexes is growing (joining of industrial

and infectious allergy agents with antibodies of IgA class). These complexes activate the system of complement, and in the result, the pathologic process includes kinin, neutrophilic and macrophage, as well as some other systems. Pathological process develops with more severity with asthmatic exacerbations. This very joining of infectious and allergic component causes the development of hypersensitivity of slow type (there are chemical and toxic factors of lymphocytes, and T-factors are activated). Thus, pathogenesis of the given version of the occupational bronchial asthma is close to infectious-allergic one.

Possible variants of the participation of the infection in the development and formation of a specific pattern of bronchial asthma in the clinics of professional diseases are shown in the following scheme:



Pathologic and anatomic pattern. People, who died from asphyxia, had swollen lungs. Bronchitis has viscous glass like content, with a big number of eosinophilic granulocytes, Kurchman's spirals, and Charco-Layden crystals.

Eosinophilic infiltration of bronchi walls and the thickening of the basal membrane of their mucous tunic take place. Formation of perivascular infiltrates with mononuclear cellular profile and granulation changes in the mucous tunic of the perivascular genesis should be determined as a specific peculiarity of the occupational asthma.

Classification. There are two main forms of bronchial asthma:

- occupational bronchial asthma, which is very much like atopic asthma; and
- occupational bronchial asthma of the joined sensitization (occupational and bacterial allergic agents).

In comparison with the general clinics, these are correspondingly: atopic (immune) and infection-dependant (non-immune) bronchial asthma.

Besides, it is necessary to take into consideration the gravity degree (light, mild and severe), progress phase (acute state, fading acute state and remission) and complications (lung emphysema, pulmonary collapse, pneumothorax, miocarda myocardium dystrophy, cor pulmonare, cardio-pulmonary decompensation, etc).

Clinic. Main clinical manifestations of the occupational bronchial asthma is a fit of asphyxia. Mostly it appears at night. The beginning of it is the feeling of stuffed nose, coughing and complicated breathing. Inhaling and especially exhaling are obstructed. Abdominal muscle tensing is observed. Breathing is noisy and accompanied by distant buzzing and whistling crepitations. the patient has to take a specific position (sitting and supporting himself/herself with arms), where shoulder belt is fixed: shoulders are moved up and forward, the head is like drawn into the shoulders, the chest is in the aspiratory state, and movement are limited. Cyanosis of lips, and the top of the nose is observed.

Above the lungs, there can be hears box percussion sound. And after auscultative examination, breathing is coarse with prolonged exhaling. Dry buzzing and whistling crepitations can be heard.

Pulse is frequent, heart flattening is not determined (the result of the emphysema presence), and the body temperature is normal or increased. On the ECG in the IInd and IIIrd standard portions, more pointing waive is observed.

The fit is over with the discharge of viscous phlegm of gray color and the renewal of normal breathing.

As to the frequency and expression of asphyxia or asthmatic syndrome fits, as well as the respiratory compromise, complications and the disease character, the progress of the occupational bronchial asthma can be divided into light, mild and severe.

At the light progressing of the occupational bronchial asthma, asphyxia fits are rare (1 to 2 times a month or less), they last for several minutes to half an hour, and usually they are light on the background of pleuroral administration of bronchiolitic means. Sings of worsening of bronchial permeability appear in during mild or significant physical activity, sometimes, on the background there

are light whistling crepitations, coughing or asthmatic fits. In-time termination of the contact with the professional allergic agent, as a rule, leads to clinic convalescence.

Light disease progressing is characteristic to occupational bronchial asthma, which is like atopic one, as after the timely termination of the contact with production factors, remission takes place. With these forms of the disease, the development of emphysema can be observed though a comparatively long period (8 to 9 years), and some patients can have periodical light asphyxia fits, caused by the action of sharp odors, as well as physical and emotional tension.

For the *mild* disease progress, it is characteristic to have asphyxia twice or three times a week, which last for one hour (rarely, more); they terminate after an injection or aerosol inhalation. Between the fits, patients can have periodical crepitations in the chest and some complicated breathing. Worsening of the state is observed with moderate or insignificant physical activity. Termination of the contact with an occupational allergic agent is accompanied by significant improvement – typical fits of asthmatic state terminate, though expiratory dyspnea and coughing appear periodically.

Severe disease progressing is characterized by the appearance of frequent, often daily fits of asphyxia, up to the development of asthmatic state. Complicated breathing takes place during insignificant physical activity. To receive therapeutic effect, there is the necessity to use corticosteroid hormones. Termination of the contact with the production is not accompanied by the improvement of the patient's state.

Mild and severe progress of the occupational bronchial asthma is characteristic fro asthma of joined sensitization (professional allergic agent and bacterial one). Due to frequent acute condition and absence of remission, lung emphysema and signs of cor pulmonale among these patients, in spite of the rational change of occupation, appear already in 3 to 5 years after the beginning of the disease. There is also an opportunity of the development of broncvhoectasies, chronic pneumonia and asthmatic status. Severe and prolong fits of bronchial asthma, as well as spread obstruction of bronchiole with viscous phlegm can become a direct reason of the death.

Between fits, clinical signs of bronchial asthma can be absent. This state is more characteristic for initial stages of he disease, and in more marked stages of bronchial asthma, even between fits, there are the following indications: complicated breathing, moderate dyspnea at physical activity, coughing with mucous phlegm. Coarse breathing can be heard in lungs, often with dry crepitations, especially when breathing is forced.

Patients with occupational bronchial asthma have changes in peripheral blood (eosinophilia, Kurshman's spirals, crystals of Charko and Leyden), protein spectrum of blood serum, increase of the level of histamine, reduction of excretion of 17-hydroxy-costicosteriods with urine.

Development of occupational bronchial asthma depends on the peculiarities of the occupational anamnesis (character and type of allergic agents). Thus, for the occupational bronchial asthma, which is like atopic one, presence of symptoms of exposition and elimination (appearance of fits of asphyxia when contacting with the allergic agent and its termination after the termination of the action of an allergic agent). Especially, it is obvious after the rest (vacations and weekends) during the period of the so-called monovalent sensitization. Timely rational work change at the stage of pathologic process can prevent its further progressing. In case of continuation of work under conditions of occupational factors, which had caused the disease, its progress leads to worse conditions due to the development of polyvalent allergy. During this period, termination of the contact of patients with occupational factors does not bring the improvement of their condition (the period of polyvalent sensitization).

The first fits of asphyxia of such patients follow allergic affections of upper respiratory tracts and skin. In the peripheral blood, there is eosinophilia. And in the phlegm there are eosinophiles as well as Kurshman's spirals.

Under the condition of the influence of matters, which cause local irritation onto the respiratory organs or cause dust (toxic and dust) bronchitis or pneumoconiosis, occupational bronchial asthma of joint sensitization develops. For such a form of disease, it is characteristic to have no clear elimination syndrome, thought worsening of the state with more frequent fits of asphyxia takes place among the patients of the group as well, when they renew their contact with occupational factors (exposure symptoms). When the sick terminate their contact with an allergic agent, asphyxia is not replaced by the complete remission though. In the clinical pattern, there are symptoms of inflammatory process in the bronchial tree, and upper respiratory tracts. Mucopurulent sputum is discharged, where pathogenic bacteria are seeded.

The sick are characteristic to have subfebrility as well as insignificant leukocitosis. Gradually, the number of asphyxia fits increases, they also have worsened dyspnea, and not only at physical activity in contact with an allergic agent, but also due to irritating cold. And with this form of occupational bronchial asthma, a pattern and frequency of joining asthma with allergic changes in the upper respiratory tracts and skin exist. Usually there is no heredity in complicated allergic diseases. As a rule, in all the cases, initial fits of asphyxia are interconnected with infectious – inflammatory diseases of respiratory organs in the form of repeated respiratory infections, acute bronchitis and pneumonia. Inhalation testing with occupational allergic agents proves the development of an allergic reaction on the immediate-slowed down type.

After radiological examination, patients with asthma joint with allergies, have the intensification of vascular – bronchial pattern in lower portions of lungs. In some cases together with this, there are pleurodiaphragm commissures in the result of infectious-inflammatory diseases of respiratory organs.

The equivalent of the bronchial asthma is the asthmatic bronchitis, which is evident through expiratory dyspnea, absence of large-scale fits of asphyxia, as well as presence of catarrhal phenomena in the lungs when production allergic agents with production dust or irritating matters. In the anamnesis of the development of asthmatic syndrome, there is acute respiratory viral infection, bronchitis and pneumonia. The symptom of elimination in the clinical pattern of the disease is absent. Radiological examination allows determining the intensification of vascular-bronchial pattern in lower portions of lungs, and pleural-diaphragm commissures. As a rule, inhalation testing eliminates positive reaction to immediate-slowed down and slowed-down types.

Diagnostics. Clinical manifestation of the occupational bronchial asthma does not differ from those, which take place with the asthma of different etiology. Specific difficulties can take place in the process of definition of the etiologic factor in the genesis of this or that form of asthma. Thus, it is very important to study the occupational anamnesis of the patient, sanitary and hygienic characteristics of his/her workplace, as well as the data on allergen anamnesis, clinical manifestation and immune methods to examine a patient.

The presence of the contact with industrial allergic agents, production dust and irritating matters, fits of asphyxia at work and significant improvement of the state during vacations or staying at hospital on sick leave, correspondence of the clinical pattern, as well as all the factors, which can assisting the development of asthma (heredity, hormonal disorders, diseases, life conditions, etc), enables to suspect occupational bronchial asthma which needs specific allergen examination.

Methods of allergen examination, which need immediate participation of the patient (skin allergen tests and provocative inhalation testing) is conducted in case of satisfactory feeling of the patient during the remission stage. General contra-indications to use these methods of diagnostics are acute fever states and inflammatory processes; active TB form, pregnancy, decompensation diseases of heart, liver and kidneys; thyrotoxicosis; as well as complicated forms of bronchial asthma.

Mostly, scratch test or internal tests are used. To carry out scratch test, one drop of allergen is put onto the palm portion of the forearm, and through it the scratch is made. The reaction is assessed in 20 to 30 minutes, then 24, 48 and 72 hours. As a rule, immediate positive reaction takes place. When conducting of the allergen reaction under skin, it is necessary to administer from 0.05 to 0.1 ml of the allergen, which contains one skin dosage. Positive reaction is of the slowed-down type and it is assessed in 24, 48 and 72 hours since the administering of the allergen.

Provocative inhaling testing is conducted only in the phase of bronchial asthma remission and only in hospital. After the percussion and auscultative examination of lungs, spirogram is taken with the definition of Tifno index. Then within 3 to 5 hours, test-control liquid is given to the patient through an

aerosol inhaler. If within 5 to 10 minutes, the patient does not feel worse, another spinogram is made and in case of absence of significant sighs, inhalation of the least concentration of allergen is conducted for 2 to 3 minutes. After this, characteristics of the Vital Pulmonary Capacity are checked, as well as indexes of forced exhaling in 20 min, 1 hour, 2 hours and in 1 day. Provocative inhalation testing is considered positive, if VPC is reduced by 10 %, and Tifno index – by 20 % comparing with initial data.

Among the methods of allergen laboratory diagnostics to find out sensitization to industrial allergens, the following are used:

- reaction of a blood cell to the hapten in vitro – reaction of specific blood leukocyte accumulation (RSAL), tests on damage and alternation of blood neutrophyls (PPN) and reaction of direct specific damage of blood basophiles (RSPB);
- serologic reactions – reaction of compliment binding (RZK) and reaction of passive hemagglutination (RPGA).

Specific cell reactions on hypersensitivity in vitro – reaction of specific rosette formation (RCR), reaction of termination of blood leukocyte migration (RGML)

Each method of diagnostics with the attraction of the given above reactions is based on specific peculiarity. Thus, RSAL of the periphery blood – on the effect of intensification of adhesion of white blood cells in case of adding to it a specific allergen of the reaction cell, which is one of the first phases of specific allergic reaction of the blood cell. Reaction is assessed as positive when RSAL is equal to 1.4 and higher. PPN – on the immune phenomenon, which develops according to the reaction type of target cells onto the immune complex, which is created in the serum in the result of adding a specific antigen. Reaction is defined as positive when the indicator is 0.05 or higher. RSPB – due to the fact that blood basophiles and mast cells of the connective tissue serve as target cells in realization of reactions of immediate action. Reaction is positive if the indicator is 1.4 and higher.

Only complex evaluation of the occupational and allergologic anamneses, of the corresponding documentation regarding the conditions of work and dynamics of the disease, and also results of specific allergologic and immune examination of the patient enable professionals to state the professional genesis and etiological factor of bronchial asthma.

Treatment. Treating methods with occupational bronchial asthma should take into consideration the data on etiological and pathogenesis. With atopic non-infectious form, especially in the initial stages, termination of the contact with production factors can cause disappearing of fits.

The most grounded method of treatment of bronchial asthma is specific hyposensitization of the body. However the complexity of defining the majority of allergens of the occupational character, short term of the achieved effect, a

threat of the development of complications (anaphylactic shock) do not let us consider this therapy method as efficient.

In complex treatment, it is important to liquidate the concentration of the chronic infection. Among recent medicinal drugs, particular attention is paid to the drugs which mostly stimulate β_2 – adrenoreceptors of bronchi. In particular, they include salbutamol, terbutalin, and alupent. It has been proved that in comparison with other adrenoceptor agonists (adrenalin and ephedrine), which influence not only β_1 and α-adrenoreceptor and assist to the increase of the arterial blood pressure, tachycardia, anxiety, increase of and asphyxia; but they have less influence onto the cardio-vascular system.

At the same time, aminophylline (IV of 5-10 ml of 2.4 % solution into 10 – 20 ml of 20 % solution of glucose) is still the very therapeutic method of treatment of patients with occupational bronchial asthma, used most often. To prevent fits of asphyxia, it is possible to use retarded forms of theophylline – theopec and retafil.

Besides bronchodilatory methods, antihistamine drugs are often used to treat patients with bronchial asthma: Dimedrol – 0.03-0.05 g 1-2 pills a day; phencarol – 0.025-0.05 g 1-3 pills. Ketotifen inhibits release of histamine from mast cells, and they are prescribed in 0.001-0.002 g in the form of pills or capsules twice a day. Disodium cromoglycate as a method of biochemical preventive measure, stabilizes the membrane of mast cells and does not let release of biologically active matters from them, and they are prescribed in the dosage of 20 mg in the from of microionized powder four times a day using an inhalator. Calcium channel blocking agent are prescribed to patients with bronchial asthma on the background of physical tension, as well as to those who suffer from ischemic heart disease. Glucocorticsteroids are administered only then when all usual methods of treatment did not give the expected effect. Prednisolone is prescribed in pills 0.005 g; in acute cases, treatment starts with 20-40 mg a day, after it achieves the curing effect, the dosage is reduced to 5-10 mg and less. In emergency cases, prednisolone is used for injections. It is prescribed intravenously or intramuscularly in the dosage of 100-200 mg a day. It is also possible to use synthetic steroid hormones – beclometasone in the form of aerosol for inhalations.

Expectorant and antitussive methods: 3 % solution of potassium iodide in the dosage of 0.3 to 1 g a day; Tarasov's mixture internally – 1 teaspoon – 1 table spoon with warm milk – 3 – 4 ties a day after meals.

Antibacterial means, particularly when there is purulent bronchitis, antritis, and pneumonia; ampicillin and biceptol.

Immunomodulators: considering the fact that patients with bronchial asthma have reduced activity of the T-immunity, decaris is used, 100 mg – the first four days in a row with a two-day break.

Among non-medicinal methods of the therapy for patients with occupational bronchial asthma, reducing diet therapy, needle reflexo-therapy,

curing gymnastics, respiratory gymnastics, physiotherapy (ultraviolet, ultrahigh frequencies, and electrophoresis), sanatorium-and-spa treatment (Crimea) and pneumatotherapy.

Verification of workability. When making decision on workability and job of patients with bronchial asthma, it is always necessary to remember that independently from the degree of the disease severity, they are contra-indicated the contact with matters of sensitized and irritating action, staying under unfavorable meteorological conditions and significant physical activity.

Workability of patients with bronchial asthma of mild degree is usually kept, but they need rational job.

When bronchial asthma of mean severity among patients can be significantly restricted or completely lost. In connection with the development of respiratory insufficiency and decompensation of chronic cor pulmonale of patients with bronchial asthma of the severe degree, as a rule, inability to work, and many of them require external assistance and supervision.

Preventive measures. The task of medicinal preventive measures is to keep workability of workers and employees, and to prevent development of occupational medicinal examinations to select those who had to start working under conditions of possible contact with allergens. It is also important also to define initial sings of the disease and rational work beyond contacts with production allergens.

EXOGENOUS ALLERGIC ALVEOLITIS

It is a general term of the group of allergic pneumonias, which progress with involvement into a diffusive dispersed inflammatory process of some groups of alveoles.

Etiology. The reason of the development of exogenous allergic alveolitis is the allergen, which enters the organism with inhalation, together with the inhaled air. Such allergens can be weevil (wheat), extract from the dust of cacao beans (cacao beans), serum protein, antigens of bird droppings (feature and droppings of pigeons, chickens, and parrots), thermophilic actinomycetin (rotten hay), penicillin (medicinal drugs), salts of heavy metals (chemical matters), etc.

Size and number of particles are very important in the development of alveolitis. It is considered that particles up to 5 micromicrons easily achieve alveoli and are capable to cause sensitizations.

Pathogenesis. Allergen, which gets to the organism, causes sensitization, accompanied by the creation of antibodies. These precipitant antibodies together with allergen create immune complexes, capable to deposit in the walls of alveoli, and bronchial tubes. They cause inflammation (bronchiolitis and alveolitis), increased permeability of vessel walls (due to discharges of mast cells and basophiles of vasoactive amines), formation of granulomas (granulomatous pneumonitis), which leads to the development of interstitial fibrosis and disorders of ventilation function of lungs of the restrictive type.

Pathologic and anatomic patter. For an allergic alveolitis, it is characteristic to have granulomas in the walls of alveoli and bronchioles, as well as inflammatory infiltration of lymphocytes and plasmatic cells, as well as accumulation of exudation. Granulomas consist of epitheloid cells, which in the center are surrounded with lymphocytes and plasmatic cells. On the later stages of pathological process, pulmonary fibrosis is present.

Clinics. Clinical pattern of the disease is characterized by general symptoms (fever, pain in muscles, reduction of body weight). Sings, connected with the affection of respiratory organs, show involvement of bronchioles and alveoli into the pathologic process.

Often the disease starts with the growing dyspnea and coughing. When using auscultative methods, it is often possible to hear crepitations, mostly in interscapular regions. Acute form can be recognized rather easily. At functional research, decrease of blood saturation with oxygen, increase of partial pressure of CO_2 in the arterial blood, also clear respiratory alkalosis can be observed. Pulmonary capacity is reduced in the majority of cases, in particular, lung vital capacity.

Alveolitis can be chronic. It develops in the result of repeated less intensive influences of disease causing agents in several months after coming across them and are characterized with progressing respiratory insufficiency. Patients are bothers with dyspnea, sometimes with moderate fever and drowse. With X-ray examination, interstitial fibrosis can be observed.

One of the examples of allergic alveolitis is "**farmer's** or **thresher's lung**". That is the disease when inhaling of organic dust causes the reaction of increased sensitivity on the alveolar level, connected with the production of precipitin, and which is characterized with allergic diffusive affection of alveolar interstitial structures of lungs. The disease can be met among agricultural workers, which come across damp moldy hay, grain, silo and other herbal materials. It is more often can be observed in winter and autumn period of the year, when hay stocks are used as feed for domestic animals. Mostly, the development of the "farmer's lung" disease is caused by thermophilic actinomycetes: Micropolyspora faeni and Thermoactinnomyces vulgaris.

Acute forms are characterized by their sudden initiation. In 3 to 6 years after the exposure, temperature suddenly increases up to 39 to 40 ºC, headaches appear, as well as pain in muscles, and coughing with poor phlegm, and sometimes with the mixture of blood. Sometimes, there is nausea and vomit, voluminous hidrosis, and progressing dyspnea. During examination, cyanosis, tachycardia, frequent breathing at rest can be observed; and crepitations and single dry rales can be observed during auscultative examination. After radiological examination, intensification of pulmonary picture and small nodular types of different intensiveness can be observed. If the action of the allergen is eliminated, symptoms of the disease disappear in 7 to 10 days.

The repetition of the contact with disease causing agents leads to the development of subacute form of the disease, where clinical and radiological indications disappear much slower. There are indications of growing respiratory decompensation in restrictive or obstructive type, but the latter does not happen often; they have dry coughing, and chill at night. When examining, it is possible to find out that the chest acquires barrel-like look; crepitations in lower portions of lungs can be heard. Radiological examination shows more marked changes in the form of diffusive nodular shadows can be observed in lungs, which are located mostly in mean and lower portions of lungs. The disease stops in 4 to 8 weeks, if further contact with herbal dust is terminated.

Chronic form of the disease appears in the result of constant exposure to insignificant amount of dust of moldy hey to the organism of the human body. In the clinical patter, there is mostly dry coughing, dyspnea at physical activity, subfebrile temperature; total condition is worsening, and body mass is decreasing. At auscultation, crepitations, as well as fine and mean bubbling rales can be determined. If contact with dust continues, irreversible changes can take place – fibrosis of lungs and decompensated cor pulmonale.

At the functional research of external respiratory, restrictive form of ventilation decompensation can be observed. Lung vital capacity is decreased, and their diffusion ability decreases.

Diagnostics. Diagnosis can be made based on the occupational anamnesis (sick people, which do not have inclination for atopic reactions; the disease develops in a rather long-term contact with the allergen), peculiarities of clinical patterns (duration of the latent period, and characteristic signs), as well as radiological changes. The diagnosis is proved after skin testing (with blood serum or an extract of placenta) and serologic research (to find precipitant antibodies with methods of immune electrophoresis and radioimmunology). In some cases, biopsy of lungs or analysis of bronchoalveolar lavage is recommended (increase of T-lymphocytes).

Differential diagnostics. Exogenic allergic alveolitis should be differentiated with sarcoidosis, for which it is characteristic to have absence of the connection with the profession, affection of other organs, besides, lungs, development of hypercalcium areas, increase of near root lymphatic nodules on the radiogram, weak or negative reaction onto tuberculine and positive Quame's reaction.

Alveolitis should be also differentiated from pneumonia of infectious origin, for which it is characteristic to have the connection with colds, segmental or area shadowing on the radiological photograph, as well as expressed intoxication syndrome.

Treatment. The most efficient method of treatment is the termination of contact of the patient with the allergen, which caused the disease.

To treat exogenic allergic alveolitis (in particular of subacute and chronic forms), corticosteroids are used. Prednisolone is prescribed in the dosage of 1 mg/kg a day for 7 to 14 days, then the dose is gradually reduced.

Verification of the ability to work. Issues as to the ability to work of patients with the disease of lungs, conditioned by the impact of rottening herb dust, is solved the same way as in case of corresponding forms of dust diseases of lungs, caused by other types of dust.

Preventive measures. Main preventive measures for the patients with exogenous allergic alveolitis are in preventing the contact of the patient with corresponding allergens by the change of technological process (decrease of concentration of the allergen in the exhaled air), as well as the usage of respirators and other means of individual protection of respiratory organs.

Chapter 3

DISEASES OF BRONCHOPULMONARY APPARATUS of TOXIC-CHOMICAL ETIOLOGY

In various spheres of industry – metallurgic, chemical, oil-processing, pulp and paper, pharmaceutical, and mechanical engineering – mix of chloral, sulfur and nitrogen have become rather popular. They all can be in the air of the work zone in the form of irritating gases: chloral, chloro-hydrogen, sulphur trioxide, hydrogen sulphide, nitrogen oxide and ammonia.

Chloral is a gas of green and yellowish color with strong odor. It is 2 ½ times heavier than air and possesses good oxidation power. It has irritating and reflexive action onto the body. In case of irritation of interoreceptors of mucous tunic of upper respiratory tracks, spastic phenomena can take place in bronchi, the heart activity changes, and phenomena of irritation of respiratory and vessel centers are observed.

Poisoning with chloral is possible in pulp and paper, and textile industries, where chloral is used for bleaching; as well as in pharmaceutical industry to make chloride of lime or bleaching powder.

Sulphur trioxide is colorless gas with strong irritating smell. It can be well dissolved in water, ethyl or methyl alcohol. Mostly, sulphur trioxide is in the industrial atmosphere of metallurgic workshops, workshops where sodium sulfite is produced, as well as in refrigerators. The main way of its introduction to the human body is through respiratory organs. In the organism, it can be found in blood. It acts as an irritant of mucous tunic of eyes and upper respiratory tracks and later it can affect lungs. It also has resorptive properties as it affects metabolism processes.

Sulphuretted hydrogen is colorless gas with characteristic smell of a bad egg. It is a bit heavier than air and thus it is accumulated in hollows, like pits and trenches.

Sulphuretted hydrogen is emitted into the air when producing viscose fiber, as well as when using dues at textile enterprises, and during mining and processing of polysulfide oil. It can be contained in sewage water in canalization pipes. Main way of getting to a human body is via respiratory organs. During accidents, such a big amount of sulphuretted hydrogen can get into the body, which caused acute poisoning.

Nitric oxides are gases of yellow-brown color. They look like a mix of the most spread nitrogen dioxide. These gases are created under production conditions when producing sulphuric acid, chromic acid, nitric acid, aromatic nitrocompounds, aniline dyes, as well as during oxy-acetylene welding, flame cutoff and electric welding.

ACUTE TOXIC AFFECTION OF BRONCHOPULMONARY APPARATUS

Main clinical forms of acute affection of bronchopulmonary apparatus of toxico-chemical etiology are as follows: acute toxic laryngitis-pharyngitis-tracheitis, acute toxic bronchitis, acute toxic bronciolitis, acute toxic swelling of lungs, as well as acute toxic pneumonia.

Under industrial conditions, they can appear in accidental situations, in case of inhaling toxic matters of significant concentrations. Appearance of intoxication is caused by the following: high concentration of the chemical matter in the air; duration of its action; general reactivity of the organism; as well as physical and chemical properties of poisoning matters.

Pathogenesis. Irritating matters when having impact onto the body of the worker, get in contact with the moisture of respiratory tracks and creation comounds, which have burning properties (hydrochloric acid with the action of chloral, salt cake - sulphuretted hydrogen, etc). These compounds cause disorders of respiratory functions due to local irritating action, as well as the disorder of the reflector character (impact onto the interoreceptors of bronchis). The result is spastic contraction of heart muscles, respiratory and cardio-movement centers. Spreading and heaviness of affections depend on the degree of dissolving of toxic matters in water.

Matters, which are easily dissolved in water (chloral, chloropicrin, ammonia, and sulphuretted hydrogen), have impact mostly onto the mucous tunic of tracheas and large bronchi. Clinically, it is manifested by an acute laryngotracheitis and acute toxic bronchitis.

Matters, which are difficult to be dissolved in water (nitrogen oxide, phosgene, chloropicrin and dimethyl sulphate), they deeply permeate into the bronchopulmonary system, and affect small bronchi and bronchioles. And clinically, this can be conditioned by the appearance of acute bronchitis and broncholitis.

ACUTE TOXIC LARYNGOTRACHEITIS. In the clinical development, there are three phases of severity.

Mild phase is characterized by hyperemia of mucous tunic of upper respiratory tracts; and in some places small hemorrhages are observed.

Mean phase: swelling of mucous tunic, coarse voice and sometimes its complete loss are observed.

Severe phase is characterized by the necrosis of mucous tunic with creation of ulcer in it. Possible development of acute swelling of lungs with further asphyxia and death of the patient are observed.

ACUTE TOXIC BRONCHITIS. There are three phases of the severity of acute toxic bronchitis.

Mild phase: (superficial or catarrhal toxic bronchitis) is characterized by the following: painful coughing, pain and “scratchy” throat, squeezing and scorching throat, as well as obstructed breathing. Patients have epiphora and

light phobia. During the percussion, it is possible box sound can be heard, mostly in lower side portion of lungs, during auscultative examination, scattered dry rales can be heard on the background of coarse breathing. Duration of this phase of bronchitis is from 3 to 7 days; complications are absent.

Mean phase: patients complain to have rhinorrhea, epiphora, obstructed breathing via a nose, intensive pain in the chest, fit-like coughing with phlegm discharge. Cyanosis and dyspnea can be well heard. Above lungs, signs of emphysema are observed; during auscultative examination – dry rales and sometimes some moist rales can be heard. As to the cardio-vascular system – tachycardia is observed. As a rule, the temperature of body is increased to low grade, moderate neutrophilic leukocitosis, as well as increased ESR. The duration of this phase of bronchitis is from 7 to 10 days.

Severe phase is characterized by the presence of marked cyanosis, and dyspnea at rest. Objective signs of emphysema include dry and moist rales all over the surface of lungs. This phase lasts for 2 to 6 weeks, and in case of adequate treatment, complete recovery is possible. Further progress of acute toxic bronchitis is possible under conditions of joining infection, its transition into a chronic form with the development of pneumosclerosis.

ACUTE TOXIC BRONCHIOLITIS. It develops during the impact of toxic matters of irritating action, first of all those, like dimethyl sulfate, joining of beryl.

In *mild cases*, patients complain to have coughing, small amount of phlegm, moderate dyspnea, and low grade fever. Above the lungs, basal emphysema can be observed; small moist crepitations in small amount can be heard.

From the point of peripheral blood, small leukocitosis and increased ESR can be observed. This stage lasts for from several days to 2-3 weeks.

In more marked phases, dyspnea is increased, coughing becomes unbearable, and sometimes it is fit-like, and it is accompanied by pain in the chest, and thick mucoid sputum. Patients complain to have headache, loss of appetite, increase of temperature to 38 – 39 ºC, and general weakness. Marked cyanosis takes place, and tachyponea (30 – 40 a minute). Objectively, there are sings of emphysema, ends of lungs are lowered; their movement is reduced. Above all the surface of lungs and especially in lower portions, big number of medium and small bubbling moist capitations can be heard. As to the cardio-vascular system, tachycardia can be observed; decrease of arterial pressure and muffled heart sounds can be heard. Liver increases, it becomes more painful during palpitation.

In the peripheral blood, increase of hemoglobin, eretrocytes, leukocytosis with stab neutrophil disorders, relative lymphopenia, and sometimes, eosinophilia can be observed, and ESR increases up to 50 mm/h. Proteinuria and cylindruria can be observed in urine.

On the radiogram of chest organism, decrease of transparency of lung field in mean and lower portions, and there are also fine formations, which merge in some places. Lung roots are expanded.

Patients with acute toxic bronchiolitis either recover or receive various complications: bronchopneumonia, transition to the chronic form with obliteration of the lumen bonchioles.

ACUTE TOXIC PNEUMONIA. It appears in the result of short-term impact of the toxic matters and is characterized of acute beginning, short-term progress, and absence of the inclination to the appearing of new centers.

Pneumonia in the result of the action of gas appears in an acute form, within several hours after accidental aspiration of petroleum or its permeating into the respiratory ways. Cyanosis, dyspnea and intensive pain in the half, which is the location of pneumonia infiltration, are observed, as well as coughing with prune-juice sputum. The temperature increases to 40 ºC.

During percussion, atrophy of percussion sound is observed on the side of affection; and during auscultative examination: bronchial breathing and crepitations are observed. In the peripheral blood, there are sings of the inflammatory process (leukocytosis, lymphopenia and the increase of ESR).

On the radiological picture, an area of pneumonic infiltration in the form of homogenous shadowing, which is localized mostly in the front portion of lungs.

Treatment. Oil and alkaline inhalations are recommended, as well as antitussive drugs (tussuprex and libexin), and antibiotics.

ACUTE TOXIC SWELLING OF LUNGS is the most serious and dangerous form of acute toxic affection of bronchopulmonary apparatus. Etiological indications of its appearing can be nitrogen oxides and chloropicrin.

Pathogenesis. In the mechanism of development of toxic swelling of lungs, an important place is taken by the impact of toxic matter onto the activity of ferment systems, which contain SH groups. They are accompanied by high increase of the permeability of alveolar membranes with the disorder of metabolism. In the interstitial tissue, high protein liquid is accumulated in alveoli.

Besides an immediate action of toxic matters onto the ferment systems, the mechanism of toxic swelling of lungs is impacted, which is proved by the decrease and often prevention of the development of lung swelling due to elimination of some sections of the nervous system (vagosympathetic blockade and cutting of the vagus nerve on the neck).

In the development of toxic swelling of lungs, five periods can be named:

The *first one* or the period of irritation. Clinical manifestation: epiphora, coryza, coughing, “scratchy” throat, and squeezed chest; duration: 15 to 20 minutes.

The *second one* or the period of imaginary well-being. Clinical manifestation: insignificant dyspnea, pulse instability, and signs of moderate emphysema; duration from 3 to 8 hours.

The *third period* or the period of the increase of the swelling. Patients complain to have squeezed chest, dyspnea, coughing with phlegm discharge. During the percussion, box sound above the lungs; during the auscultative examination: fine moist rales. The number of rales increases fast.

On the radiological picture of lungs, the pattern is vague and roots are expanded.

As tot eh peripheral blood, increase of ESR is observed, as well as leukocytosis with the motion of the formula to the left and lymphopenia.

The *fourth period* or the period of the end of swelling. Clinical manifestation: dyspnea and coughing increases, blood phlegm is discharged, and breathing is bubbling. During auscultative examination, significant number of heterogeneous moist rales.

Radiologically, it is possible to observe spotty shadows, which are conditioned by the accumulation of swelling liquid in alveoli. These shadows create big merged areas in some places.

When researching blood, its clotting is observed, which is manifested through the increased of hemoglobin, eretrocytes and leukocytes. Blood viscosity is observed.

The amount of oxygen in the arterial blood decreases sharply, but the amount of carbon dioxide increases; the so-called hypercapnic (blue) type of hypoxemia develops. The arterial pressure is normal or a little increased. Blue asphyxia can develop into the gray one.

The gray type of hypoxemia is characterized by low contain of blood of carbon dioxide, merging of the swelling of lungs with the decrease of cardio-vascular activity (collapse). The patient’s face gets gray pale hint and is covered with cold sweating. Mucous tunics are of dirty gray color. Limbs are cold and damp when touched. The pulse is frequent and thready; the arterial pressure decreases fast.

The *fifth period* or the period of reverse development of the toxic swelling. It is characterized by the decrease of dyspnea, cyanosis, coughing, and the amount of discharged phlegm, as well as crepitations in lungs. The pattern of peripheral blood is normalized. In 2 to 4 days, a patient recovers. The temperature stays increased (37.5 to 38.0 ºC) during the week.

Complications: pneumonia and the development of pneumosclerosis.

Treatment. Patients are recommended to take tea, coffee, and put a hot-water bottle. They are recommended to take oxygen (oxygen inhalations are compiled with vapors of 30 % alcohol with the purpose to prevent the creation of foam). Eyes are washed with 2 % solution of sodium chloride and instill with albucid (30 % solution), Novocain 1 – 2 drops of 1 % solution. A nose and throat are rinsed with the solution of sodium bicarbonate.

In case of the development of spasms of glottis, 1.0 ml of 0.1 % solution of atropine hypodermically.

People, who had contacts with damps, need to be examined in hospital. With the purpose to decrease the permeability of vessels, 5 to 10 ml of 10 % solution of calcium chloride are introduced intravenously.

With strong hypoxemia, the following is recommended: oxygen therapy (oxygen with alcohol vapors); bloodletting (from 300 to 500 ml of blood) under the control of the arterial blood.

With the gray type of hypoxemia are recommended: inhalations with oxygen (60 % with the addition of 5 % of carbonic acid); means which stimulate the respiratory center (caffeine, Corazol, and ephedrine); antibiotics and sulfanilamides medicines with the purpose of infection prevention (development of bronchopneumonia).

Verification of the ability to work. In cases of mild affection of bronchopulmonary apparatus with matters of toxic and chemical action, patients return to their work.

For patients with mean or severe phases of affection, it is necessary to make sure that results of the treatment stay for long; these patients need temporary termination of work in the areas with the contact with chemical matters of irritating nature. In case of the decrease of qualification for the term of more than two months, they can receive a sick leave or receive an invalidism group due to their occupation disease.

Preventive measures. Preventive measures envision the following: sealing-in of the equipment, utilization of individual means of protection of respiratory organs, as well as conduct of preliminary and periodical medical examination of workers.

CHRONIC TOXIC DISEASES OF BRONCHOPULMONARY APPARATUS

Chronic diseases of bronchopulmonary apparatus develop among workers of chemical enterprises and are the result of long-term impact regarding small concentrations of toxic matters of irritating action. They can also be the result if one (or several) acute affection of bronchopulmonary apparatus.

Chronic toxic diseases are more often had in such forms as affection of upper respiratory tracts (chronic rhinitis, pharyngitis, laryngitis and tracheitis), as well as chronic toxic bronchitis.

Affection of upper respiratory tracts (nose, throat and larynx) has, at first, catarrhal and then atrophic character. A patient has the feeling of dryness in the nose; heartburn, disorder of nose breathing, scratchy throat, sometimes nose bleeding as well as coarse voice. After the examination, hyperemia of mucous tunic is observed, as well as its thickening. For the action of chromium and fluorine (especially, in high concentrations), it is characteristic to have necrotic tonsillitis affection in the area of nose septum.

At dynamical examination, it is possible to find some pattern in the development of deep erosion, which is finished with the formation of connective tissue scar or the perforation of the septum.

CHRONIC TOXIC BRONCHITIS is a diffusive affection of the bronchial tree, which is characterized by the recurrent and progressing development of the disease.

Pathogenesis. In case if the organism of a worker is impact by irritating matters, hyperplasia of cup-shaped cells of bronchial glands with the hypersecretion of mucus and the change of their properties. Secretory, cleaning and protection functions of the mucous tunic of bronchi are affected, what assists fast to the development of inflammatory process. Chronic toxic-infectious inflammation, metaplasia and atrophy of epithelium are formed. They are manifested with swelling, collapse of small bronchi, and scar changes, which finally form broncho-obstructive syndrome.

Pathologic and anatomic patter. When chronic diseases of toxic and chemical ethiology develop (e.g. chronic toxic bronchitis), peribronchial tissue is affected. Interstitial tissue can be also affected with further development of pneumosclerotic secondary bronchitis. The result of the action of toxic matters on the alveolar septums is the development of emphysema.

Morphological peculiarities of toxic bronchitis are as follows: 1) severe affection of bronchi with the involvement of peribronchial tissue of lungs with vessel and lymphatic systems into the process; 2) joining of chronic bronchitis with obliterating bronchiolitis, which develops very slowly with further access to spot carnification; 3) stable progressing pneumonia is characterized by a marked proliferative process in alveolar septums and its development next to brochiosclerosis of the diffusive pneumocyrosis of lungs.

The result of toxic bronchitis and interstitial pneumonia is the diffusive pneumosclerosis with its unchangeable components, like lung emphysema, pneumosclerosis, bronchiectasis and appearance of chronic pneumonia and bronchiectatic disease.

Clinic. There are three phases of chronic toxic bronchitis: mild, mean and severe.

Mild phase. Patients suffer from dry coughing, sometimes with small amount of purulent phlegm or mucopurulent sputum character. In lungs, some capitations can be heard on the background of coarse breathing.

Acute conditions are rare in this stage. Usually they appear under the condition of unfavorable microclimatic factors or joining of an intercurrent infection.

Deviations on the radiological picture at this phase are not found. When researching functions of external respiration, some small deviations on fast indications are found.

Mean stage. It is characterized by the intensification of coughing; amount of phlegm discharge increases (it is coming closer to purulent one); dyspnea appears during physical exercises. Fit-like coughing can develop. Above lungs, percussion sound gets box sound on the background of coarse (sometimes, weak) respiration; scattered dry and moist crepitations can be observed. Acute conditions are more frequent and longer. On the radiological picture, intensification and deformation of lung pattern can be observed, mostly in lower portions; lung fields become more transparent. When researching functions of external breathing are determined by its stable disorder on obstructive type.

Severe stage. As a rule, it is a complicated bronchitis. More often, marked bronchospasmodic syndrome with the transfer to the secondary bronchial asthma or with appearance of bronchiectasis take place.

Manifestations of bronchospasmodic syndrome (complicated breathing with fits of suffocating coughing) remind fits f bronchial asthma, but eosinophilia and the change of phlegm. However, there are cases of the progressing of infection – depending bronchial asthma with progressing clinical pattern, presence of corresponding changes in the phlegm and blood, development in further progressing respiratory insufficiency.

In some cases, clinical pattern reminds the progress of bronchoectatic disease (patients discharge from 300 to 50 ml of purulent sputum, and frequent hemoptysis is observed).

Coughing becomes permanent, much amount of purulent sputum, often with blood and unpleasant odor, are observed. Obstructed respiration, which transits into fits of dyspnea are observed. Patients get cyanosis, frequent respiration; nails get the form of clock glass, and phalanx of fingers look like drum sticks.

During auscultative examination, in lungs, it is possible to hear scattered dry and moist rales, mostly in lower portions of lungs. From the side of cardio-vascular system, there is tachycardia; tones of the heart are dull; the accent of III tone can be heard above the lung stem; stagnant phenomena can develop, fist in small and then in the large blood circulation circle.

In the blood, there is compensatory polycythemia (increased amount of hemoglobin and erythrocytes).

During radiological examination, it is possible to note deforming diffusive pneumosclerosis and lung emphysema. Independently from the overbalance of some clinical form, the severity of the state of such patients is conditioned by the increasing decompensation of the function of external respiration and decompensation of cor pulmonale.

Treatment. To treat chronic affection of upper respiratory tracts, alkaline and oil solution inhalations are used; if there is erosion – ulcer defects of mucous tunic, it is recommended to use synthomycin emulsion or the solution of retinal.

The treatment of chronic toxic bronchitis envisages means, which dissolve sputum and facilitate its discharge: inhalation with proteolytic ferments (trypsin and pancreatin), mucolytic and expectoration medicinal drugs. Significant place in the treatment of is given to drugs, which renew the bronchial permeability. First of all, these are sympathomimetic agents (asthmopent and salbutamol), derivatives of theophyllin (euphyllin). The effect of these medicinal drugs is increased by antihistamines, which together with the elimination of spasms of smooth muscles of the bronchi show anti-swelling action.

Significant place in the treatment of chronic toxic bronchitis is played by oxygenerotherapy, curing respiratory gymnastics, massage of the chest, and physiotherapeutic procedures (inductotherapy, electropharesis of Novocain or Calcium chloride).

In case of infection development, antibacterial means are recommended (antibiotics and sulfanamides); with cardiac decompensation – cardiac glycosidea (coglucon and strofantin), diuretics (furocemid, triampur and on-potassium-sparing diuretics).

Verification of the ability to work. In case of chronic affection of upper respiratory tracts, patients are able to work according to their occupation under condition of dispensary observation and normalization of work conditions. Presence of ulcerous-inflammatory processes is the condition to provide a medical leave (temporary work).

Patients with mild and mean phases of chronic toxic bronchitis need rational work beyond the area with dust action, matters of irritating action and physical overstrain. In case of the impossibility to get such a job and the decrease of qualification, they are sent to the Verification Commission to get the invalidism group.

Patients with complicated forms of bronchitis are unable to work as a rule; and sometimes need external help (II or I group of invalidism).

Preventive measures. Preventive measures are in the implementation of progressive technologies into the production process (sealing-in of the equipment, its further mechanization, effective ventilation and keeping to safety rules), as well as utilization of individual protection means and medical examination of workers.

Chapter 4

OCCUPATIONAL DISEASES, CAUSED BY CHEMICAL FACTOR IMPACT

OCCUPATIONAL DISEASES WITH PREDOMINANT AFFECTION OF BLOOD SYSTEM

Among occupational diseases of the blood system, four main pathogenetic groups can be defined nowadays (Table 7).

Table 7

Classification of occupational diseases in the blood system

Pathogenetic grouping	Main clinical form	Etiological factor
Diseases, conditioned by toxic affection of marrow	Hypoplastic and aplastic states	Benzol, xylol, toluol, and ionizing radiation
Diseases, conditioned by the change of blood base	Acute methemoglobinemia, acute carboxyhemoglobin-emia	Amido- and nitrocompounds of bezol (aniline, nitrobenzol, and trinitrotoluene) and carbon oxide
Disease, conditioned by hemolytic action	Acute toxic anemias, conditioned by intravascular hemolysis and chronic toxic anemias, conditioned by intracellular hemolysis	Arsenious hydrogen and phenylhydrazine
Diseases, connected with the disorder of the hemoglobin synthesis	Chronic sideroachrestic anemia and disorder of porphyrinic exchange	Lead

Intoxication with aromatic hydrocarbon

One of the simplest representatives of aromatic hydrocarbons is **benzol**. It is a colorless liquid with peculiar pleasant smell. It evaporates well at the room temperature. It is badly dissolved in water, but well in alcohol, ether and chloroform.

Benzol is widely used in various spheres of the industry: in rubber, chemical, pharmaceutical, polygraphic, paintwork, in the production of synthetic caoutchouc, explosive and medicinal matters. It is used as a solvent for fats and caoutchouc. The allowed concentration of benzol in the air of the work zone is 5 mg/m^3.

Under production conditions, benzol gets into the body mostly in the form of vapors through respiratory organs and undamaged skin.

Benzol is discharged partially in the unchanged state through lungs, partially, it oxidizes to hydroxyl compounds – phenol and dihydroxybenzene, which are discharged via urine together with sulphuric acid or gluconic acid.

Benzol belongs to poisons with general toxic polytropic action; but its well-known action is the action onto the haematogenous system. This conditions listing of benzol with the group of “Blood poisons”.

In the result of the action of benzol, both acute and chronic intoxications can be observed.

Pathogenesis. With acute poisoning, the action of benzol is mostly obvious in the central nervous system and it progresses according to the type of poisoning with narcotic poisoning.

Mostly, pathogenesis of chronic poisoning is in the inhibition of haemopoiesis – affection of proliferation of progenitor cells on haemopoiesis. Obviously, from the intensiveness (concentration of benzol vapors in the air of production territories) and the duration (number of work years in contact with benzol) impact, as well as from individual properties of the organism and its haematogenous organs (inherited inclination and previous diseases, which influence the blood system) depends the depth and the stage of affection of the marrow.

With the great intensiveness of toxic impact, the deepest affection of haematogenous organs is possible. In such cases, total inhibition of haemapoesis, disorder in proliferation of stem haematogenous cells and partially – predecessor of haemapoesis take place. Also, ability of these cells to differentiate can be affected. The result of such deep disorder of haemapoesis is progressing pancytopenia.

Less intensive toxic impact onto the marrow is accompanied by the inhibition of proliferation of differentiated blood cells (myeloblasts, erythroblasts and megacaryoblasts). Prevalent affection of granulocytopoiesis is possible here (progressing leukopenia) or thrombopoiesis (thrombocytopenia or hemorrhagic syndrome). Affection of germ of haemapoiesis can be assisted by pathologic changes or the impact ontpo a corresponding germ of haemapoisesis (fibromyoma, prolonged and excessive menses, gastric achylia, toxic impact onto the leucopoiesis of some medicinal drugs).

It has been stated that the toxic impact onto haematogenous cells are caused by not only benzol, as its transformations (phenols), which are created in the marrow, where benzol is accumulated. Thus, mutation in the chromosomal apparatus of haematogenous cells and the disorder of mitosis are conditioned by toxic impact of phenols.

Pathologic and anatomic pattern. Phenomenon of asphyxia is characteristic for acute intoxication with aromatic carbohydrates. Plethora of internal organs and spot hemorrhages in lungs, pleura, epicardium and mucous tunic of gastrointestinal tract; swelling, plethora of brain and its membranes as well as fine hemorrhages are observed.

At toxic intoxication with benzol, changes mostly take place in the haemopoiesis system. Hemorrhages into skin, mucous and serous tunics, internal organs, soft brain membranes, matter of cerebral hemispheres, and

its ventricles. The permeability of walls of blood vessels increases; perivascular intermediate sclerosis. Marrow has mucus-like consistency and it is pink-yellow. On the microscopic level, hypoplasia of marrow is noticeable; more rarely, atrophy and aplasia pf the panmyelophthisis take place (Fig. 3).

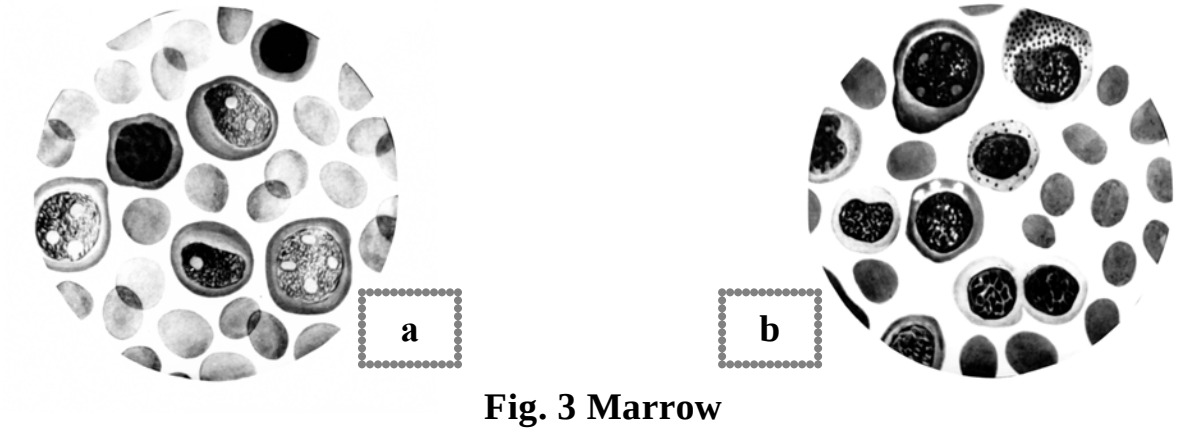


Fig. 3 Marrow
a – hypoplasia b - aplasia

All the shoots of marrow are affected. Sometimes, some areas of haemopoiesis stay. Together with the inhibition of haemopoiesis processes, there are cases with marked hyperplasia of marrow, up to leukemic pattern. Spleen is reduced, with phenomena of hemosiderosis. Liver is increased, and has signs of fat degeneration, hemosiderosis, intermediate sclerosis, and infiltrates with lymphoid and plasmatic cells. There are regenerated changes of epithelium of twisted channels in kidneys; ulcer on the mucous tunic, also much fat deposits in the hypodermic cellular tissue can be observed.

A c u t e i n t o x i c a t i o n s under production conditions can be observed rather rarely. They belong to accidental situations due to violation of safety rules. In the clinical pattern, changes in the central nervous system can be observed.

With *light poisoning*, general weakness, headache, dizziness, nausea, vomiting, noise in ears and jiggling when walking are observed. However, other transformations from the side of other organs and systems are not observed. Sometimes, it is possible to notice some leukocitosis with stab shift, which passes fast.

With more *marked intoxications*, conditioned by the impact of significant concentrations of benzol, loss of consciousness and pupil change reaction can be observed. Intense respiration is slowed down, pulse is increased, and weak filling and arterial pressure are decreased. At the same time, marked leukocitosis can be often observed.

In case of provision of the corresponding assistance, recovery takes place comparatively fast. There are cases of sudden death from the

paralysis of higher nervous centers with the action of high concentration of benzol (cleaning of tanks where benzol was).

C h r o n i c i n t o x i c a t i o n develops fast with prolonged impact of subtoxic concentrations of benzol. The severity and character of phenomena, which develop with poisoning, depend on work conditions (character of production processes, the temperature of the environment, concentration of benzol in the air, as well as duration of staying under these conditions), and from the organism state.

There are mild, mean and sever forms of chronic poisoning with benzol.

In *mild cases* of chronic poisoning, the most characteristic sign is the decrease of the number of leukocytes. At first, transitory leucopoenia is observed, then the decrease of the number of leukocytes is stable enough, and reaches the level $4.0 \cdot 10^9$ per liter and lower. At this background, the decrease of the share of neutrophilic granulocytes with toxigenic grain, hyper segmentation of nuclears, and increase of the number of stab neutrophilic granulocytes can be observed.

From the central nervous system, neurasthenic syndrome prevails. Signs of hypertension are observed.

Chronic benzol poisoning of the *mean degree* of severity is characterized by further decrease of the number of leukocytes (up to $2.8 – 2.0 \cdot 10^9$ per liter). It is also accompanied by thrombocytopenia, the number of thrombocytes decreases to $120 – 80 \cdot 10^9$ per liter. From the side of the central nervous system, asthenovegetative syndrome can be observed, which is manifested through headache, dizziness, general weakness, adynamy, increased irritability, sleep disturbance and hyperhidrosis. There can be changes in the peripheral nervous system according to the type of vegetative multiple neuritis, especially among those who has contact with benzol through arm skin.

Changes of the state of the cardiovascular system are characterized by hypertensia, liability of cardiac activity, hypertension, and moderately marked myocardium degeneration.

Patient’s functions of stomach towards hyposecretion are in disorder, digestion process gets worse and the acidity of digestive juices reduces. The liver is moderately increased, and its function is in disorder. The number of γ-globulin and sometimes, β-globulins is a little increased. Albuminoglobulin ratio is decreased.

Hemorrhagic syndrome (skin hemorrhages, epistaxis and menamenorrhagias), as well as positive symptoms of tourniquet and cuff takes place. The duration of hemorrhage increases; thromboplastic activity decreases; fibrinolisis is intensified, and the retraction of blood clot is in disorder.

In the marrow, various manifestations of a mild form of hypoplastic state with the intensification of proliferate activity of myelocariocytes.

With the *severe form* of poisoning, marked anemia is observed; the number of erythrocytes decreases down to 1.0·1012 in one liter; thrombocytopenia achieves the level of 30 - $35 \cdot 10^9$ per liter; the duration of bleeding increases to 15 – 20 min, and resistance to infections decreases. The following is characteristic: intense general weakness, drowse, noise in ears, dizziness, blackout, paleness of coverlets, and systolic noise in the upper part of the heart. Profuse hemorrhage appears (nosal, gastrointestinal and uterine hemorrhages). They are often accompanied by infectious complications (pneumonia, necrotic tonsillitis and septicopyemia). The latter can cause the death of patients.

The forecast in case of presence of a severe form of chronic intoxication with benzol can be rather unpleasant.

The described pattern of the chronic intoxication with benzol and the progress of hematological changes are rather conditioned. Thus, if in the air of the work zone where a worker is, the concentration of benzol is rather small, the pattern of poisoning is different. At first, anemia can take place, which later on is accompanied by leucopoenia and thromobpenia. In some cases, blood transformation starts with thromobopenia, and then the number of leukocytes and erythrocytes decreases.

Prolonged action of benzol can cause the development of chronic leucosis, which does not differ much according to its clinical pattern from the non-occupational one. Mostly it is chronic myeloid leucosis, sometimes – lymphoblastic leucosis and arethmia.

Treatment. With acute poisoning, it is necessary to terminate contact with benzol and stay outside as much as possible. In case of irritation of the mucous tunic of eyes, it is necessary to rinse them with 2 % of sodium bicarbonate, if respiratory tracts are irritated, then dionine is prescribed (0.015 g) or codeine phosphate (0.03 g two or three times a day). Good effect is provided by inhalation with the solution of baking soda; more severe forms of acute poisoning are the indication to prescribe oxygen. Together with it, hypodermic injections of 20 % of the solution of sodium caffeine-benzoate are prescribed in the amount o 1 to 2 ml or cordiamine of 2 ml. If in the future, changes in the liver take place, confinement to bed is prescribed; 15 to 20 intravenous injection 20 ml each of 40 % solution of glucose, 1 to 2 ml of 5 % solution of ascorbic acid. It is also possible to recommend a course (10 to 15) of intravenous injections of 10 % solution of calcium gluconate 10 ml each, which alternate with glucose injections (every other day). In addition, 20 pills three times a day are prescribed (total 15 to 20 days), vitamins B_1 and B_6.

With chronic intoxications with benzol, curing measures are determined by the character of main clinical manifestations of the poisoning and the degree of their definition.

With the presence of neurological symptoms, sedative drugs are to be prescribed (sodium bromide – 0.05 – 0.1 g two to three times a day; tincture of valerian – 6.0:200 ml – 1 table spoon three times a day; good result is provided by mild tranquilizers – meprobamate – 0.2 g or tazepam – 0.01 g two to three times a day). The duration of the course is two to three times. Together with it, it is recommended to administer 6 % solution of the vitamin B_1 with 1 ml intramuscularly during 15 to 25 days; 5 % solution of vitamin B_6 – 2 ml intramuscularly. The duration of the course is 1 to 1 ½ months.

In the clinical pattern of the blood affection, treatment should be conducted in a differentiated way with the consideration of current transformations. Hypoplastic state is the indication to prescribe pentoxile, which stimulates leucopoiesis and the production of antibodies. It is taken in after meals for 0.2 to 0.4 g three or four times a day, the course duration is 15 to 20 days. Leucopoiesis can be also stimulated by leucogen for 0.2 three to four times a day during a week.

Administering of vitamins B_1, B_2, B_6 and B_{12}, often shows good results. Sometimes, together with listed medicinal drugs, glucocortocoids, iron-containing drugs and transfusion of blood of the same type are prescribed.

In case of anemic syndrome, administering of hemostimuline is prescribed (0.5 g three times a day during meals).

Patients with toxic hepatitis are prescribed to take syrepar (intramuscularly – 2 ml during 30 days) and essentiale (2 capsules three times a day during meals).

Expertise of the ability to work. In case of acute poisoning with benzol of the mild degree, phenomena of intoxication pass fast (if further contact of the patient with benzol is terminated) and are not accompanied by the loss of the ability of the patient to work.

Recovery comes slower (on the fifth or seventh day) with the ointoxication of the medium degree, and a patient should receive a sick leave for this period. In the future, such patients are considered able to work on their occupation.

After a severe acute intoxication, stable outcomes of acute intoxication might stay after the recovery in the form of some syndromes of affection of the nervous system. Such patients are considered to have limited ability to work and should be assigned to face the Expert Commission to receive the invalidism group in the result of the occupational disease.

Expertise of patients with chronic intoxication is conducted with the consideration of the character and the degree of the expression of hematological changes and the presence of other syndromes. With vaguely marked leucopoenia ($4.0\text{-}3.0\cdot10^9$ per liter) and the absence of other transformations of blood and normalization of the pattern of blood, temporary promotion to another position with easier conditions and beyond the action of benzol for the period of two months and a sick leave should be provided for this period. Later, it is possible to continue work according to the occupation under conditions of strict keeping to sanitary and hygienic norms.

With more marked hematological transformations (II phase), patients should terminate the contact with benzol and its homologues completely. Patients should be assigned to face the expert commission to receive III group of invalidism and temporary pension for the period of re-qualification due to the occupational disease.

With the severe form of intoxication, patients should be considered unable to work (II group of invalidism) due to their occupational disease.

Preventive measures. To prevent poisoning with aromatic hydrocarbons, it is necessary to carry out sanitary and hygienic supervision of technological processes (sealing-in of the equipment, and effectiveness of ventilation), connected with the utilization of benzol and its homologues.

When carrying out some types of work, it is necessary to use individual means of self-protection to protect respiratory organs. With this purpose, filtering respirator is used, and in some cases, it is possible to use isolating respirator.

Considering the possibility of the permeation of benzol through undamaged skin, it is necessary to consider inexpedient to wash hands and uniform with paint spots with benzol, as well as the contact of bare parts of the body with it.

Besides benzol, its homologues are widely used in industries (toluol and xylol) and chloral derivatives (chlorbenzene, dichlorobenzene, etc).

Xylols – liquids, used as solvents to receive phthalic acids. Toxic action of xylols is characterized with the affection of the central nervous system and irritation of mucous tunic of eyes and respiratory tracts. Chronic intoxications are accompanied by the decrease of the level of erythrocytes, poikilocytosis, anisocytosis and moderate leucopoenia with relative lymphocytosis. Sometimes, some thrombocytopenia can take place. In case of getting to skin, the development of dermatitis is possible.

Dichlorobenzenes – are liquids, and paradichlorobenzene is a solid matter. Dichlorobenzenes are used as solvent, intermediate products to receive some dyes, as well as insecto-fungicides. In the clinical pattern of the intoxication with dichlorobenzenes are observed in the following cases:

irritation of mucous tunic of eyes and respiratory tracts; in the blood the level of hemoglobin is decreased, the decrease of the number of erythrocytes and thrombocytes, as well as the appearance of methemoglobin.

Intoxication with carbon oxide

Carbon oxide is a colorless gas, and in pure form, it has no smell or taste. It is lighter in weight than air; it does not dissolve in water and is well dissolved in the liquid ammonia.

Carbon oxide is a constituent part of a number of gases, which are used or created in industries. Producer gas contains from 9 to 29 % of carbon oxide, and blast-furnace gas contains up to 30 %.

Under unfavorable sanitary and hygienic conditions, as well as if not to keep to rules of safety measures, occupational poisoning with carbon oxide at the industry can take place. Poisoning is possible in boilers, producer gas, blast furnace, open-hearth and foundry shops, as well as during testing of engines. Significant discharge of carbon oxide is possible during gunnery, bombardment and machine-gun fire, as well as in tank, armored cars and cartridge towers. The increased content of carbon oxide can be observed in the air of some shops of ceramic, brick, cement, construction industry, as well as engine-rooms of diesel locomotives, cockpits, garages, auto machines, motor boats and in chemical industry during synthesis of some matters, output material for which is carbon oxide. Due to the fact that the main part of carbon monoxide is carbon oxide, it is necessary to consider the possibility of domestic poisoning.

The main way for carbon oxide to get into a human organism is via respiratory organs. It can be discharged with air exhaling in an unchangeable state. Partial oxidation in the organism into carbon dioxide is possible.

Pathogenesis. According to toxic properties, carbon oxide is a strong poison, which impacts blood. High likeness of carbon oxide with bivalent iron to hemoglobin, which is almost 300 times higher than the likeness of hemoglobin to oxygen and it conditions its toxic action onto the body. Carbon oxide, squeezing oxygen from its compounds with hemoglobin, creates carboxyhemoglobin. Whereas a part of hemoglobin is inactive, what infringes the transportation of oxygen to tissues and leads to the development of hypoxia.

The number of created carboxyhemoglobin is proportional to partial pressure of carbon oxide and is inverse to the pressure of oxygen in the exhaled air. At the increased content of carbon, oxide in the exhaled air initiates the process of dissociation of carboxyhemoglobin, which is mainly over after 7 to 9 hours after single impact of carbon oxide. Such existing dependence is given in Table 8.

Table 8

Dependence between the stage of blood saturation with carboxyhemoglobin and clinical symptoms of intoxication with carbon oxide

Content of carboxyhemoglobin in blood, %	Clinical symptoms of intoxication with carbon oxide
Up to 10	Fatiguability at physical activity
10 – 20	Dizziness when moving, and headache
20 – 30	Headache, excitement, light fatigability, and fuddle consciousness
40 – 50	Headache, collapse and loss of consciousness
60 – 70	Unconsciousness and possible death
80	Fast death

Formation of carboxyhemoglobin is accompanied by the decrease of content of oxygen in the arterial blood with 20 to 12 %, arterial-venous difference up to 4-2 % (6 – 7 % in the norm), content of carbonic gas from 45 to 35 % (data in percentages are characterized with volumes of matters).

When poisoning with carbon oxide together with hypoxia, reduction of transportation form of iron in the blood takes place. Besides, at bigger concentration in blood carbon oxide has direct impact onto the cells of tissues, inhibits tissue respiration in the blood of brain, and carries out inhibitory impact onto the cytochrome-enzyme system.

Hypoxia and carboxyhemoglobin excites reflexes with carotid glomerules, have marked impact onto metabolism and the state of endocrine and vegetative system.

The boundary permitted concentration (BPC) of carbon oxide in the air of the industrial zone is 20 mg/m^3. If working for not more than one hour, BPC can be up to 50 mg/m^3; if working for not more that 30 minutes – it can be up to 100 mg/m^3; and if working for not longer than 15 minutes – up to 200 mg/m^3. Maximal single BPC in the atmosphere air is six mg/m^3, and average daily one – one mg/m^3. For residential facilities of BPC is two mg/m^3.

Pathologic and anatomic pattern. In the pathologic and anatomic pattern of acute intoxication with carbon oxide are observed in the dissemination of vascular changes. In many organs and systems (skin, muscles and brain), plethora, small and large hemorrhages, as well as degenerated changes and necroses can take place.

A characteristic sign is a relative coloration of skin and mucous tunic, which gains pink coloration.

Clinics. The clinical pattern of the acute poisoning with carbon oxide is diverse and is characterized by mainly changes of blood systems, disorder of the activity of cardiovascular and central nervous systems. The coloration of mucous tunic and skin is bright pink and intensiveness usually corresponds to the degree of the severity of intoxication.

One of the syndromes, which have a decisive meaning in the diagnostics of acute intoxication with carbon oxide, is the change in the nervous system. At the action of some concentrations of carbon oxide take place passing symptoms of the disorder of the central nervous system, which are accompanied by a headache of pulsing character, mostly in the area of temples, nausea, vomiting, dizziness, general fatigability, weakness in legs, fast heart beating, and heart weakness.

At physical activity of a patient from the area, gas-laden with carbon oxide, and the provision of a corresponding assistance, all the listed above phenomena disappear gradually.

In case of prolonged action of significant concentration of carbon oxide, a severe form of poisoning develops, which is accompanied by the loss of consciousness and comatose state with complete inhibition of reflexes. When inhaling much carbon oxide, coma can take place immediately. During the coma, trismus or lockjaw can be defined, significant rigidity of muscles of the torso and limbs, dot cramps, pathological reflexes, disorder of cardiac activity and respiration. Depending on the severity of the intoxication, state of coma can last from several hours to several days. During growing disorders of cardiac activity and respiration (it becomes very rare and superficial), death can be caused by the respiratory center paralysis.

If the progress of toxic process is more favorable, then coma is replaced by a short-term period of movement excitement, in the basis of which there is the disorder of corticosubcortical activity, which appears on the background of external boundary dormancy, which is kept in the cortex. Patients jump, intend to run, become aggressive, and cannot orient in time or space. Excitement goes away, after what they gradually lose consciousness. However, complete renewal of the psycho activity does not take place immediately. For a long time, patients are in spellbound state, which can be characterized by dormancy of psycho processes, indifference to the environment, and disturbance of memory.

In the distant period after severe forms of poisoning, in particular after prolonged coma, stable affection of nervous system can be observed. They include phenomena of Parkinsonism, which can be clinically defined in several months after poisoning. Obviously, changes, which take place on the height of intoxication in extrapyramidal system, and for some time they can develop clinically compensated. At the progressing of the process, a corresponding clinical symptomatology develop: anemia, movement stiffness and rigidity of muscular system. Peripheral sectors of the nervous system at acute poisoning with carbon oxide suffer much more rarely. Cases of the progressing of neuritis and polyneuritis are described.

If the form of intoxication if severe, swelling of retina can take place; in the fundus of eye sudden expansion of veins can be observed,

small hemorrhages along vessels, which can later cause atrophy of optic nerves. In some cases, complete lose of sight is possible, caused by the affection of central sectors of sight analyzer.

Severe acute poisoning with carbon oxide can be accompanied by trophic changes of skin and other organs. Patients, who have been under the impact of carbon oxide for a long time, often have affected skin. In the initial period, these changes of skin are more or less well-outlined erythem, which is pigmented further. In a number of cases, on the background of erythema, blisters of different sizes, filled with transudation of yellow color, which remind burns. Blisters are localized on the skin of chest, hips and limbs. They burst easily; in case of infecting, it can be complicated with purulent process.

Main pathologic processes, which are observed at acute poisoning with carbon oxide, include changes in the peripheral blood. Thus, at the light degree of intoxication, polycythemia, increase of the content of hemoglobin, sometimes, neutrophilic leukocitosis, increase of the blood viscosity and slowing down of ESR can take place. On the height of intoxication, determine carboxyhemoglobin is determined in blood.

Patients with acute intoxication with carbon oxide have changes in the cardiovascular system. They are characterized with the appearance of tachycardia, widening of heart boundaries, and tone dullness. Often, there are various types of arrhythmia can be met. On the height of intoxication, arterial blood pressure is increased.

The possibility of c h r o n i c p o i s o n i n g with carbon oxide are denied by some researchers, but others consider them the result of numerous mild acute poisonings. Patients complain to have a headache, buzzing in the head, dizziness, increased fatigability, irritability, poor sleep, worsening of memory, short-term disorder of orientation, heart beating, dyspnea, states of unconsciousness, disorders of skin sensitivity, hearing and sight. Functional disorders of the central nervous system can be observed, like asthenia, vegetative dysfunction with angiodystonic syndrome, inclination to vessel spasms, and hypertension with further development of a hypertonic disease.

Chronic poisoning causes the development of arteriosclerosis. Possible disorders of a menstrual cycle, generative function among women, as well as unfavorable progress of pregnancy, and weakening of male sex functions.

The amount of hemoglobin and erythrocytes increase in the blood, and moderate anemia and reticulocytosis can be observed.

First aid and treatment. A sick person should be immediately taken outside in the fresh air, and stay in calm state and be warmed up. Oxygen inhaling should be started as soon as possible.

At severe intoxications, urgent hyperbaric oxygen therapy is recommended for 1 to 1.5 hours, and in case of the necessity, this procedure should be repeated.

On the background of oxygen therapy, the rest of therapeutic measures should be taken. In mild cases, alcohol, tea, coffee can be used; in case of nausea – 0.5 % solution of Novocain can be used. Cordiamin and camphor can be administered hypodermically. During the first hours, 10 to 50 ml of chromosome, 20 ml of 5 % solution of ascorbic acid, 50 ml of 2 % solution of Novocain with 500 ml of 5 % solution of glucose and 1 or 2 ml of 5 % solution of pyridoxine can be administered intravenously. In case of brain swelling, the following lytic cocktail can be introduced intramuscularly: 2 ml of 2.5 % solution of aminazine, 2 ml of 2.5 % solution of Dimedrol, 2 ml of 2.5 % solution of promethazine, 1 ml of 2 % solution of promedol; 200 ml of 40 % solution of glucose (by drops intravenously) simultaneously with insulin– 10 units hypodermically can be administered. In case of cramps- enema with the solution of chloral hydrate (2 %, 100 ml) or barbamyl (10 %, 5 – 10 ml), with disorders of respiration – 2.4 % solution of aminophylline 10 ml intravenously repeatedly, lobeline (1 %, 0.3 – 0.5 ml), and artificial respiration.

Verification of work ability. After treating of patients with acute poisoning of mean form in hospital, they are provided with an occupational sick leave and they stay under observation. Depending on the presence of severity of complications, their work ability can be limited, what conditions the invalidism of the occupational character.

Patients with initial signs of chronic intoxication are promoted to another job with the provision of an occupational sick leave for two months. In case of little effectiveness of the conducted treatment and preventive measures or marked symptoms, it is recommended to promote the patient to another job permanently with possible invalidism group on the occupational disease.

Preventive measures. Sealing-in of equipment and pipelines, where carbon oxide is possible to be emitted, full-time control over the concentration of carbon oxide in the air of facilities and fast withdrawal of the gas accumulated there, and automated alarm on unsafe concentration of carbon oxide.

Individual protection: if necessary, work in gas masks and respirators.

Intoxication with Nitrocompounds and Oilsperses of Benzol and its Homologues

Nitrocompounds and oilsperses of Benzol line are rather widely used in carious spheres of the industry, first in the chemical industry (production

of organic dyes, pharmaceutical preparations, explosive matters, reagents and some pesticides).

Aromatic nitrocompounds of the benzol line, most frequently met, include nitrobenzene, dinitrobenzene, dinitrotoluene and trinitrotoluene. Most frequently used in the industry oilspereses include aniline, benzidine and aminobenzene.

Aniline is a colorless oleaginous liquid with mild aromatic smell; it darkens fast in the air; it dissolves well in ether, alcohol, and it dissolves badly in water. It can be used to produce dyes, artificial raisings, and color pencils, in pharmaceutical and rubber industries, as well as to produce explosive matters.

β - Naphthylamine is used in the production of azo dyes.

Nitrobenzene is a slightly oleaginous colorless liquid, which has the smell and taste of bitter almond. It dissolves well in ether and alcohol. It is practically non-dissolved in water and fats. It is used in the chemical industry to receive aniline, dyes, in perfumery, to produce soap and explosive matters.

Dinitrobenzene is a solid crystal matter of yellow color. Its vapors are 5.8 times heavier that the air. It is used in chemical industry as an outcome product to receive dyes and nitroaniline.

Nitrotoluene is used in chemical industry to produce synthetic dyes, and in pharmaceutical industry.

In the organism of people, these compounds get with inhaled vapors or dust, through undamaged skin as well as in case of swallowing dust thought esophagus. Particular danger is in poisoning though the skin, as oilsperse and nitrocompounds of benzol can dissolve well in fats and lipoids. Favorable factors in this are hot seasons and skin dampness.

Aromatic oilsperses and nitrocompounds of the benzol line transform into aminophenol in the organism. In liver, they are neutralized by attachment with glucuronic acid and sulphuric acid with the creation of non-toxic vapor compounds, which are discharged by kidneys with urine.

Pathogenesis. Nitrocompounds and oilsperses of the benzol line are methemoglobin forming. Under the impact of these matters, blood pigmenthemoglobin is transformed into methemoglobin, which has dark brown color. Methemoglobin differs from the normal hemoglobinby the fact that bivalent iron hema is reverse and under the impact of oxidants, it transforms into a trivalent form, losing its ability to attach and transport molecular oxygen to tissues. Hypoxemia and hemic hypoxia develop. Nowadays, it is considered that pathological pigment is not created by the very aromatic nitrocompounds and oilsperes, but products of their transformation in the organism (phenyl-hydroxyl and nitrobenzene).

When poisoning with various aromatic nitrocompunds and oilspereses, methemoglobin disappears from blood after the termination of

the impact of these matters within 2 to 6 days, renewing into an active form of heboglobin. Under appropriate conditions, oxidation of α-methyl group of porthyrin with further attachment of sulfur atom to pyrol rings progresses. Along with this, a pathological derivate of hemoglobin is created – sulfhemoglobin, which can also attach molecular oxygen. However, comparatively with methemoglobin, it is an irreversible pathological pigment, which is saved in erythrocytes until the end of their life cycle. Thus, presence of sulfhemoglobin in the blood is an important diagnostic criterion of the toxic influence of aromatic nitrocompounds and oilspereses.

Toxic impact of aromatic nitrocompound and oilsperses onto blood is accompanied by not only the creation of methemoglobin and sulfhemoglobin, but also with the appearance of inclusions in the form of oval grain, which are called bodies of Geins – Erlikh. Protein granulas, created from denaturated protein after dissociation of hemoproteid onto haems and globins are believed to take place. The number of bodies depends on the intensiveness of toxic impact. They appear more often just in several days after the beginning of intoxication. It is always a sign of a very deep damage of erythrocytes. Thus, in the basis of the creation of bodies of Geins – Erlich, there is denaturation and sedimentation of the pigment. The duration of life of erythrocytes with bodies of Geins – Erlich is reduced; they undergo mass ruining. Acute hemolytic anemia takes place, signs of which are the decrease of osmotic resistance of erythrocytes and the level of hemoglobin, ruining of erythrocytes and the increase of the content of indirect bilirubin, and marked urobilirubinuria. In the blood, there is a big amount of reticulocytes and even normoblasts. Changes of leucopoiesis are characterized by moderately marked neutrophilic with shifting to the left.

Thus, for poisoning with aromatic nitrocompounds and oilspereses of the benzol line, specific changes in blood are characteristic, like methemoglobinemia, sulfhemoglobinemia and presence of bodies of Geins and Erlich with further secondary hemolisis and the development of hemolytic anemia.

Besides, these compounds influence nervous system (like strong narcotic poisons they impact vitally important centers – respiratory, vasomotor and termoregulatory centers); liver (directoly on hepatocytes, assisting the accumulation of free fatty acids, triglycerides, lipids with the development of fat infiltration of cells, which is accompanied by the formation of strong cell poisons – alcohols, ketones, aldehydes and causes disorganization of cell metabolism and denatu**ration of proteins); cardiovascular system (directly onto the heart muscle – hypoxemic effect); skin and urinary tracts.**

Pathologic and anatomic pattern. Skin, mucous tunic of conjunctiva, tongue, and gums become pale gray, or pale yellow. Dot hemorrhages, and sometimes dingle ulcers are possible.

Blood becomes chocolate brown. Internal organs are plethoric. Marked fatty degeneration of liver can be observed, epithelium of intertwined channels are changed in a degenerated way and it contains hemosiderin. Spleen is increased, and is marked with deposits of hemosiderin.

Acute poisoning is characterized by the change of marrow. Yellow bone marrow is replaced with functioning red one. For chronic intoxication, sclerotic changes in all internal organs are characteristic.

A c u t e p o i s o n i n g. According to the clinical patter of acute poisoning with aromatic nitrocompounds and oilspereses of the benzol line, there are the following types of poisoning: mild, mean and severe.

In case of *mild poisoning*, presence of cyanosis of lips in some areas, nail plates on fingers. Face skin is pale with grayish shade. Patients complain to have weakness, drowse, and headache. The content of methemoglobin does not exceed 15 % of the general content of hemoglobin. In 1 to 2 hours after the termination of work, these phenomena disappear.

With poisoning of mean phase, the symptoms of intoxication are marked more. Well-marked skin and mucous tunic cyanosis takes place. Unusual coloration of skin and mucous tunic is conditioned by the increase methemoglobin in blood; creation of sulfgemoglobin and reduced hemoglobin. Patients complain to have acute headache, drowsiness, noise in ears, blinking in eyes, the state of shock, and sometimes loss of consciousness and decrease of muscle tone. Heart tones are dull and the pulse is accelerated. The content of methemoglobin achieves 30 to 40 % of the general content; the blood gets chocolate brown color; viscosity is increased and ESR is slowed down.

When poisoning with nitrobenzene and dinitrochlorbenzene, high level of methemoglobin is for 4 to 6 days, and when poisoning with aniline it is for 2 to 3 days.

In 3 to 4 days, pathological phenomena of the central nervous system weaken, but some changes in the peripheral nervous system can take place (pain in limbs and feeling of shivering.

In case of a *sever form* of acute poisoning with aromatic nitrocompounds and oilsperses, changes of blood go through the following three stages:

The first one: formation of methemoglobin and bodies of Geins-Erlich, which appear in blood on the 2^{nd} – 5^{th} day after intoxication.

The second one: hemolisis of degeneratively changed erythrocytes. The number of erythrocytes decreases sharply; the content of hemoglobin

decreases; the content of indirect bilirubin increases. The term of progressing of this stage is from 5 to 8 days.

The third one: renewal of the blood content. Sharp increase of the number of reticulocytes takes place; basophile – grainy erythrocytes emerge; a general number of erythrocytes and the content of hemoglobin increase.

The renewal of the blood content takes place without utilization of specialized treatment methods. Complete renewal of blood is carried out for 3 to 4 weeks.

With acute poisoning, in particular, with nitrobenzene, the development of toxic hepatitis is possible. Slight increase of the liver is observed; dyspeptic phenomena increase, like nausea, vomiting, and loss of appetite. On the 3^{rd} – 5^{th} day, icteritiousness of sclera and mucous tunics appear. Skin becomes pale with yellowish shade of coloration. In blood, there is direct slowed down reaction of bilirubin; and bile pigments – in urine are observed. The progress of toxic hepatitis is non-malignant. By the end of 2^{nd} or 3^{rd} week of hepatitis is subject to a reverse development.

Severe acute poisoning with aromatic nitrocompounds and oilsperses can be met very rarely in industrial conditions. In the clinical pattern, there are mostly general brain phenomena, loss of consciousness and coma. In case of getting out of coma, retrograde amnesia, disorientation, obnubilation, headache and drowse can be observed. Pulse is from 120 to 150 beatings a minute; and the arterial pressure is lowered down. Sometimes, the content of methemoglobin is from 60 to 70 % in blood.

Phenomena of toxic hepatitis can be observed, and there is a threat of affection of kidneys (toxic nephropathy), especially in those cases, when intravessel hemolisis of erythrocytes and hemoglobinuria (hemolytic kidney).

C h r o n i c p o i s o n i n g. Chronic poisoning with aromatic nitrocompounds and oilsperses of the benzol line should include more or less stable diseases, which appear in case of prolonged impact of small dosages of these production poisons. Under their chronic impacts, biggest disorders can be observed from the side of such systems like blood, nervous system, liver and esophagus.

Toxic anemia is characterized by the development of stable anemia, for which it is characteristic to have the decrease of the coloration indicator and reticulocytosis, and the presence of 5 – 7 % methemoglobin is observed, and up to 1.5 % sulfhemoglobin and bodies of Geihns-Erlich.

Changes leucopoiesis cause leukocitosis, and then leukopenia develops. Oxygen capacity of blood decreases significantly due to the decrease of the ability of hemoglobin to attach oxygen.

Affection of the nervous system can be characterized by complaints of patients to have headache, drowsiness, general weakness, worsening of

memory, increased fatigability, sleep disorder, listlessness, and apathy. From the side of vegetative nervous system, hyperhydrosis and labiality of the arterial pressure are observed. Further, functional changes on the side of the central nervous system progress and transit into the microorganic symptoms system with the involvement of hypothalamus, as well as the development of hypothalamic syndrome.

Toxic hepatitis is shown through complaints of the sick to have pain, heaviness in the right hypochondrium, bitterness in the mouth, and non-perception of fat food. When examining a patient, moderate increase and pain in the liver, and in some cases, positive symptoms in gall bladder can be observed. The research of the functional state of the liver, as a rule, shows affections of hydrocarbon, anti-toxic, pigment, protein forming, excretory and other functions of this organ.

Disorder of pigment exchange at toxic hepatitis is rather specific. Light hyperbilirubinemia (without a clinical pattern of jaundice), which is characteristic for these patients, conditioned by the decrease of the fraction of free bilirubin, which appears due to low activity of the ferment of β-glucoronidase. In case of moderately marked chronic toxic hepatitis, the activity of such organ specific ferments for the liver, like ornithine-carbamyl-transferase, fructose bisphosphate-aldolase, and D-sorbitol-6-phosphate-dehydrogenase does not increase. There is also no disorder of fat exchange – the content of lipoproteins and cholesterol do not leave the boundaries of physiological waves.

Duodenal probing shows the absence or the weakening of cystic reflex, changes of the chemical composition of the B bile, in particular the decrease of the concentration of cholesterol and bilirubin. At cholecystorrhaphy, there are disorders of the movement function of the gall bladder.

Thus, chronic toxic hepatitis, which develops due to the action of aromatic nitrocompound and oilsperses, is characterized by the moderate expression (persisting hepatitis), mild disorder of main functions of the liver and dyskinesia of the gall bladder.

At chronic intoxications of with aromatic nitrocompounds, pathological process involves the esophagus as well.

Patients complain to have scattered pain in the suprastomach area after eating, nausea, belching, sometimes with “rotten egg” sensation, constipation, abdominal distension, and instability of defecation.

Functional and morphological research of the stomach enables to diagnose functional secretory and motor disorders, and more seldom, gastritis with mostly saved acidity. From the side of pancreas, decrease of the activity of pancreatic ferments in the duodenal content and the increase of their concentration in blood can be observed, but these changes bear functional character.

As to the mechanism of such changes, it is necessary to consider both direct impact of aromatic nitrocompounds and their metabolites, and mediated impact due to the disorder of its neurohormonal regulation.

As to the heart and vessel system, patients suffer from the pain in heart, heart beating, and dyspnea at physical strain. Research of cardiovascular system enables to find the weakening of tones, inconstant systolic noise on the top of the heart and the inclination to hypertensia.

Changes of sight are characteristic only for some representatives of aromatic nitrocompounds – nitro derivative of toluene (trinitrotoluol). Bilateral toxic cataract of I – II degrees develops.

In case of prolonged action of some aromatic oilspereses (benzidine and β-naphthylamine), chronic irritation of the mucous tunic of the urinary bladder with products of metabolism of toxic matters (hemorrhagic cystitis, papilloma and malignant tumor of the urinary bladder) takes place. This disease can develop without symptoms for a long time and can be diagnosed only during cystoscopy.

At the action of such aromatic nitrocompounds and oilspereses, like Ursol, dinitrochlorbenzene, there is a threat of developing of allergic disease – bronchial asthma, dermatitis and eczema. There are cases of the change of the skin and hair coloration among those who work in contact with nitrocompounds and oilsperes of benzol for a long time. These matters are rather closely connected with proteins of skin and hair and dye them into brown when contacting with aniline, or in yellow – when contacting with trinitrotoluene.

Treatment. In case of development of acute intoxication, it is necessary to take the patient out of the contaminated zone. If toxic matters got to the skin, it is necessary to take off the contaminated clothes and to rinse the skin thoroughly with warm water and soap or a weak solution of potassium permanganate. Periodical inhaling of oxygen (from 20 to 30 minutes) is carried out for 5 to 10 hours. It is possible to alternate oxygen inhaling with carbogenes (5 to 7 minutes), which excites the respiratory center, improves the ventilation of lungs and assists the dissociation of oxyhemoglobin.

Good results are observed when prescribing cystamine, lipoid acid and ascorbic acid, intravenously, 10 to 30 ml of the preparation of “Chromosmone” (1 % solution of methylene blue in 25 % solution of glucose), 30 to 50 ml of 40 % solution of glucose, 20 to 30 ml of 30 % solution of sodium thiosulfate. At severe poisoning, article replacement of blood is used (3 to 4 liters) with the purpose to reduce the concentration of toxic matters and their metabolites, dilatation and reduction of blood viscosity.

With this purpose, it is possible to conduct a forced diuresis. Incase of appearance of acute hepatitis, it is recommended to administer vitamins of Group B, tocopherol acetate, as well as lipotropins.

Treatment of patients with chronic intoxication should be complex with the consideration of the general state of a patient, as well as the major affection of corresponding systems and organs.

If functional disorders of the central nervous system have been found, it is recommended to administer sedative means, and in case of hypothalamic syndrome, intranasal iontophoresis or medical ionization is prescribed: the solution of 10 % calcium chloride, 2 % Dimedrol, and 5 % sodium bromide' with the inclination to arterial hypotonia – a complex of vitamins of the group B, eleuterokok, Chinese Schizandra and beloid are prescribed.

To get rid of hypoxemia and hypoxia, oxygen therapy is carried out. At toxic hepatitis, Diet No. 5, as well as lipotropic preparations (choline chloride), essentiale and legalon are recommended. Positive results are given by repeated duodenal probing, prescription of cholagogue preparations (alcohol, cholenism and hips). Patients with marked dyskinesia, bile-excreting tracts are prescribed to administer papaverine, no-shpa, platyphyllin and atropine.

Treatment for disorders of functions of stomach and kidneys should be individual with the consideration of the phase of progressing of the disease, character of changes of the secretory and movement functions, the degree of morphological changes of the mucous tunic. Diet, vitamins and physiotherapy, as well as therapeutic exercises, small dosages of insulin (6 to 8 units) in combination with glucose (20 ml of 40 % solution) intravenously are prescribed.

Verification of the ability to work. At mild poisoning, patients are not able to work for a short period of time (for several days).

At acute intoxication of mean and severe degrees, temporary inability to work is 3 to 4 days. Then with the purpose to ensure the results of the treatment of patients, they are transferred to lighter work beyond the impact of toxic matters with the provision of a sick leave on occupational inability to work for 1 to 2 months. Further, they are considered capable to work according to their speciality.

In cases of mild chronic intoxication to ensure the treatment effect, patients are recommended to be transferred to another temporary position outside the impact of toxic matters for the period of 2 months with the additional payment if needed to provide average monthly payment according to the sick leave on the occupational inability to work. Further, they are permitted to work according to their occupation, but only under condition of keeping to sanitary and hygienic norms of labor.

If the disease is a relapse, patients should be reemployed rationally (without the loss of qualification) at another place, which is more favorable in industrial meaning. In case of impossibility of such employment, a decision is made on temporary provision of invalidism group (for 1 to 2 years) due to the occupational disease until a new profession is not acquired.

At the moderately marked form of intoxication, further working contact with toxic matters is not recommended, and patients are subjects to rational employment; and in case of the reduction of the qualification – they should be sent to the Expert Commission to acquire an invalidism group.

Preventive measures. The basis of preventive measures is further limitation of the contact with toxic matters. It can be achieved due to mechanization of production processes, sealing-in the equipment and reconstruction of ventilation. Wet cleaning should be done in premises. All those who work in possible contact with these matters, should use individual protection and should have an opportunity to take a shower at work. Those, who are being employed or employees who contact with oilspereses and nitrocompounds of benzol, should go through preliminary and periodical medical examinations.

Intoxication with Lead

Lead is a soft, silvery-white or grayish metal. In the nature, it is mostly met in the form of sulphuric lead.

Poisoning with non-organic compounds of lead are more real in the mining and metallurgic industries, as well as in the production of lead paints and pigments, accumulators, during hardening of metal items in lead baths, in production of crystal, when soldering (utilization of lead solders), when cutting metal items, which are painted (red lead), as well as in polygraph enterprises.

Under production conditions, lead gets to the organism in the form of vapors and aerosol mostly via respiratory tracts. A danger of the lead getting through a gastrointestinal tract only exists in case of failure to keep to sanitary and hygienic rules (contamination of hands and eating and smoking at work places). A low content of protein, calcium and iron in the meals of those, who work under conditions of the contact with lead, can assist tot the increase of adsorption of lead in gastrointestinal tract. In the life, lead gets to the organism mostly though gastrointestinal tract with water, meals and in the result of contamination of hands.

The permitted concentration of lead in the air of the production zone is 0.01 mg/m^3.

Lead circulates in blood in the form of highly dispersed colloidal phosphate or albuminate of lead. It is mostly extracted via a large intestine

and kidney. It can be detected in all the secretions (saliva, digestive juices, bile-excreting and breast milk). In urine in the norm – from 0.04 to 0.05 mg/l of lead; in faeces – twice or three times as much. Lead mostly deposits in bones, then in the liver and kidneys. At the disorder of acid-base balance lead can leave in the form of readily soluble lead phosphate and circulate in blood again. Such unfavorable impact can cause trauma, infection and alcoholism.

Lead is an anitplasmatic poison with a wide spectrum of the action. It causes mostly changes in the nervous system and cardio-vascular systems; disorder of ferment reactions, which participates in the synthesis of hemoglobin and a vitamin exchange; and decrease of immunobiological reactivity of the organism.

Pathogenesis. Lead interacts in the organism with active groups of proteins – sulfhydryl, amine and carboxyl. In the result, the activity of much ferment is affected; first, they participate in porphyrinic exchange including dehydratase δ-aminolevulinic acid and pherochelatese. The process of the transformation of tryptophan is affected. These changes slow down the formation of heme, and in the result of this and cytochrome, complicate the synthesis of pyridin nucleotides. In the result, energetic processes in cells are affected. Besides, under the impact of low concentrations of lead, the synthesis of RNA and DNA changes, and thus, plastic processes in cells are affected as well. It decreases adaptation opportunities of the organism and causes the increase of general sickness rate increases. The considered mechanisms are in the bases of syndromes, characteristic for the intoxication. The clinics of lead intoxication are given thought the connection merging of several syndromes.

Pathologic and anatomic pattern. At the lead intoxication in pathomorphological pattern, changes of nervous cells of anterior horns (*cornu ventrale*), where vacuolization, pigmentation, nuclear pycnosis, and dissolving of chromatophilic substance. Dystrophic changes in peripheral nodules can take place of the sympatic part of the vegetative nervous system and in general in periphery nerves. In cases of a severe affection in the brain and bone marrow, there are portions of hemorrhages and stases.

Clinics. Cardinal used to be characteristic for chronic intoxication with lead – lead border (dark gray, and sometimes, violet-flaky narrow line along the end of jaws) and the lead coloration (sallow gray color of a face) – now due to the improvement of the environment at the production, connected with lead; they lost their diagnostic meaning. Chronic intoxication with lead can be characterized with mostly affection in the blood system, affection of the nervous system and gastrointestinal tract.

Changes of biochemical indications in the blood, caused by the intoxication with lead, comprise disorders of порфириновый porphyrinic exchange; first of all aminolevulate- dehydrase reacts when an increased

amount of lead gets into the organism, the activity of which in erythrocytes decreases; the content of aminolevulinic acid, protoporphyrin and coproporphyrin increase in erythrocytes, which are considered the most reliable and specific sings of poisoning. The detected dependence of the expression of changes of porphyrinic exchange from the degree of the impact of lead. its content in blood and the severity of poisoning. Changes in the morphological pattern of blood – reticulocytosis, increase of the amount of basophile-grainy erythrocytes – refer to non-specific signs of saturnism, their diagnostic value is insignificant. Anemia at saturnism belongs to the group of hypochromic anemia, as its characteristic sign is hypochromia of erythrocytes at the increased content of iron in the blood serum (the so-called sideroachrestic anemia). In its development, a significant part is played by the direct impact of lead to erythrocytes, what leads to the reduction of the long term of their life. In the clinical pattern of the chronic lead intoxication, three stages can be distinguished:

Initial form of the chronic lead intoxication can be characterized by the absence of clinical signs and is determined based on the so-called laboratory symptoms of the intoxication. The content of aminolevulinic acid in the urine achieves 15 mg per one gram of creatine and coproporphyrin- 300 mkg per one gram of creatine. The level of lead in blood does not usually exceed 500 mkg/l, and in the urine – 100 mkg/l; reticulocytosis – up to 20 – 25 %, the amount of basophile-grainy erythrocytes increases up to 35 %.

Mild form of chronic lead intoxication is characterized by the joining of clinical symptoms. At this form of intoxication, the initial form of polyneuropathy can be diagnosed. Here, vegetative-trophic disorders can be diagnosed: pain, parasthesia, the feeling of numbness in limbs, especially at night at rest. Objectively at the neurological examination, the change of coloration of the skin on fingers can be observed (light cyanosis or paleness of the skin), hyperhydrosis, hypothermia, symmetrical distant disorders of the sensibility, first in the form of hyperstesia, and then – hyperstesia, muscular hypotonia, dormancy of dermatographism, labiality of arterial pressure, and tendency to bradycardia. The decrease of the excitement of olfactory, gustatory and visual analyzers can be observed.

Changes in gastrointestinal tract at the mild form of chronic lead intoxication are expressed through the affection of stomach secretion (increase or decrease), processes of adsorption into the intestines, intestinal mobility with the development of dyskinetic syndrome. Functional disorders of the liver are possible.

Disorders of biochemical indicators at this form of intoxication of the lead are more marked: the content of aminolevulinic acid and coproporphyrin in urine can increase up to 25 mg and up to 500 mkg per 1 g of creatine correspondingly, the content of lead in blood, as a rule, does

not increase 800 mkg/l, and in urine it reaches up to 150 mkg/l; reticulocytoosis – up to 40 ‰, and the number of erythrocytes with basophile grains – up to 60 ‰. Some decrease of the content of hemoglobin is possible.

Marked form of chronic intoxication with lead is characterized by the development of marked polyneuropathy, at this with sensitive disorders, movement disorders can be observed, and asthenovegetative disorders can develop.

The classical form of polyneuritis at the lead impact onto the body of a worker is the so-called antebrachial type of the paralysis. The syndrome is characterized by the major affection of extensors of hands and fingers (Fig. 4). The process starts with the affection of bending extensor of fingers, and later it is accompanied by paresis of other finger extensors and hands, which stays in the position at right angle in a semi prone position. Fingers are bent; a thumb bends towards the palm (the so-called “hanging hand”).

Fig. 4. Sensitive and movement forms of polyneuritis at lead intoxication

At the marked form of chromic intoxication with lead, the following can be observed very often: the so-called lead colic, which is expressed with fit-like pain in the abdomen, persistent constipation (the duration can be up to 10 – 14 days), which cannot be cued by laxative preparations; increase of arterial blood pressure, often with bradycardia, increase of the body temperature, as well as moderate leukocitosis and dark red color of the urine (due to the excretion of a big number of porphyrin). Sometimes, lead colic is accompanied by the affection of urinary tracts, and it develops as kidney colic. It is necessary to take into the consideration the possibility of the development of atypical vague forms of lead colic, progressing of which takes place during a long period of time in a wave-like form (from 3 to 4 months) and which are characterized by less marked clinic pattern and laboratory symptoms.

Recently, new data have been collected as to the mechanism of the development and progressing of lead colic. It is considered that at the action of lead onto the organism, autoantibodies are created, which, even

before the appearance of clinical indications of the lead intoxication assist to the development of immune complexes. Autoantibodies appear in the result of changes of antigenicproperties of erythrocytes due to metabolic disorders at the formation of heme or at the expense of creation of metal protein. These immune complexes, as well as erythrocytes with antigenic properties circulate in the peripheral blood, and first they affect normal blood provision in organs (at the expense of “plugging in” capillaries). It is caused by the disorder of microcirculation of organs and conditions a pain syndrome.

Nowadays under production conditions, lead colic starts gradually, with prodrome: increased fatigability in the end of a work day; general indisposition; pain in cortical bones, muscles and in the waist zone; loss of appetite, inclination to delaying of bladder emptying, irritability and sleep disorder. Sometimes, these phenomena appear together with pain in the stomach, which increase much and get cutting character.

For the marked form of chronic lead intoxication, the development of the anemic syndrome with the decrease of the level of hemoglobin lower than 130 g/l in men and 120 g/l in women is characteristic.

At the prologuned contact with lead, affection of the determined portions of bones and limbs can be noted: appearance of homogeneous levelly darkened intensive shadows in the metaphases in long cortical bones, which are much separated from the diaphyses of bones. Changes in the bone tissue at the intoxication with lead are not accompanied by the destructive processes, changes in periosteum are absent. Mostly, large and small cannon-bones, hip, shoulder, elbow and spoke bones, as well as ribs are affected.

Biochemical disorders at the marked intoxication with lead are the most expressed. The content of the aminolevulinic acid and coproporphyrin in the urine is over 25 mg and over 500 mkg per 1 g of creatinine correspondingly. The concentration of lead in blood achieves 800 mkg/l and higher, and in the urine 0 over 150 mkg/l; reticulocytosis is higher than 40 ‰; and the number of basophile-grain erythrocytes is over 60 ‰.

Treatment. The most effective therapeutic means at chronic intoxication with lead is complexing agents, which create strong non-dissociating small toxic complexes together with lead, which can be easily taken out of the organism though kidneys. Mostly, 10 % solution of titacin calcium, which is administered intravenously once or twice a day for two to three days (20 ml in 500 ml of 5 % solution of glucose). Pentacin (especially at lead colic). Both preparations have high extracting activity regarding lead and are capable to terminate one of the most complex manifestation of saturnism – lead colic. Preparations are administered intravenously. Pentacin is administered in isotonic solution of sodium chloride or in 5 % solution of glucose – 200 ml once or twice a day, a daily

dosage 2.0 to 4.0 g. The course of the treatment comprises three stages from 3 to 5 days of breaks between cycles. To treat intoxication with lead, D-penicillin is used in the daily dosage of 600 to 900 mg; it should be administered in 30 minutes after meals.

When treating patients with chronic intoxication with lead, which is accompanied by the neurological disorders, the following can be used: vitamins of group B; ascorbic acid; preparations, which have spasmolitic action, ganglionic blocker, and physiotherapeutic methods of treating.

If anemic syndrome is defined, the following is recommended: preparations of iron with hydrochloric acid, hemostimulin (hemostimulating agent, three times a day throughout a week); vitamin therapy – vitamin B_{12} (100 mkg) every other day intravenously (15 injections), vitamin B_6 intramuscularly (10 injections).

In case of kidney syndrome, the following is recommended: diathermy of the area of the liver; intravenous injections of 20 ml of 40 % glucose solution, hypodermic insulin injection (5 units); 300 mkg of vitamin B12 (10 to 12 injections); as well as vitamin K: 1 pill for five days.

Verification of the ability to work. The issue on verification of the ability to work at saturnism is solved depending on the expression of poisoning. At the initial form of intoxication, it is necessary to promote a person to another temporary workplace beyond the contact with lead for 1 to 2 months. In future, such patients can return to the same workplace (under condition of complete normalization of indicators of porphyrin exchange). In case of relapses of the intoxication, the worker has to terminate the contact with lead completely.

At the expressed form of intoxication, patients should be released from work with lead completely, even when complete disappearing of signs of saturnism can be observed in the result of treatment.

Preventive measures. The most effective preventive measure is, certainly, replacing lead and its compounds with other non-toxic matters at corresponding productions.

Maximum mechanization of operations of processing of materials which contain lead; sealing-in of sources of dust discharge; equipping of production zones with rational ventilation, mechanical purification of work premises from dust. In premises with much dust, people should work in respirators or industrial filtering gas masks.

When working with lead and its compounds, it is necessary to keep closely to the rules of personal hygiene, prohibit eating at work places; smoking should be permitted only on specially equipped rooms. Significant role in prevention of intoxication with lead is on preventive eating products with pectin matters (fruit non-clarified juices and apples), as well as preliminary and periodic medical examinations.

Intoxication with Hydrogen Arsenate

Hydrogen arsenate is a heavy colorless gas. Under industrial conditions, it is a by-product, which is created under the action of technical acids onto metals (tin, copper and bismuth) and compounds with arsenic. Hydrogen Arsenate is extracted when soldering and treatment of metal products with acids, filled in accumulator batteries on submarines and galvanization. The permitted concentration is 0.3 mg/m^3.

Hydrogen arsenate permeates into the organism through respiratory organs, and less often through undamaged skin and gastrointestinal tract.

Pathogenesis. Arsenic compounds block sulfahydril groups of ferments and create stable toxic complexes. Arsenic disturbs carbohydrate and lipid exchange, as well as tissue respiration. In the pathogenesis of intoxication with arsenic, capillary toxic and hemolytic effects, as well as irritating action onto the skin and mucous tunic.

Hydrogen arsenate is a strong hemolytic poison and methemoglobin creator. Mechanism of hemolytic action of hydrogen arsenate, which has not been clarified yet. Probably, hemolysis is conditioned by a number of factors:

1) Inhibition of catalase, which is contained in erythrocytes, in the result of which a big number of hydrogen peroxide, which causes hemolysis;
2) formation of the following products inside erythrocytes, first of all, which have the hemolytic property, first of all, hydrolases; together with this hydrogen arsenate destroys glutathione, which can inhibit hemolysis;
3) accumulation of metal arsenic in erythrocytes, which ruin the structure of the latter, what causes their dissociation.

The result of hemolysis is anemia of parenthymatosic organs, nervous and cardiovascular systems. Changes of intermediary metabolism, which is characteristic for hypoxemic states, can be observed: increase of the content of sugar and lactic acid in the blood, and decrease of alkaline reserves in blood.

Thus, anoxia, which develops at intoxication of hemolythic character, conditions the general hypoxia with accompanying manifestations. Serious disorders of functions of kidneys and liver, which can be observed at the action of hydrogen arsenic, cannot be explained but by hemolysis. An important part is played by direct action of arsenic, which is formed in the process of oxidation of hydrogen arsenate.

At the action of hydrogen arsenate in people can be observed in blood (hemolytic anemia, a big number of retyculocytes, basophile-grain erythrocytes appear, and the number of SOE is increased), of kidneys (coloration of urine change – the color of “meat slops”, proteinuria, high specific gravity and uremia can be observed), of liver (toxic hepatitis

phenomena), of cardiovascular system (increase of heart sizes, tachycardia, and arterial hypotonia), of nervous system (headache, dizziness, drowse or sudden excitement).

Clinics. A c u t e p o i s o n i n g appears suddenly. On the clinical progress, three forms of acute poisoning with hydrogen arsenate can be distinguished.

Mild poisoning can be characterized by general weakness, illness, headache, nausea, desire to vomit, and pain in limbs. Sclera and skin coverlets are weakly icteric. Sometimes, blood urine can take place. The number of erythrocytes and hemoglobin in blood gradually decreases. The aforementioned phenomena disappear fast and recovery takes place in several days.

With poisoning of *mean severity* besides the given above complaints, patients are chilling, they get much ache in the waist, and temperature rises. On the first day, jaundice develops with specific bronze coloration of skin coverlets. In the coverlet, the content of indirect bilirubin increases; the number of erythrocytes and hemoglobin decreases; retyculocytes and basophile-grain erythrocytes appear. Hemoglobinuria, albuminuria and detritus occur. Often, on the third or fourth day, increased liver, moderate tachycardia can be observed; systolitic noise can be heard on the upper part of the heart; and blood pressure decreases. When treating, improvement of the health state occurs gradually, and recovery takes place in four to six weeks.

Severe poisoning. After the latent period, which lasts for 2 to 3 years, first indications of intoxication take place: intense headaches, feeling of cold, increase of temperature up to 38 – 39 ^{0}C and vomiting.

Urine gets brown coloration and contains a great number of renewed hemoglobin and protein can be found in it. Then, sudden fall of the number of hemoglobin and erythrocytes can be observed; the content of bilirubin increases. At the end of two to three days, there are symptoms of affection of livers, and direct bilirubin can be determined.

If the disease progressing, kidney decompensation can develop, which is observed on the fourth or fifth day and is characterized by the presence of uremia, and marked hemolytic of jaundice. Headache and drowse increase, nausea and vomiting appear. Further, urine is terminated and phenomena of uremia take place. Death occurs mostly at the end of the first week.

At relatively favorable progressing of the disease, the recovery period can be accented. The state of the patient gradually improves, jaundice disappears, the number of erythrocytes and hemoglobin increases. Complete recovery can be stated only in 2 to 2 ½ months.

Distant consequences of poisoning of hydrogen arsenate can involve the following: severe anemia and obesity of parenchymal organ, as well as

the decrease of resistance of organism as to the infection; secondary non-specific pneumonia, changes in kidneys in the form of glomerulonephritis; chronic hepatitis and disorder of functions of bile-excreting tracts.

C h r o n i c i n t o x i c a t i o n. It occurs very rarely. For it, secondary anemia is characteristic, which develops without marked clinical manifestation of hemolysis.

Patients complain to have headache, sleep disturbance, bad appetites, pain in epigastric area, indigestion disorder, fast fatigability, loss of weight, and sometimes - petechial hemorrhage on the skin and general asthenia. In the blood, anemia, basophile grain of erythrocytes, and moderate leukocitosis can be found.

Treatment. Confinement to bed, complete tranquility. Warmth is recommended. During the first hours of the development of hemolysis – bloodletting in the amount of 300 ml of blood with its further transfusion, and intravenous administering of glucose for 500 ml of 10 % solution of ascorbic acid (300 – 500 mg) by drops. Hypodermic injections of isotopic solution of sodium chloride – up to one liter. Rectal drips of 5 % solution of glucose (500 ml), insulin (5 – 10 units) hypodermically. Administering of alkaline. Diathermy of spots of kidneys. Cordiamin, Corazol and adrenalin. With strong vomiting – injections of morphine with magnesium sulfate. Intravenous administering of 20 to 30 ml of 30 % solution of sodium thiosulfate. Oxygen inhalations. Immediate administering of mecaptid hypodermically or intramuscularly in the dosage of 1 ml of 40 % oil solution: the first day – three injections every 4 to 5 hours, and on the second and third days – two injections in 8 to 12 hours.

At stable anuria, usage of peritoneal dialysis, hemodialysis with the utilization of artificial kidney apparatus and generative restorative treatment.

Verification of the ability to work. After severe acute intoxication, as well as in case of marked chronic obligatory release from the contact with hydrogen arsenate and other toxic matters.

Preventive measures. General sanitary and hygienic means. Chemical control on the state of air environment. Previous and periodical medical examinations.

OCCUPATIONAL DISEASES WITH PREDOMINANT AFFECTION OF HEPATOBILIARY SYSTEM (TOXIC HEPATITES)

Wide development of chemical industry, organic synthesis, and utilization of pesticides caused the increase of the number of cases of hepatitis. This group of diseases includes affection of liver, caused by chemical agents, which are used at the production.

The most systemized classifications the classification of hepatoxic matters with the consideration of chemical structure:

- chlorinated hydrocarbon;
- chlorinated naphthalene and biphenyls;
- benzol, its homologues and derivatives;
- some metals and metalloids.

1. Chlorinated hydrocarbon (methyl chloride, chloroform, carbon tetrachloride, ethyl chloride, dichloretan, and tetrachloritan) is widely used in machine engineering, airplane and vehicle manufacturing, as well as in production of shores, to clean clothes, during worming, disinfection and disinfection.
2. Chlorinated naphthalene and biphenyls are hard wax-like mass of yellow or brown coloration. Under the name of “galowax”, and “savol” to cover electric wires, filling in condensators; they can be used as replacements of resins, wax, and caoutchouc.
3. Due to the development of industrial chemistry, benzol, its homologues and derivatives (nitrobenzene, toluol and aniline) are wider used to produce aromatic compounds, organic dues and explosive materials.
4. Metals and metalloids (lead, mercury, gold, manganese and phosphorus) are gradually replaced by less harmful compounds in the industry.

Liver completes an antitoxic function, independently of the ways of permeating and the place of the action of poison in the organism. At the same time, different on their construction, matters can cause affection of liver. Some of them, the so-called hepatobiliary poison, have particular similarity with tissues of the liver and cause specific hepatobiliary effect, getting to the organism in small dosages. Selection of a group of hepatobiliary poisons, without having bad tropism to the liver tissues, but still damage the latter, without affecting some functions.

Pathogenesis. Due to the action of the majority of hepatoxic matters, direct affection of parenchyma and disturbance of exchange ferment processes in its tissues take place. Depending on the chemical nature and the dosage of poisoning change the mechanism of its action. Neucrosis of hepatocytes at poisoning with tetrachloride carbon is the result of the disturbance of fermentative systems of endoplasm reticulum. In other cases, degenerated changes of hepatocytes under the impact of tetrachloride carbon and allyl alcohol are connected with the intensification of re-oxidation of unsaturated fatty acids of membrane lipids. In case of the action of heavy metals, pathogenesis of the damage is caused by the blockade of sulfhydril of ferment groups.

Affections and other ferment systems of the liver – cholinesterase (mercury) and phosphatase (fluorine) are possible. Particular attention is

paid to the disorder of intralobar changes of corresponding areas (chlorinated hydrocarbons). At the action of trinitrotoluene in the organism, deficit of cystine due to its connection with nitrogroups takes place.

Besides, main (hepatoxic) actions, allergic affection of the liver (ursol) can take place. Some poisons cause both direct toxic and sensibilizing action (gold salts, some compounds of arsenic, and sulfanilamide substances).

Among syndromes, which characterize the affection of the liver, conditioned with the impact of occupational indicators, the syndrome of cytolisis and cholestasis prevail (excretory- biliary). The syndrome of cytolisis develops in the result of functional inferiority of hepatocytes at the change of permeability of their membranes with further hyperferremia. The latter, which develops with the increase of the activity of ferments, is characteristic for acute intoxication with hepatotropic poisons. Hyperbilirubinemia with increased content of fraction of free bilirubin in the blood serum at chronic toxic hepatitis can be explained by fermentative disorders – decrease of the activity of glucuronid – transferase system of hepatocytes.

Under the impact of low concentrations of chemical matters, which have sensibilizing action, for example, formaldehyde, toxic and allergic reaction of the liver is manifested through signs of cholestasis (increase of the activity of alkaline phosphatase of the blood serum) and initial indicators of the affection of intermediate tissue (increase of thymol indicator).

It is necessary to accentuate that preliminary affections of the liver with alcohol and viral infection increase the sensibility to hepatotropic poisons. Toxic effect is intensified at the bacterial infection and deficit of proteins in the meals.

Pathologic and anatomic pattern. Toxic affection is manifested with various morphologic changes in the liver: massive and submassive necroses of parenchyma, fatty degeneration or ballooning degeneration. The term “hepatitis” in the majority of cases does not correspond to the morphological pattern of acute toxic damages; some researchers consider changes in connective tissue at toxic affection as a secondary inflammatory reaction onto degenerated affection of cells of the liver, and it is possible to indicate it as “reactive hepatitis”. In case of appearance of acute poisoning with matters of hepatotropic action, the picture of zonal affection of the liver can be observed (fatty degeneration, necroses of cells in the central part of the lobe), what as a rule is over with complete renewal of the normal structure of the liver. At acute intoxications, massive liver necrosis can develop, which is sometimes transformed into a large-nodule postnecrotic cyrosis.

For patients with chronic intoxication, the most characteristic morphological indication of the affection of the liver is some stage of fatty degeneration – from the diffusive to the nuclear ones, which is often united with albuminous degeneration.

Clinics. According to the clinical pattern, the following types of toxic hepatitis can be distinguished: acute and chronic ones.

A c u t e o c c u p a t i o n a l h e p a t i t i s is one of the clinical syndromes of the acute poisoning at short-term impact of the high concentration of toxic matters. They appear during accidents, disorders of safety techniques and rules of keeping of toxic matters. These matters permeate through lungs.

The clinic of toxic hepatitis reminds viral hepatitis, however it has no preicteric period. Acute affection develops on the second – fifth day after the impact of toxic matters in comparison with high concentrations. The disease is manifested through the increase of the liver size, pain at palpation and increasing jaundice. Increase of the activity of intercellular ferments in the blood serum can be observed (alanine aminotransferase, aspartate aminotransferase, fructosemonophosphataldolase, lactate dehydrogenase, hyperbilirubinemia, urobilinuria, and bile ferments). Besides, hypoalbuminemia, decrease of β- lipoprotein and phospholipins of blood. The system, which are responsible for blood coagulation and anti-coagulation of the blood, are affected; hypocoagulation takes place; fibrinolytic activity increases; the content of heparin increases and hemorrhagic syndrome develops.

Patients with mild and mean forms of the disease have blurred passing jaundice, and with severe forms, it is intensive, with hemorrhagic syndrome. Severe forms of toxic hepatitis can cause acute liver decompensation. Significant meaning is possessed by the accompanying affection of kidneys.

If to follow the development of acute poisoning, then sings of the affection of the liver have cyclic character.

The *first period* is characterized by symptoms of the affection of the nervous system. Headache, dizziness, nausea, vomiting, and disorder of coordination take place. In severe cases, a patient can lose consciousness, with the development of the narcotic coma with severe affection of the respiratory and vasomotor centers. Main danger of this period is in the possibility of the development of coma and complications, conditioned by narcosis (aspiration of vomiting masses and asphyxia due to sunken tongue).

The second period of intoxication starts with the end of the first day, when symptoms appear, which prove the liver affection. Phenomena of acute toxic hepatitis depend on the stage of the expression of inflammatory and necrotic processes in the liver tissues.

In case of light poisoning, jaundice can be absent. In more severe cases, there is pain in the right hypochondrium, and jaundice develops gradually. The characteristic fever, caused by resorption of necrotically changed liver proteins. On the second – fourth day of the intoxication, signs of functional decompensation of liver take place: phenomena of hemorrhagic diathesis increase (shadows under the eyes and epitasis); jaundice increases; and sleep is in disorder. Liver increases. Biochemical research show the decrease of prothrombin index, cholesterol, sudden increase of the level of the activity of ferments of the "necrosis" (aminotransferase and aldolase), the amount of bilirubin in blood increases, mostly by its direct fraction.

In some cases, toxic hepatitis can cause acute atrophy of liver. Liver decompensation is shown through inhibition, change of the sleep rhythm, tremor, increase of the content of ammonia in blood and significant hemorrhagic syndrome.

Acute intoxication can be ended with the development of hepatic coma. Significant meaning is possessed by accompanying toxic hepatitis of kidney affection. When researching the urine, it is possible to define protein and formed elements. However, only on the 5^{th} to 7^{th} day of the poisoning, more marked symptoms of acute kidney decompensation start showing.

The third period of intoxication is characterized by the expressed decompensation of the kidney function, in the base of which there is acute toxic necroso-nephrosis. Oliguresis is replaced by anuria. In the blood, the concentration of nitrogen scora increases (residual nitrogen, urea and creatinine).

Thus, the peculiarity of the acute poisoning with poisons of hepatotropic action is recurrence of the clinical pattern, conditioned by marked disorder of the functional state of the liver and kidneys, until the development of sings of their decompensation.

Together with general clinical manifestations, which are characteristic to all the toxic hepatitis, some patients have specific affections, characteristic toe some poisons. In case of massive impact, chlorinated hydrocarbon cause narcotic action. Neurological disorders prevail in the form of dizziness, ataxy, dormancy, psychomotor agitation, and in more seldom cases, state of coma. It is also joined by toxic hepato- and nephropathy. At acute poisoning with tetrachloride carbon or chloride naphthalene, jaundice appears on the first or second day after poisoning, and is accompanied by acute increase of aminotransferases of the serum at the unchangeable proteingram with severe affection of kidneys. The lethal end takes place within the first two weeks of the disease.

For intoxication with trinitrotoluene, methemoglobinia and appearance of Geins body in blood are characteristic. Liver affection at the

action of aniline develops on the background of the nervous system and blood (due to formation of methemoglobin), which are characteristic for the later.

In the majority of cases of toxic hepatitis after the termination of the contact with hepatotropic poisons, there is dyskinesia of the gall bladder and biliary tracts. After severe poisoning with massive necroses of parenchyma, macronodular cyrosis of the liver can develop.

In correspondence to the current existing classification of *chronic* diseases of the liver, toxic hepatitis with its clinical and morphological indications and the progressing, it is closest to chronic persisting hepatitis and of the so-called “non-specific” reactive hepatitis.

The disease appears in case of prolonged impact of subtoxic dosages of hepatotropic matters. The affection of workers’ livers, who are in contact with small concentrations of toxic matters, is manifested in the form of hepatobiliary syndrome and functional decompensation of biliary cells and are characterized by dyspeptic and pain phenomena.

Patients complain to have bitterness in their mouth, decrease of appetites, unstable emptying, dull pain in the right hypochondrium, which is intensified after spicy or fat food. During the time of objective examination, often weak icteritiousness of scleras, more seldom, the jaundice of skin coverlet, increase of liver sizes, pain when palpating, positive symptoms, and irritability of gall bladder (Orthner, Merphy and phrenicus-symptoms). “Vascular stars” and “palmar erythema”, and increase of spleen can be rarely met.

Pain syndrome can be explained by dyskinesia of bile-excreting tracts. It takes place even in the initial stages of the impact of toxic matters. On the background of dyskinesia of the gall bladder, secondary infection can develop, and signs of cholesterol can be observed.

Chronic toxic hepatitis develops for a long time. Its progress is non-malignant, without the inclination to progressing. Mild forms of the disease have the tendency to reverse development. Stabilization of the process with patients both with mild forms of the disease and with forms that are more marked is possible. Severe progressing of toxic hepatitis can be rarely observed. Usually, it can be met in case of the affection of the liver, which has mixed character; for example, on the background of the past viral hepatitis or alcohol abuse.

More severe progressing of toxic hepatitis can be observed among workers with long period of work, among elderly people, and in case of the presence of dyskinetic syndrome.

The clinical picture of chronic affections of the liver, in case of the action of various poisons, can have its peculiar progressing. For instance, toxic affection of the liver among patients with chronic intoxication with benzol takes place on the background of the affection of the haematogenic

system; with intoxication with trinitrotoluene – the development of the professional cataract, functional disorders of the nervous system, and unstable anemia; with chronic intoxication with lead – changes in the blood and nervous system. Summing up the given above, it is possible to make the conclusion that in the clinical pattern of intoxication, liver affection can be a prevailing syndrome (poisoning with dichlorethane, tetrachlorated carbon, or trinitrotoluene). At some intoxication, the liver affection takes place in parallel with the development of other symptoms or fades into the background (lead, benzol or fluorine).

Thus, the main peculiarity of toxic and chemical affection of the liver is the following:

1. Toxic hepatitis, no matter how little it is marked, it never develops in isolation, but always on the background of general phenomena of acute or chronic intoxication. At acute intoxications, this pattern is more marked.
2. Acute toxic hepatitis appears fast without syndromes on the background of general toxic action of the poison. Jaundice does not always take place. Diagnostic meaning is possessed also by the simultaneous affection of other parenchymatous organs, mostly kidneys, in particular in case of peroral intake of the poison (dichlorethane and tetrachlorated carbon).
3. Chronic toxic hepatitis is characterized by the lack of symptoms. Its progressing is rather favorable with long remissions. Functional disorders of the liver and dyskinesia of bile-excreting tracts can be saved for a long period of time. Cyrosis can be very rarely observed.

At chronic intoxications, fatty hepatitis can be formed; chronic persisting hepatitis takes place, and in some cases, latent forming of liver cyrosis is possible.

Treatment. At acute poisoning with matters of hepatropic action, first, it is necessary to terminate its intake into the organism. Further actions should be targeted at the neutralization of the poison and its excretion from the body. It is recommended to conduct gastric lavage with 10 to 15 liters of water with further administering of 100 to 200 ml of liquid paraffin or 30 to 50 g of salt laxative in case of peroral administering of the poison; even in case of minimal signs of intoxication, artificial diuresis with the utilization of diuretic means (urea, mannitol, or furosemide), as well as peritoneal dialysis and hemodialysis.

Particular place is taken by antidotal therapy. At acute poisoning with heavy metals (lead or mercury), it is recommended to administer sodium thiosulfate intravenously; and unithiol – intramuscularly or hypodermically. At acute poisoning with iron salts, it is recommended to administer deferral: internally, 5 to 10 g of the preparation dissolved in

drinking water and intramuscularly - 1 to 2 g of the preparation every 3 to 12 hours.

Pathogenic therapy includes lipotropic preparations – 30 ml of 20 % solution of chlorine chloride together with 600 ml of 5 % solution of glucose is administered intravenously in drops; vitamin of the group B; antioxidants – vitamin C intramuscularly 1 ml 4 to 6 times a day; antiprotease preparations – trasilol and contrical 500 000 units a day intravenously by drops in 5 % solution of glucose or isotonic solution of sodium chloride. It is recommended to administer cocarboxylases intramuscularly 150 to 200 mg a day; glutamine acid (up to 8 g a day) intramuscularly and antibiotics. In case of indications, symptom therapy is recommended (tranquillizers, and cardio substances). In cases of severe poisoning, it is recommended to administer intravenously glucocorticosteroid hormones.

Treatment of chronic toxic affection of the liver includes curing methods, which are conducted in case of presence if some intoxication, as well as the treatment of the liver affection itself.

At the mild form of the disease, curing diet, vitamin therapy (especially, vitamins of the group B), cholagogic means, and duodenal intubations are recommended. Antibiotics are recommended in the cases, when toxic hepatitis is complicated with inflammatory processes in biliary tracts.

With more marked forms and acute chronic toxic hepatitis, it is recommended to undergo hospital treatment. In case of the presence of toxic hepatitis, which develops for a long time, it is recommended to prescribe sirepar or other preparations of the cattle liver.

Treatment with steroid hormones and cytostatics are prescribed in case of sings of high activity of the process in the liver, what is rarely met in the clinics of occupational hepatitis.

Sanatoria and health resort treatment is recommended at moderate disorders of the functional state of the liver and the presence of dyskinesia of the biliary system, beyond the phase of the acute stage. Balneology resort centers are recommended, like Berizivsky Mineral Waters and Truskavets.

Verification of the ability to work. At acute intoxications with hepatotropic poisons, the ability to work is determined by the severity of intoxication and the possibility of reversibility of the pathological process. Mild stage of the acute intoxication envisages the possibility to return to work under the condition of dynamic follow-up and keeping to sanitary standards.

In case of severe diseases, presence of jaundice, high hyperfermentis, decrease of functional tests, and increase of the liver, the patient should undergo treatment in hospital conditions with the following sanatoria and

health resort treatment. In the future, this person should be transferred to the job beyond the contact with toxic matters for the period of two months, with the payment in compliance with the occupational list of inability to work.

In case of stable residual phenomena of intoxication or in case of the transfer of the disease into a chronic form, the patient should be rationally employed beyond the contact with toxic matters.

If the patient has toxic hepatitis, it is recommended to transfer him/her to work beyond the contact with toxic matters (rarely temporary – up to two months, in case of favorable outcome). In case of stable disorders of the functions of the liver, the patient should be transferred to the work beyond the action of toxic matters for a long period of time with further re-qualification and provision of the invalidism group for the period of re-qualification on the occupational disease.

Preventive measures. Prevention of occupational hepatitis is in keeping to safety rules, correct keeping of toxic matters, and general and individual hygiene. An important place is taken by a correct occupational selection of workers at the manufacturing, where contact with hepatotropic poisons takes place, as well as full-value meals with sufficient quantity of protein, and vitamins; and exclusion of alcohol abuse. It is important to have periodical medical examinations of people, who work in contact with matters of hepatotropic actions, with the purpose to find early and most reversible forms of the disease.

OCCUPATIONAL DISEASES WITH MAJOR AFFECTION OF KIDNEYS AND EXCRETORY TRACTS

Etiology and pathogenesis. Pathology of kidneys and excretory tracts is taken by relatively small spread among the expanded group of occupational diseases.

The contact with the kidney parenchyma and excretory tracts with toxic matters, accumulation of these matters and their transformation in kidney structures is determined by the possibility of the affection of kidneys and excretory ways. The character of the affection of excretory system depends on the chemical composition of the compounds, concentration, tracts of their permeating to the organism, health state, and especially the health state of kidneys. Depending on the localization of the affection and the character of the pathological processes, chemical compounds can be divided into two groups.

The first one includes those compounds, which mostly affect parenchyma of kidneys and cause the so-called toxic nephropathy. Toxic nephropathy (toxic nephritis) means functional changes or structural changes in kidneys, caused by the impact of exogenous chemical products and their metabolites. Development of toxic nephropathy is caused by

chemical matters, used in national economy: metals and their salts (lead, and mercury), nitrogenated compounds (aniline, nitrobenzene and ammonia) and their halogen derivatives (carbon tetrachloride and hexachloroethane), glycols (antifreeze), ethers (dixan and ethyl acrylate), as well as carbon oxide, acids and other matters.

Occupational diseases of kidneys can be observed among workers, engaged in the production of synthetic caoutchouc, polymer materials, and chlororganic poisonous chemicals. Utilization of the latter in the agriculture caused frequency of the affection of kidneys among the population as well.

It has been stated that the affection of kidneys often appears if the concentration of dust and vapors of nephrotoxic matters in the air of o production premises is higher than the permitted norm. Permeability of poisons into the organism is activated in the process of production activity, especially under conditions of increased temperature of the environment. Frequency and intensiveness of the affection grows in compliance with the increase of the work period in contact with poisoning chemicals.

Poisons permeate into the organism mostly through the digestive apparatus and respiratory apparatus, though other ways are possible as well. Thus, nickel and cobalt permeates through the skin in toxic amounts, and are accumulated in the liver and kidneys in the form of crystals.

Decisive meaning is possessed by the direct impact of poisonous chemicals onto the kidney parenchyma, however the functional capability of kidneys can be also affected by the changes of neuroendocrine regulation of the organs and due to vasomotor disorders.

Disorders of kidney hemodynamics, decrease of the kidney blood flow on the background of the disorder of the general blood flow in response to the chemical trauma is one of the pathogenic mechanisms of the toxic affection of kidneys.

There are cases, when toxic effect is provided by not only chemical matters, which permeated into the organism, and their metabolites (e.g. dock acid when getting poisoned with glycols or products of interaction with other organs and tissues, in particular hemoglobin when affected with hemolytic poisons).

Obstruction of kidney channels takes place with products of hemoglobin degradation (poisoning with arsenious hydrogen, essence of vinegar or copper sulfate), myoglobine, crystals of oxalates (poisoning with glycol or dock acid). Immune mechanism of the affection of kidneys (toxic-allergic) is possible, when acute renal insufficiency develops in case of permeation of small quantity or little toxic chemical compounds. Increased individual sensitivity to the chemical matter is significant.

In case of toxic nephropathy, there are changes of the activity of a number of ferments in the blood and urine, processes of re-amination in the

mitochondrion of livers and kidneys; the content of amino acids in biologic environments, what proves the disorder of intercellular processes, and increase of the resistance of cellular membranes. There are the data regarding the role of the hyperaminoaciduria, caused by toxic irritation of mucous tunic of the urinary bladder and can lead to the appearing of hemorrhagic cystitis, non-malignant (papilloma) and malignant (cancer) tumors of the urinary bladder. These are mostly aromatic amino compounds (benzidine, dianisidine as well as α- and β-naphthylamine) used in the production of dyes.

Clinic. In case of permeation into of the significant quantity of nephrotoxin into the organism during a short period of time, acute renal insufficiency can be observed, progressing of which has four stages: initial (shock); oligo and anury one; renewal of diuresis or poliuric; and recovery.

Clinical signs of the *initial stage* are usually symptoms of the main disease, and in particular hemodynamic disorders in parenchymal organs, and in particular in kidneys. The definitive sigh, which should be particularly considered, is a circulatory collapse, which is sometimes unnoticed though their short term of duration.

The decrease of the arterial pressure is accompanied by the decrease of diuresis. Symptoms of the initial stage are often unnoticed due to the severity of the main disease and the shock.

The duration of the initial stage – from several hours to one to three days.

In the *second (oligo-anury)* period of the acute renal insufficiency, it is possible to notice sudden decrease of complete termination of urination. Often, the disease develops unnoticed. After the normalization of hemodynamic disorders, patients feel a little better, and a period of imaginary well-being takes place, which lasts fro 3 to 5 days. However, at this moment, less and less urine is produced, and its relative thickness decreases (up to 1007 to 1010), at the same time the content of urea, creatine, nitrogen and chlorides in the daily amount of the urine. In cases of hemolisis or moilisis, hemin pigment can be found in the urine. Many erythrocytes and leukocytes, epithelial cells and bacteria can be observed.

On the 5^{th} to 7^{th} day, patients start feeling much worse. They become drowsy, adynamic, lose appetite; patients suffer from vomiting and thirst. Depending on the “background” of the acute renal insufficiency, the body temperature can be normal or increased. Due to the decrease of the resistance of the body and in case of purulo-septic complications, the temperature increases insignificantly, however, it can be subfebrile among some patients without the presence of infectious complications.

“Uraemic” intoxication and changes in water-electrolytic homeostasis often cause the affection of the consciousness; Patients do not orient in space and time. In addition, sometimes, there are “convulsive

crises", which remind epilepsy. When dehydration takes place, asthenia and drowse come along with anxiety, acute psychosis and hallucinations. In very severe cases, coma can be observed.

In case of prolongued anuria, skin becomes dry and then peels off. In addition, often, rash takes place, which reminds scarlatinous or measles rash. At intravascular hemolisis, skin and sclera are icteric. Through the disorder of coagulating properties of the blood, hemodermic hemorrhages, and in particular, in the injection areas and on conjunctive take place. A tongue is dry and is covered with white or brown incrustation. Often, stomatitis can develop. Vomiting with stomach mucus and bile can take place.

During the initial period, oligo– and anuria stages, constipation can be observed, which is replaced with diarrhea in case of the growth of azotemia. When palpating the stomach, the abdomen is painful. Coarse breathing can be heard in lungs, and in severe cases, there are stagnant crepitations can be heard in lower portions. At hyperhydration, which takes place in the result of irrational introduction of liquid, lung swelling takes place – aquatic lung? Effusion into the pleural cavity can be observed. At the significant acidosis, dyspnea increases, and patients who are in severe state have breathing after the type of Kusmaul.

Cardiologic affection can be shown through miocarditis (dull cardiac tones, systolic noise, size increase and pain in the heart, as well as changes on the EKG.

The most serious changes from the side of the heart can be observed in oligo- and anury stage in the result of changes in the content of potassium in blood. At hypercaliemia, bradycardia, arrhythmia, dyspnea, and vascular insufficiency develop; and changes on the ECG can be determined.

Changes in the blood can be characterized by the marked hypochromic anemia, the decrease of the number of erythrocytes and the decrease of the content of hemoglobin. Anemia is well marked in the beginning of the acute renal insufficiency.

During the period of oligo- and anuria, concentration of urea and creatinine has sudden increase in the blood plasma. Hypoproteinemia with the decreased albumno-globuline ratio is characteristic. Hypoalbunemia is joined with the increase of the percentage of α- and γ-globulines.

Acid-base balance is affected. Intensified catabolism causes the accumulation of acid products in tissues and the development of metabolic acidosis. Metabolic acidosis can be changed by respiratory alkalosis via intensified ventilation of lungs and excretion of a large number of bicarbonate ions from the organism. This assists the support of the pH level of plasma within normal oscillations, though alkaline reserve is decreased.

The disorder of the water exchange is expressed in hyperhydration or dehydration. Outer cellular and inner-cellular dehydration can be distinguished.

Clinical pattern of the intercellular dehydration consists of symptoms of the brain swelling (vomiting, headache, state of coma, and affection of breathing rhythm), symptoms of intercellular (swelling) and intervascular hyperhydration (hypervolemia, increase of the arterial pressure, left ventricle incompetence with lung swelling).

Extracellular dehydration can be clinically manifested with hypervolemia and skin dryness. Arterial blood pressure is decreased. Pulse is weak. Collapse can often have place.

The duration of oligo- and anuria stages is from two to three weeks.

The *third stage (renewal of diuresis)* can be characterized by the increase of the amount of the urine excretion. Together with the increase of diuresis, patients feel better. They become less drowsy; their concernedness is clearer, headache and muscle ache lessen, lung-swelling decreases as well. The skin becomes dry. Appetite improves. Together with the increase of diuresis, the degree of azotemia increases; and concentration property of kidneys increases.

The *fourth stage (recovery)* can last for 3 – 6 months to one-two years. The state of patients, who underwent acute renal insufficiency, improves gradually. The most stable symptoms are asthenia, anemia and decrease of the concentration of renal properties. Complete renewal of the functional state of kidneys will be in one or two years.

In specific cases, transfer of the acute renal insufficiency into the chronic one can take place. Changes in kidneys, caused by the impact of chemical matters, which create toxic metabolites, that have impact onto kidneys, can be considered as toxic nephropathy. Marked forms of nephropathy can develop in case of severe acute poisons with chemical matters (chlorinated hydrocarbon, organic compounds of mercury, chlor- and phosphoro-organic pesticides, etc) and are accompanied by various degrees of the expression of the acute renal insufficiency.

C h r o n i c p o i s o n i n g with chemical nephrotoxic matters is manifested with changes on the side of the central and peripheral nervous system and organs of blood formation. The first symptoms usually take place after the contact with the poison for three or more years. At first, after three to five years of work, functional activity of kidneys can be intensified: renal blood stream and plasmostream increase, glomerular filtration and clearance of urine increase. For the following 6 to 10 years, some normalization of functional properties of kidneys takes place.

If the work period is over 10 year, the activity of compensatory mechanisms with gradual and non-simultaneous inhibition of the listed functions, increase of the filtration fraction, decrease of the ratio of

purification of the urine, oliguria and nocturia weaken. At first, relative thickness of the urine increases a little, and then it decreases. In urine, some amount of protein, erythrocytes, hyaline cylinders, and cells of renal epithelium can be observed. The activity of cholinesterase decreases. Thus, it is possible to define three phases of chronic toxic nephropathy; the increase of the kidney activity, adaptation and the decrease of the functional property of kidneys.

At chronic intoxication with various chemical matters, the toxic nephropathy is often determining syndrome of intoxication; usually, functional disorders of kidneys is determined on the background of the expanded clinical pattern of intoxication. Only at intoxications with cadmium and β-naphthol, through the affection of kidneys and early forms of these intoxications are diagnosed based in the indicators of the functional state of kidneys.

Nephrotoxic action of heavy metals progresses with relatively mild clinical symptoms. Significant interest is caused by the affection of kidneys in the result of the lead intoxication. In cases of heavy forms of chronic intoxication with lead, changes in renal vessels, hemorrhage, necrosis of epithelium, and fibrosis changes occur. In case of chronic intoxication with lead, passing proteinuriais caused by irritating action of the lead onto the channel epithelium and reverse functional disorder in the area of secernent epithelium.

For saturnism, presence of spastic state of vessels of kidneys, alternative changes of channel epithelium cells with their intranuclear destruction are characteristic to be present. Under the impact of lead, oscillation of the concentration function of kidneys can be observed. Though nowadays, the very lead etiology of the chronic nephritis is not supported by the majority of researchers, but still in those cases, when lead intoxication is headed by the kidney disease, intoxication with lead can significantly worsen the non-specific inflammatory process in kidneys.

Occupational diseases of the urinary system include tumors of the urine bladder. It has been proven that the cancerogenic action is possessed by β-naphthylamine, benzidine B, adiacetylbenzine and some of their derivatives. These matters permeate into the organism of a human being through the skin, respiratory organs and esophagus.

The beginning of the disease can be characterized by the symptoms of chronic irritation of the mucous tunic of the urinary bladder. Often, patients do not have any complaints for a long time, and except some non-constant more frequent urination mostly during the daytime. Not much deviation can be found in the urine. Further, urination is more often accompanied by sharp pain, some complication and non-constant hematuria.

Later, chronic irritation of the mucous tunic of the urinary bladder, what takes place in the result of the discharge of aromatic amines, is manifested by the disorder of urination on the background of which hemorrhagic cysts can develop often with painful impulses to urinate, as well as marked heamturia. At this time, cystoscopy can be expressed mostly in the area of a triangle and a neck of urinary bladder. Sometimes, they are spread onto other areas of the mucous tunic. In complicated conditions, there is a threat of epithelium peeling off.

Sometimes, the appearance of complaints on dysuric phenomena corresponds with the development of papilloma or malignant tumors of the urinary bladder. A tumor can develop in any section of the urinary bladder, but mostly it locates in the area of he triangle.

Affection of kidneys, renal pelvis, urinary tracts and the hind portion of the urinary channel with a tumor process can be both in separate cases, which are about 4 to 5 % among tumor cases of the urinary bladder. Main symptoms of he tumor of the urinary bladder are profuse heamturia, which often follows microheamturia. Non-malignant papillomas are difficult to be separated from malignant new formations, thus a decisive; meaning in the diagnostics is possessed by the morphological research.

Histologically, malignant new formations of the urinary bladder are often papillary fibroepitheliomas and papillary cancer.

In the diagnostics of occupational diseases of the urinary bladder, cystoscopy is more considered. In the analysis of the urine, microhematuria can be noticed. When analyzing urine on the testing of Kakovsky – Addison, small amount of erythrocytes can be in norm as well.

Macrohematuria usually appears on the background of a profound pathological process in the mucous tunic of the urinary bladder. To find cells of a malignant tumor in the urine is usually very complicated, in spite of the developed special methods of separation of cells from mineral and organic parts of the sedimentation.

Taking into consideration the forecast of occupational diseases of the urinary bladder and urinary tracts, it is necessary to note that hemorrhagic cystitis causes the development of a tumor rather rarely. Regarding the papillomas, they are not usually subject to reverse development and can reduce. Recurrent papillomas are often capable to turn into malignant tumors. The possibility to cure the cancer of a urinary bladder, as well as tumors with other location, is determined by the stage of the disease.

Treatment. In cases of severe poisoning with the development of acute kidney insufficiency, for example, due to intoxications with mercurial salts and arsenious hydrogen, patients are subject to obligatory hospitalization in specialized establishments.

At the *first stage* of the disease, the treatment and preventive measures of the acute renal insufficiency is reduced to the prescription of

specific antidotes, eradication of circulatory disorders, exchange blood transfusion at hemolisis as well as eradication of destroyed tissues. At the *second stage,* therapeutic measures should be directed at the decrease of protein catabolism, to support water-electrolyte and acidic-alkaline state, prevention of cardiovascular decompensation and infections. If it is impossible to achieve compensation, it is necessary to use methods of extrarenal cleaning – hemodialisis with the utilization of the artificial kidney apparatus or peritoneal dialysis.

At the *third stage,* it is necessary to keep thorough control of the electrolyte composition of the serum. If necessary, its corrections should be done.

In urinological hospitals, treatment of cysts is also carried out, as well as operative interventions on papillomas or cancer of the urinary bladder.

Lately, much success has been achieved in chemical therapy of malignant new formations of the urinary bladder.

Verification of the ability to work. Workers, who were found changes of chronic cyst or papilloma types in the mucous tunic of the urinary bladder after the prophylactic examinations, are subjects to obligatory transfer to the work, which is not connected with possible impact of toxic matters. At the development of new formations, there is a question regarding operative intervention and transfer to the invalidism group. Issues on rational work position should be solved individually.

Preventive measures. It is necessary to introduce non-stop technological processes, to use hermetic machines, improve automation and remote control. It is necessary to check up that workers use means of individual protection.

An important role in prevention of these diseases is played by the conduct of preventive and periodical examinations of workers.

OCCUPATIONAL DISEASES WITH MAJOR AFFECTION OF NERVOUS SYSTEM

Occupational intoxications, which develop with mostly the affection of the central nervous and periphery nervous system are called neurointoxication or neurotxicosis.

Classical poisons, which mostly impact the nervous system include metal mercury, carbon bisulfide and tetraethyl lead. The neurotropic action is possessed by the many narcotic matters and hydrocarbon. Some chemical compounds can cause changes not only in the nervous system, but also in other organs and systems (lead, benzol, carbon oxide, fluoride, etc). Sometimes, the same matter in high concentration causes neurotropic effect, and in low concentrations, it has a different effect. For example, benzol at large concentrations has impact onto the nervous system

(narcotic influence), and at low concentrations, it causes the change in the haemopoiesis system. Under the impact of high concentrations of phthalate plasticizers, irritation of the mucous tunic of eyes and nasopharynx take place, and at low concentrations, it can cause changes on the side of peripheral nervous systems.

Various sectors of the central and periphery nervous system are involved into the pathological process. There are changes in all the elements of the nervous system – vessels, cells, nervous fibers and neuroglias. Changes from the side of the nervous system at the action of toxic neurotropic poisons are non-specific. Some toxic matters differ by the selection of the impact onto various sectors of the nervous system: manganese – on the striopalidar's system, carbon oxide – onto basal ganglia; and tetraethyl lead – onto the thalamohypothalamic zone as well.

The mechanism of chemical matters onto the nervous system is variable. They possess blocking action onto the ferment system, mediators and biologically active matters. Inhibiting tissue respiration, they can cause hypoxia of tissues with changes of the nervous system. Narcotic matters block synaptic conduct of the excitement into the reticular formation of cerebral column, in the area of hypothalamus.

Clinics. Clinical pattern of acute neurointoxication is manifested by the accumulation of psycho, neurological, somatic and vegetative symptoms. At severe intoxications, consciousness is in disorder, toxic coma or acute hypoxication psychosis develops.

At chronic intoxications, changes on the side of the nervous system can be manifested through syndromes of the vegetative and vessel dystonia (dysfunction), asthenovegetative and asthenoneurotic ones. In the later stage of toxic process, there are organic changes of the nervous system – toxic encephalopathy. Disorder of peripheral sectors of the nervous system can be manifested in the form of movable, sensitive and mixed forms of toxic polyneuropathies. There is also a vegetative-sensitive form of the latter.

Along the progress of neurointoxication there are two stages – functional disorder of the nervous system, which is manifested in earlier terms of the impact of poison and is characterized by the reversibility of changes, and the stage of limited changes in the central and peripheral nervous systems. Organic symptoms develop in case of long work period under unfavorable work conditions and are characterized by stable and long progressing even under conditions of the termination of the contact with the matters.

Intoxication with Manganese

Threat of intoxication with manganese in the form of oxides or salt can exist under the following conditions: when extracting manganese ores,

smelting of steels of high quality and some alloys; in case of arc welding with electrodes, which contain manganese in its coating and automatic and half-automatic welding under fluxing agent; in the process of the production of elements for pocket batteries. The permitted concentration (when calculated into MnO_2) is 0.3 mg/m^3.

Manganese permeates into the organism through lungs, and less through gastrointestinal tract and possibly through skin. It is deposited in the form of phosphate in lungs, bones and liver. It is discharged with faeces and urine.

Pathogenesis. Among many mechanisms of the action of manganese onto the organism of a human being, particular attention is required by the disorder in the exchange of biogenic amines and in the number of ferments and disorders in hormone formation.

Data has been received that proves inhibition of adrenoreactive systems and the increase of the activity of M- and H- cholinereactive systems, accumulation of acetylcholine in synapses of basal ganglia and in hypothalamus. The dependence of extrapyramidal and psycho disorders has been proved on the disorder in synthesis, and in particular onto depositing of dopamine.

Some meaning in the pathogenesis of poisoning with manganese is possessed by hyperfunction of endocrine glands (sex gland, thyroid gland, adrenal gland and hypophysis). Role in pathogenesis of poisoning with manganese of functional insufficiency of the liver is possible.

Main toxic action is caused by manganese onto the central nervous system (striopalidar's sector), and the smaller one – onto the peripheral, cardiovascular and endocrine system. This metal is a weak allergen and can cause eczema or bronchial asthma. However, its sensibilizing properties are usually manifested only under the action of small concentrations, and in case of bigger intensiveness of the action of this property, it is masked under its high toxicity.

Pathologic and anatomic pattern. Plethora of organs takes place, degenerative-dystrophic changes can be observed mostly in the nervous system. Necrotic areas are in basal ganglia, in cortex and in thalamus. Dystrophic process is accompanied by vacuolization, acute swelling and central chromatolysis, pycnosis and ectopy of nucleuses.

Clinics. The first clinical indicators of intoxication are often come across in several years of the contact with manganese and its compounds, but it can take place within a shorter period of time (6 to 9 months). Later clinical forms of neucrotoxicosis are possible (in several years after the termination of the contact with manganese).

There are three stages of chronic poisoning with manganese. For *stage I*, functional disorders of the nervous system are characteristic: asthenia, drowse, paresthesia and mild weakness in limbs; symptoms of

dysfunction of the vegetative nervous system: increased hyperhidrosis and salivation; and vegetative-sensitive polyneuritis. They can be expressed through neurological microsymptoms: mild hypomimia, muscle hypotonia, increased tendon reflexes and hypostesia on the polyneuritic types.

Relative change in the psychological activity is characteristic: the decrease of the activity, narrowing of the circle of interests, reduction of memory and attention to the disease, and the increase of the psycho asthenia.

Changes in psyche usually follow neurological symptoms of poisons. There can be sings of inhibition of the function of gonads, thyroid glands, disorder of functions of the liver and gastrointestinal tract.

Diagnostics of Stage I of the intoxication through the absence of clear symptoms is more difficult. Besides, the transition to Stage I into Stage II sometimes pass very fast. Besides, such sick people should be under thorough observation.

Stage II of chronic intoxication with manganese is called the stage of the initial toxic encephalopathy. At this stage, marked asthenic syndrome is formed; apathy and drowse develop. Neurological indicators of extrapyramidal insufficiency can be revealed: hypomimia, sensitive bradykinesia, pro-and retropulsion, muscle dystonia with the increase of muscular tonus of some muscles. Sings of polyneuritis, paresthesia in lungs is more expressed and walking up and down stairs is more complicated. Inhibition of the functions of gonads, and adrenal glands is characteristic. Functional disorders of liver and gastrointestinal tract can be observed.

Stage III of poisoning with manganese – manganese Parkinsonism. For this stage, it is characteristic to have severe disorders when moving: mask-like face, dysarthria, disorder of handwriting, “cock” walking (walking on tiptoes, conditioned by the contraction of flexor muscles of foot) or spastic-paretic with foot paresis, rough pro- and retropulsation (Fig. 5). All the movements are slow and difficult. Muscle tone is observed, mostly in legs; muscle hypotonia or dystonia can be often observed as well. Tendon reflexes are increased according to the pyramidal type, and polyneuretic type of hypoesthesia can be noted.

A different feature of manganese Parkinsonism is specific psycho disorders: patients are in euphoria, attitude to the disease is reduced or absent, and periodically they have violent emotions (weeping or laughter). Combination of euphoria, laughter and wobbly walking creates the impression of alcoholic intoxication or imbecility. Gradually, the circle of interests of the patient reduces, they become indifferent (symptom of the so-called affective flattening), and general intellect reduces as well. Change of handwriting is specific to it as well; it becomes illegible with the tendency to small image of letters (symptom of micrography).

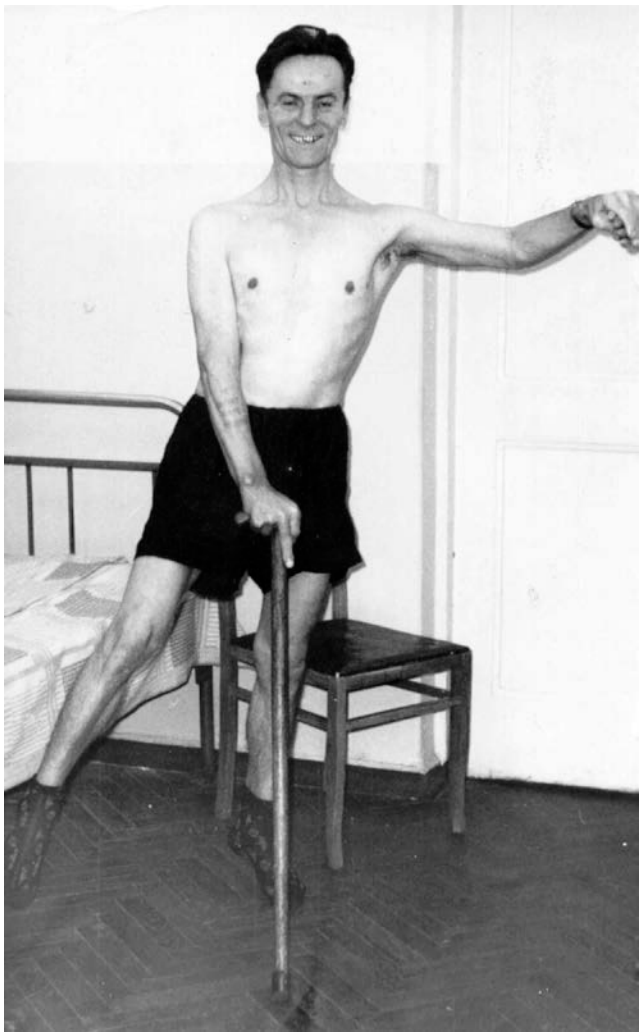


Fig. 5 Manganese Parkinsonism (“cock” walking)

Sometimes, symptoms, which are rather characteristic to intoxication, are absent. However, the general feature of various symptoms of manganese intoxication is the disorder of plastic tone of muscles with its advantage in some muscle groups.

Thus, neurological pattern of manganese intoxication is mostly characterized by the syndrome of Parkinsonism; however, it is not completely covered by it. Presence of the affection of cranial nerves, pyramidal insufficiency, and frequent affection of peripheral nervous system enable to talk about toxic encephalomyelopoly-neuritis.

Sometimes, in spite of the termination of the contact with manganese, phenomena of Parkinsonism progress, in particular during the nearest 2 to 3 years.

Clinic of Stage III of manganese intoxication is close to postencephalitic Parkinsonism. At differentiated diagnostics, it is necessary to take into consideration the data of anamnesis (contact with manganese, absence of acute beginning with fever, changes in the blood pattern, double vision, strabismus, and fits of “gaze spasms”. It is also necessary to remember that manganese Parkinsonism has the tendency of faster development, than the postencephalitic one.

Changes, noted among patients with manganese intoxication of Stage III, are not much reversing, but they are not lethal for life either. Forecast as to the possible renewal of the ability to work is unfavorable.

Chronic manganese intoxication among electric welders is characterized by specific clinical progressing. Asthenic syndrome is more expressed. Manifestation of extra pyramidal insufficiency is also possible. Changes as to muscles often manifest through dystonia, component of clinical symptoms is the development of specific polyneuretic syndrome on

the background of general asthezation. However, with the development of the pathological process, it is possible to observe merging with extrapyramidal insufficiency. Clinical manifestation of manganese intoxication of electric welders has mild progressing. Tendency to variation of the arterial blood pressure can be observed, often patients complain to have pain of pressing character in the heart. Often, palpitation and lability of pulse and dullness of heart tones can be observed; and in a number of cases - sinus arrhythmia as well as the decrease of the contraction of the heart ability.

Treatment. Already in early stages of the diseases, patients with chronic manganese intoxication should be prescribed vitamins B_1. Thiamine chloride should be administered intravenously or intramuscularly in the dosage of 1 ml of 5 % solution. It is expedient to use vitamins B_1 and B_6 together.

With more marked signs of intoxication, it is recommended to administer intravenously 0.5 % solution of Novocain in the dosage of 5 ml. Administering of Novocain should be alternated with hypodermic injections of 0.5 % solution of proserin or neostigmine, starting with 0.3 to 0.8 ml. The treatment course is 15 days.

Good therapeutic effect is provided by the preparation of benzotropine, as it causes cholinergic action, and improves synaptic transmission.

At rigid bradykinetic syndrome, it is recommended to use mitandan, which also possesses cholinergic activity. The preparation is recommended to take in the dosage of 0.1 g twice or three times a day. The treatment course duration might be from one to three months.

Due to the fact that with manganese intoxication, the content of dopamine decreases, it is better to utilize 1-dihydroxyphenylalanine. Levodopa goes through blood-brain barrier, in bazal ganglia; it is turned into dopamine and reduces hypokinesia and rigidity of muscles. It can be administered inside after meals, starting with the dosage of 0.25 g and then it is gradually increased up to 4 g for 6 to 8 months. Administration of levodopa can cause the development of dyspeptic disorders. The preparation is contraindicated at significant changes of functions of the liver, kidneys and endocrine glands.

To increase the resistance of the organism, it is recommended to administer intravenously 5 ml of 5 % solution of ascorbic acid together with 20 ml of 40 % solution of glucose. Both in the initial stage of intoxication, and at micro-organic affections of the nervous system, positive effect is rendered by radon, pine needle, and chamber galvanic bathes.

There is also information on utilization of complexing agents, and in particular EDTA (ethylenediaminetetraacetic acid) at manganese

intoxication. The preparation is administrated intravenously by drops in the form of 20 ml of 10 % solution per 300 ml of 5 % solution of glucose or isotonic solution of sodium chloride for 3 to 4 days with further break between courses for 4 to 5 days.

Verification of the ability to work. In case of suspicion of intoxication with manganese, it is recommended to have temporary transfer to another job position for the term of a months and a half with the provision of the occupational certificate and with medical supervision. All patients with chronic intoxication should be immediately removed from work where the contact with manganese and its compounds is possible.

At stage I of the intoxication, patients beyond the contact with manganese keep their ability to work and thus are subject to rational job placement. In cases, when the transfer to another job might have negative impact onto the qualification or the amount of the occupational activity, patients should receive group III of the occupational invalidism.

Patients with chronic intoxication with manganese of Stage II due to significant decrease of the ability to work or its complete loss are subject to transfer to group III or II of the occupational invalidism (depending on the degree of the functional disorders).

At Stage III of intoxication, patients are considered completely incapable to work and receive Group II or I of invalidism.

It is necessary to take into consideration that sometimes, progressing of the process can start in several years after the termination of the contact with manganese. All the patients with chronic intoxication with manganese are subjects to regular dynamic medical check-up.

Preventive measures. It is reduced to the complete sealing-in of production processes, decrease of dust creation, keeping to rules of personal hygiene (utilization of respirators, having meal outside the production premises and frequent change of a uniform), as well as the conduct of periodical medical examinations.

Intoxication with Mercury (Mercurialism)

Mercury is a rare metal; it evaporates at room temperature. It is widely used in the production of thermometers, manometers, barometers, daylight lamps, quartz lamp – source of UV-radiation, and polarographs. It is also used as a catalyst in the chemical industry and laboratory syntheses of organic matters, as a cathode at the electrochemical reception of caustic soda and chloride and at the production of paints. In mining business with the assistance of mercury, gold is separated from non-metal admixtures.

Mercury is industrial poison with acutely marked toxic properties. Under industrial conditions, it can be observed in the form of metal mercury, as well as organic and non-organic and organic compounds. According to the degree of the impact onto the organism, metal mercury

and its organic compounds (ethylmercurichloride and ethylmercuriphosphate) are referred to industrial poisons of Class I of the non-safety.

Metal mercury is in the air in the form of vapors and aerosols.

The sphere of production and utilization of metal mercury, its non-organic and organic compounds, as well as devices with mercury filling is very wide: mercury production from ore raw materials and from ores of precious metal, production of chlorine and caustic with mercury method, amalgamating, production and utilization of measuring devices, production and utilization of electro-technical devices, production of electric lamps and lamps for radios, production of corrosive sublimate or mercuric chloride, calomel, and utilization of mercurous pesticides, production and utilization of mercurous detonators, utilization of mercury to produce acetic acid, trimeperidine hydrochloride, streptomycin, etc.

In the process of producing and utilization of mercury and its compound, it is possible for their permeation to the organism through respiratory organs (in the form of vapors and aerosols), and partially through gastrointestinal tract and skin.

Danger of mercury intoxication is increased in the case of careless treatment of the metal. It is known that the physical state of mercury has significant meaning when having contact with it. Thus, after spilling mercury with its splashing into small drops the surface of the contact of the liquid metal with air increases significantly, what means intensive evaporation with sharp growing of concentration of mercurous vapor in the air of production premises. Mercury evaporates easily at room temperature and at the temperature less than 0 ºC.

Small mercury drops can easily penetrate into slits of the floor, tables, and cracks in stands and thus, for a long time it is a source for contamination of the premises.

The boundary permitted concentration of mercurous vapor in the air of production premises is 0.01 mg/m^3.

Nowadays, intoxication with mercury vapors differs by the specific of the clinical progressing and vagueness of symptoms. First, it is conditioned by the fact, that at the modern production, concentration of mercury as a rule is low. At the same time, the ability of its accumulation in the organism at long impact can be a reason of the development of occupational poisons of the type of micromercurialism.

Pathogenesis. Getting into the organism mostly through respiratory tracts, mercury initiates complex integrated compounds with protein and in the form of albuminates circulates in the blood. However, rather fast it transforms into mercury dichromate, and then into mercury chromium albuminate. Furthermore, a portion of mercury is taken out from the organism by kidneys, intestine, salivary, mammary gland, as well as with

sweat and bile. Significant number of the mercury, combined with protein, deposits in the liver, kidneys, spleen, pancreas, and brain tissue. The most stable deposit of mercury is created in the central nervous system. Depositing of mercury is not an absolute physiological reaction. During the period of weakening of the general functional state of the organism under the impact of a number of unfavorable factors (infection, trauma and alcohol), it can get to blood from the deposit and cause the development of pathological processes.

Particularly complex are pathogenetic mechanisms of the development of intoxication with mercury, in the basis of which there are disorders of fermentative processes and protein metabolism.

In the process of development of mercury intoxication, there is the change of the activity of fermentative systems, which contain SH-groups, which are connected with the disorder of protein and hydrocarbon exchange, “reactivity” of vitamins and oxidation-reduction reactions. Under the impact of mercury, there can be observed the change of the activity of glutaminoaspartic acid, transaminase and aldolase in tissues of brain, kidneys, and heart, which causes to some decrease of the synthesis and the increase of the dissociation of amino acids. Increase of the concentration of histamine in blood has been found, what is conditioned by the increase of its synthesis from histidine and inhibition of mechanisms of intoxication.

Mercury, which circulates in blood, has the property to act onto chemoreceptors of vessels and interoreceptors of internal organs. In the result of toxic action of mercury, there are complexneurodynamic and neuroreflector disorders, which are manifested through the increase of anxiety of vegetative sectors of the nervous system, corticosubcortical changes, as well as the decrease of threshold of the anxiety of visual and smelling analyzers.

Permeating into the spinocerebellar liquids, mercury directly affects thalamohypothalamic area and the cortex of cerebrum. At intoxication with mercury, its content in spinocerebellar liquids and nervous tissues achieves significant sizes.

Patophysiological essence of asthenovegetative syndrome, which characterizes the clinics of the initial stage of chronic mercurous intoxication, is determined by the increased anxiety and fast fatigability of cells of the cerebral cortex.

Sleep disorders, changes in correlation of processes of anxiety and inhibition at mercurialism, are also connected with direct impact of mercury onto the grey matter of aqueduct of cerebrum and walls of ventricle III, which participate in producing impulses of anxiety and inhibition in cerebral cortex, regulation of sleep and absence of sleep.

In the genesis of mercury vibration, significant role is played by the nuclear affection of cerebellum, striatic body, and muscle innervation disorder, and first of all, it reduces the tone of extensors.

Pathologic and anatomic pattern. At the action of mercury onto the organism, destructive changes in cells of a healthy tuber, cerebellum, red nucleus, frontal, occipital and crown areas of cerebral cortex (at encephalopathy) can be observed. The most severe changes can be observed in the area of ammonium horns, in motor center and in vascular system. Significant nuclear degenerative changes can be observed in peripheral nerves, in particular in myelinic membranes.

Clinics. There are acute and chronic poisonings.

A c u t e forms of poisoning with mercury under production conditions are not frequent. They can appear not only in case of permeation of a big amount of mercury into the organ is, for example, during accidents. Clinical pattern of acute intoxications develops fast and progresses rather actively. The following signs appear: sharp weakness, illness, headache, nausea, vomiting, feeling of metal taste in the mouth, and excessive salivation. Swelling and bleeding of gums and thrush can be observed. With time, pain in stomach appears, and acute asthenization takes place. In case of timely treatment, the forecast is favorable and patients recover.

C h r o n i c intoxication with mercury under conditions of production can be met more frequently. First symptoms of poisoning can appear in several days as a rule after the beginning of the contact with mercury. For a long time, the disease develops without obvious symptoms.

First manifestations of the intoxication are not clear; they do not attract the attention of the patient. Sometimes, in the result of acute infection of the organism and under the impact of other unfavorable factors, the latent progressing of intoxication suddenly becomes sharp.

The progressing of chronic mercurialism has three stages.

Stage I has the name of “mercuric neurasthenia. Patients complain to have headache, fast fatigability, irritation, disturbed superficial sleep, with bright dreams, which are remembered well, and drowsiness. Palpitation increased sweating, inclination to constipations, metal taste in the mouth, and excessive salivation. Gradually, these symptoms are increased, feeling of internal shivering, pain in limbs of hands and feet, and the feeling of numbness of limbs. It is very characteristic for them to have hyperesthesia no noise, bright light, and lability of mood. Memory of recent events, faces, and dates worsens; learning of new materials is more difficult. Asthenic and neurological disorders can be observed, like tremor of fingers of outstretched hands with characteristic symmetry, increase of tendon reflexes, stable red demographism, and hyperhydrosis.

Lability of pulse with the inclination to tachycardia can be observed. A number of trophic disorders take place (nail fragility, hair shedding and gingivitis).

Often there are disorders of endocrine glands: dysmenorrhea, early climax, and dysfunction of thyroid gland.

In case of timely release of patients from work, connected with the contact with mercury, this stage is considered a completely reverse.

Stage II of the chronic mercury intoxication has the name of “mercuric erythrism”. It can be characterized by asthenia, loss of weight, headache, sleep disorder, inclination to depression, and acute decrease of the ability to work. The so-called “mercuric erythrism develops – that is the state of increased anxiety, irritability, fear, diffidence, shyness with fast reddening of face when worrying, and inability to continue usual work in the presence of other people.

From the side of neurological state, trembling of eyelids, tongue, and fingers of outstretched hands develop. Hand trembling is stable, what makes completion of work, which requires little exact movements, significantly difficult. The feeling of particular distrustfulness and suspicion; the patient concentrates much on his/her sick feeling.

Often there can be seen bleeding of gums, marked gingivitis, stomatitis, and caries. It is characteristic to have copper color of gullet and soft palate. Signs of chronic gastritis, gastroenterocolitis with profuse diarrhea and intensive pain in stomach can be observed.

Due to timely treatment, the majority of signs of this state disappear, though rudimentary phenomena can stay.

Stage III of chronic mercuric intoxication is called a “mercury encephalopathy”.

Patients complain to have stable headache and permanent insomnia. It is very characteristic for patients in this state to have depression; however, sometimes, psychological disorders can be manifested through uninhibited anxiety and up to hysteria. There is also a syndrome of impulsive obsession, fear, visual and hearing hallucinations. With time, patients suffer from syncope, and epileptiform fits with losing consciousness and spasms.

Also, psycho disorder can be manifested through the disorder of the memory, schizoid-like conditions, where hallucinatory phenomena, fears, deviation into the affective –emotional sphere, and sometimes’ emotional dullness” can be observed. Also, fast development of psychoses with the forecast of loony stage is possible.

In the neurological state, there are affections of VII and XII pairs of craniocerebral nerves according to the central type, symptoms of the affection of basal ganglia and moderate pyramidal disorders. There is asymmetry of nasolabial folds, horizontal pathological nystagmus,

anisocoria and hypomia. Tendon reflexes are active, their zone is expanded, and clonus and pathological reflexes are possible.

Intention tremor of fingers, which is characteristically marked, is accompanied by choreolike twitching. This state has the tendency to generalization with spreading to the head, corps and legs, and it is often starts looking like generalized hyperkinesias. Tremor of hands conditions a corresponding change of the handwriting. In rare cases, the polyneuretic syndrome with atrophy of small muscles of hands develops; and tendon reflexes decreases. Thus, clinical manifestations of mercury encephalopathy are characterized by significant psycho disorders and clear nucleuses or spread neurological indications. Neurological symptoms are accompanied by the loss of appetite, significant lost of weight, and acute general weakness.

Treatment. Treatment of mercuric intoxications should be started as soon as possible and should be carried out in a complex. Main measures in therapy of poisons are measures to neutralization of mercury, which permeated into the organism and their discharge. On the first stages, sulphuric compounds are used, in particular intravenous administration of 30 % solution of sodium thiosulfate at 20 ml for 15 to 20 days.

With time, more effective means of antidote therapy are more developed – dithioles, which in its structure had two free and closely placed SH-groups, what provided the creation of stable cyclic complex with mercuric ions. Among such dithioles, the following is widely used: BAL, unithiol, succimer, and D-penicillin.

Good treatment effect is caused by hydrogen sulphide bathes, which are recommended every other day for 1-0 to 12 days. Activator of ferment systems – lipamyd is recommended 0.05 g three times a day for 20 days.

Among symptomatic means, there can be intravenous administration of 20 ml of 40 % solution of glucose with 5 % solution of ascorbic acid (2 ml), 0.5 % solution of vitamin B_1 (1 ml) and 0.5 % solution of vitamin B_6 (1 ml) are recommended.

In case of presence of neurasthenic syndrome, it is recommended to administer small dosages of sodium bromide (0.5 – 1 % solution) with sodium caffeine-benzol (0.1 – 0.05 %) one spoon – three times a day, antihistamine preparations (Dimedrol, suprastin and phencarol), tincture of leonury, valeriany together with small doses of caffeine. Positive effect is provided by coniferous and sea bathes, the course of 12 to 14 bathes is recommended to take every other day.

If the progressing of the disease is long, patients are sent to resort centers or preventoriums of the enterprise after staying in hospital.

Verification of the ability to work. At chronic intoxication with mercury of mild stage (functional disorders of the nervous system), patients are transferred to another temporary work for the term up to two

months with the provision of the sick leave. In case of the repeated intoxication, insufficient effectiveness of the treatment, and also intoxication of mean and severe degree at the appearing of the intentional tremor, transfer to the work beyond the contact with mercury is obligatory; at intoxication at the border with toxic encephalopathy, patients should receive invalidism group.

Preventive measures. Among preventive measures, first it is necessary to remember about elimination of possible sources of mercuric intoxication (correct keeping of mercury and its compounds, removal of mercury or its replacement with less toxic compounds), localization of sources of contamination of production areas and other zones with mercurous vapors, following corresponding safety norms (ventilation and sealing-in of the equipment) and rules of personal hygiene; regular demercurialization of premises, where sources of mercurous contamination are.

If mercury was spilt onto the floor, it is processed with 20 % solution of iron chloride or covered with sulfur powder.

Among preventive measures, it is also necessary to mention preliminary and periodical medical examinations of workers.

Intoxication with Carbon Bisulfide

Carbon bisulfide is the compound of carbon and sulfur. It is a colorless liquid. It has specific smell, like chloroform. Carbon bisulfide evaporates easily at room temperature. It can be used in the production of viscose fibers, cellophane, in chemical industry as the dissolvent for phosphorus, fats and rubber; when producing optical glass and water-resistant glues; in agriculture – as an insecticide. The border permitted concentration is 1 mg/m^3.

Carbon bisulfide permeates into the organism mostly through respiratory organs, and can permeate through undamaged skin. Most of carbon bisulfide, which permeates into the organism, is subject to chemical transformations and is discharged from the organism with urine, possible with faeces in the form of non-organic sulphates and other sulfur-bearing compounds. It is partially discharged in an unchanged look with exhaled air. Highest concentration of carbon bisulfide is in the brain, the tissue of peripheral nerves, liver and kidneys. It is known that carbon bisulfide goes through placental barrier and has embryotoxic action, and also permeates into the milk of nursing mother.

Pathogenesis. Toxic impact of carbon bisulfide can be explained by its interaction in the organism with various nucleophilic groups (H_2-, SH- and OH-groups) of proteins and other compounds, which cause the disorder of metabolism and blocking ferment systems, in particular those, which contain copper – monoaminooxidase and ceruloplasmin. Inhibition

of the latter ferment causes the binding of pyridoxamine and deficit of vitamins B_6. Changes of regulatory neurohumoral impacts cause to the disorder of fatty and other types of exchange. At this, receptor and synaptic formations of the central and peripheral nervous system with weakening of mechanisms of regulation of homeostasis suffer; neurdynamic and nervous-refectory disorders, what conditions the development of the pathological process.

Pathologic and anatomic pattern. At the action of carbon bisulfide, diffusive affection of the nervous system takes place, which is accompanied by the structural changes in cells of the cortex of cerebral hemispheres, basal ganglia, hypothalamic areas and column of the cerebrum, as well as changes in the peripheral nervous system. Vascular disorders and presence of hemorrhages are characteristic. Fat degeneration of liver and heart can be observed.

Clinics. Intoxication with carbon bisulfide can be acute and chronic.

A c u t e p o i s o n i n g. Under production conditions, acute poisonings take place rarely. Clinics of acute intoxications can be characterized by active development of the clinical symptoms. According to the degree of the expression, mild and severe forms can be defined.

At *mild* form, there is a feeling of alcoholic intoxication, headache, dizziness, sometimes nausea, and vomiting. Patients are in the state of euphoria. Often, unsteady walking and specific tactile hallucinations (feeling of a touch, “alien hand”) can be observed. In some cases, the throat is scratchy. Patients with mild intoxication recover fast.

If mild intoxications repeat, it is possible to observe the state of specific alcoholic intoxication and dizziness. With time, patients have double vision, they suffer from insomnia, have suppressed mood, headache, sensitivity disorder, smelling disorder, pain in limbs, and sexual disorders. Besides, these all can be accompanied by dyspeptic phenomena as well. There are changes in the psyche, which develops gradually (increased irritability, lability of mood, as well as memory and interest worsening) and fit the pattern of toxic encephalopathy.

According to the clinical pattern, severe poisoning with carbon bisulfide reminds narcosis symptoms. If immediately after permeating into the organism of a big amount of poisoning, the patient does not leave the dangerous area, deep narcosis takes place, all reflexes disappear, including corneal and iris contraction reflexes; heart activity termination can take place as well. Most often, the unconscious state can be alternated with acute wakening; patient jumps to escape, cries and lose consciousness again, the latter is accompanied by spasms. The acute heavy intoxication often leaves traces in the form of organic affection of the central nervous system and the disorder of the psyche.

C h r o n i c p o i s o n i n g. They are the result of prolong action of comparatively small concentrations of carbon bisulfide; it is possible to distinguish functional and organic stages.

Functional stage (carbon bisulfide neurosis) can be characterized by the patient having asthenic syndrome (period of vegetative-vascular dysfunction). The most characteristic complaints include headache mostly in the frontal area, pain in hands and legs, feeling of numbness and spasms in them, general weakness and fast fatigability. Often, there is pain in heart, dyspnea, palpitation, and increased sweating. It is characteristic to have insomnia or increased drowsiness, bright dreams of scary or production content.

At the objective research, there is labile mood: agitated state with talkativeness and euphoria alternates with the state of drowsiness and depression. Such marked vegetative disorders are characteristic like widening of irises, stable red dermographism, positive symptom of levator cushion, pain in muscles and along the way of nervous columns, pulse lability after the tendency to hypertension and hypotension, regional vascular disorders (on the type of Reino syndrome), increase of pressure in temporal arteries and in the central artery of the retina, changes of venous tonus, narrowing of arteries in the eye-ground with the expansion of veins, spasms or spastic-atonic state of capillaries. Thermal asymmetry on the head and face skin can be observed. Vestibular-vegetative reactions are intensified.

With time, astheno-vegetative syndrome develops. Patients complain to have flabbiness, sharp general weakens, feeling of heaviness in the head, emotional unbalanced state with the tendency to depression and whining. They are often disturbed with sleep disorder on the type of insomnia or very superficial anxious one with many bright dreams.

Typical phenomena are phenomena of the decrease of memory and attention. Patients complain to get oblivious. They lose interest to work or entertain. On the background of apathy and depression, fits of sharp impetuosity and fidgeting take place. Impotence and dysmenorrhea develop.

From the side of the peripheral nervous system, there are numbness of fingers and toes, sensitivity to cold in limbs and their aching. Symptoms of vegetative-vascular disorders are intensified. Thus, there is some consecution of clinical symptoms – asthenic or astheno-neurotic reactions gradually develop into the stable astheno-vegetative syndrome with further development of nuclear, mostly hypothalamic affection. Though, it is necessary to note that earlier at the absence of complaints, among patients, which have long worked in the contact of carbon bisulfide, at objective research, it is possible to determine the decrease of the excitement of vestibular-motor reactions, and the change of pain sensitivity. Reflexes

from the mucous tunic of the gullet membrane and at the irritation of cornea are decreased. They have moderately expressed symptoms of the increase of functions of the thyroid gland, and tendency to the reduction of secretion of the digestive juices. With the development of the disease, astheno-neurotic and vegetative reactions are manifested more markedly.

With further development of the pathological process, there is diffusive organic affection of the central nervous system, which develops according to the type of encephalopathy or encephalo-neuritis (*organic* stage).

C a r b o n b i s u l f i d e e n c e p h a l o p a t h y. Severe affection of the nervous system develops at the prolong period of work at enterprises, where sanitary-hygienic and sanitary-technical requirements are not kept.

The clinical pattern is polymorphic. Depending on the localization of changes in the central nervous system, the clinical manifestations of encephalopathy can vary. The following can be observed: mild hypomimia, blurred asymmetry and the irregularity of the facial innervations, increase of tendon reflexes, and positive symptoms of the oral automatism. Marked vegetative disorders develop on the type of crises, often sympathoadrenal with the increase of secretion of catecholamines together with urine.

Changes of psyche have well-defined character. Patients are apathetic, gloomy, inhibited and often depressive. Loss of memory can be of high level, and visual and hearing hallucinations are possible. It is very characteristic to have tactile hallucinations with the feeling of touching of a shoulder by an “alien hand”.

Sometimes, on the background of chronic intoxication a sudden pattern of acute psychosis with phenomena of deliria can develop.

C a r b o n b i s u l f i d e p o l y n e u r i t i s. Polyneuritis can be joined both with vegeto-asthenic syndrome and with toxic encephalopathy. It is possible development of sensitive and vegetative forms of polyneuritis.

Already on the early stage of carbon bisulfide polyneuritis asthenia at the close examination of the nervous system, anisoreflexia and hypestesia in distant sectors in limbs can be observed. Later, complaints to have numbness of toes and fingers, sensitivity to cold in them, fast fatigability of legs when walking and weakness in hands can be observed. At objective examination, there are disorders of sensitivity first in the form of hyperesthesia and then hypoesthesia. In some cases, disorders of the superficial feelings encompass not only in distant, and in proximal sectors of limbs, and sometimes – segments of the corps and face.

Vegetative disorders are manifested through the decrease of skin temperature in hands and feet, change of the coloration and disorder of trophism of the skin.

Often, carbon bisulfide polyneuritis can be joined with symptoms of the affection of the head and sometimes of the spinal cord. In case of the marked degree of intoxication with the presence of extrapyramidal syndrome, there can be frequent episodes of trembling, which got the name “tremor shots”.

From the side of internal organs, there is lability of the arterial pressure, and decrease of the mean pulse blood filling. Pain in heart is combined with unstable rhythm, and sometimes signs of myocardosis on the electric cardiogram, and decrease of the contraction function of the heart muscle. Indications of the external respiration worsen, what is conditioned by the irritating action of carbon bisulfide onto the mucous tunic of upper respiratory tracts.

People with chronic carbon bisulfide intoxication can be diagnosed to have chronic gastritis with the decreased secretory and excretory functions of the stomach. Affection of liver, dysfunction of the thyroid gland and cortex of the adrenal gland can be observed. The following is characteristic for the intoxication with carbon bisulfide: disorder of the sexual sphere in the form of impotence in men and frigidity – in women. Cases of dysmenorrhea are common, and in the number of cases – early climax. Many years’ contact with carbon bisulfide leads to the development of early arteriosclerosis with most affection of vessels of the brain and kidneys.

Diagnosis of intoxication with carbon bisulfide is made according to the data regarding the contact, with characteristic symptoms of determination of carbon bisulfide and its final exchange programs – non-organic sulphate in blood and urine. Urine has specific smell of black radish. When testing with Feling’s solution, it becomes dark brown due to the presence of hematin, which is created in the result of decomposition of hemoglobin. Indirect indications of intoxication with carbon bisulfide are the increase of the content of copper and 4-pyridoxine acid in urine as the result of the impact of carbon bisulfide onto ceruloplasmin and vitamin B_6.

Progressing of carbon bisulfide neurosis under conditions of timely termination of the contact with poisoning is favorable. Carbon bisulfide polyneuritis is also characterized by the favorable forecast, though its progressing is lengthy. Unfavorable forecast is expected at the toxic encephalopathy; however, it is necessary to remember that many of their manifestations under the impact of a corresponding therapy are subject to the reverse development.

Treatment. In case of development of acute intoxication with carbon bisulfide, a patient should be taken outside the dangerous zone. He/she is recommended to stay in rest, and take strong tea or coffee. During the first hours of poisoning, he is given oxygen and carbogenes (carbogenes for 15 minutes, and then oxygen – for 45 minutes). 1 ml of 1 % solution of

lobeline and 1 ml of 2 % solution of cytitone are administered. Sudden decrease of heart activity; it is necessary to administer 1 ml of 25 % solution of Cordiamin and 1 ml of 10 % solution of caffeine. Then, therapy should be conducted, which includes vitamins, tranquillizers (diazepam) and antidepressants (imipramine). As an antidote therapy, it is recommended to use preparations which contain sulfhydric groups, glutamine acid, vitamin B_6 and copper acetate.

Patients with chronic carbon bisulfide intoxication are administered 20 ml of 40 % solution of glucose with 1 ml of 5 % solution of vitamin of B_1 intravenously, glutamine acid inside in the dosage of 0.5 g three times a day, 5 % solution of vitamin B_6 intramuscularly in the amount of 1 to 2 ml each day. Treatment course is from 3 to 4 weeks. It is necessary to use 0.05 % solution of copper sulfate in the dosage of 15 drops three times a day. At disorders of sensitivity, it is recommended to have hypodermic injections of the solution of proserin 1:1000 (starting with 0.2 ml and gradually increasing up to 0.8 – 1.0 ml), totally – 12 to 15 injections. Patients with toxic encephalopathy are recommended to have tranquillizers, antihistamine preparations (Dimedrol, suprastine, and promethazine), antidepressants and oxygen therapy.

At vegetative-vascular syndrome of effective combined treatment with small dosages of central anticholinergic drugs, e.g. benactyzine in the doses of 0.001 g a night together with vitamins “A plus E” and cocarboxylase.

Best results of treating patients with dienecephal syndrome enable the administering of pyroxene. It is taken in the dosage of 0.03 g three times a day for one month. It is also effective to take in vitamin B_6 and copper acetate.

From physiotherapy methods at initial stages of chronic intoxication with carbon bisulfide, it is recommended to prescribe galvanization according to Scherbak’s method. It is recommended to have carbonic bathes with gradual decrease of water temperature.

Verification of the ability to work. At the initial stages of chronic intoxication with carbon bisulfide, patients are still able to work. Transfer to the work, which exclude contact with carbon bisulfide, are people with stable functional disorders of the nervous system, as well as with polyneuropathy at the absence of the effect from treatment. Indications to determine group III of invalidism or partial loss of the ability to work are difficulties with job placement, as in case of the continuation of the contact of patients with carbon dioxide, disorder of homeostasis can become progressing.

Preventive measures. The following is envisaged: obligatory mechanization of work, connected with contact with carbon bisulfide, sealing-in of equipment, utilization of individual protection – industrial gas

masks under conditions of increased content of vapors of carbon bisulfide in the air of the work zone. It is necessary to conduct preliminary and periodical medical examinations, and treatment of accompanying diseases.

Intoxication with Tetraethyl Lead

Tetraethyl lead is an organic compound of lead; it is a colorless liquid with sweetish sugary smell. It can evaporate at room temperature. It dissolves well in organic solvents, fats and lipids. It is well absorbed by clothes, concrete, and plaster. It is used to produce ethyl liquid, which is used as an antiknock agent in combustion engines. The boundary permitted concentration of tetraethyl lead is 0.005 mg/m^3.

Ethyl liquid, which contains 50 % of tetraethyl lead; it is added to benzene and receives ethyl benzene for vehicles (1.5 ml of ethyl liquid per one liter of petrol) and airplanes (4 to 8 ml per one liter). For indication, the ethyl liquid is colored in red, thus ethyl petrol has pink coloration. Contact with tetraethyl lead is possible in the production of this product, during the work at mixing stations, and also in the process to receive its mixtures. The threat of contact also exists when transporting and saving it at warehouses, when servicing, testing, utilization and repair combustion engines, which use ethyl petrol and also for petroleum storage depot and vehicle garages.

Due to its volatility, under production conditions, tetraethyl lead can permeate into the organism through respiratory tracts, and absorb through undamaged skin. In the organism, tetraethyl lead submits to hydrolysis; the molecule of tetraethyl lead can stay unchanged in the organism up to three months. Due to its tropistic action to lipoproteins, tetraethyl lead is accumulated in the brain, and it goes through hematoencephalitic barrier. Non-organic lead, which frees in case of the decomposition of tetraethyl lead, it is deposited mostly in the central nervous system, and is partially discharged with faeces and urine; it can be found in all biological substrates. Excretion is slow, for months and even years after the termination of the contact.

Pathogenesis. Tetraethyl lead is a strong neutropic and vascular poisoning. It affects all the sectors of the brain, in particular hypothalamic-pituitary system. It causes degenerative changes in liver and heart, and affects adrenal glands.

Besides, toxic action of the very tetraethyl leads, significant meaning in pathogenesis of intoxication is possessed by products of its partial decomposition (tetraethyl lead-chloride), which have strong toxic properties. The role of lead, which is created at the final decomposition of tetraethyl lead, is insignificant. Non-organic lead (the amount of which is small as well) is partially extracted from the organism, and partially it is deposited in tissues. At this, in comparison with lead, which permeated

into the organism in the form of metal, it is localized mostly in the central nervous system and in fewer amounts in bones and parenchymatous organs. It is supposed, that tropistic action of this compound to lipids is mostly determined by the affection of the central nervous system. Besides that, some meaning is in the fact that vessels of hypothalamic sector is in the fact that vessels of hypothalamic sector have increased permeation for grand molecular compounds.

Particular sensitivity of the cortex and hypothalamic sector is connected with the presence of neuroendocrine cellular formations in these areas. Besides, there is an assumption that the poisoning, causing the irritation of vasoreceptors, and conditions a sudden change of functional state of hypothalamus in a reflex way.

Vascular component certainly plays an important role in the development of intoxication. A property of tetraethyl lead to have an impact onto the tone of vessels is well known. Changes of blood flow cause anoxia of the cerebrum, and in its turn, it deepens functional and organic changes in the central nervous system.

Besides, some role in the genesis of changes at intoxication with tetraethyl lead is performed by the impact of this compound onto the progressing of tissue oxidizing processes, and in particular in the nervous tissue. It has been stated, that tetraethyl lead blocks coenzyme part of dehydrogenase of pyruvic acid, which disturbs processes its oxidation and conditions the increase of the content of pyruvic acid and acetylcholine in tissues. Inhibition of cholinesterase takes place, the degree of which is proportionate to the severity of the toxic impact.

Clinics. Depending on the intensiveness and the duration of the impact of tetraethyl lead onto the organism, acute or chronic poisoning can appear.

A c u t e p o i s o n i n g. Acute intoxication is possible at significant contamination of the external environment, crashes with tetraethyle lead or with massive spilling on themselves with tetraethyle lead or ethyl liquid, as well as after the accidental swallowing of these matters.

In the initial stage (I) of acute poisoning, patients have sudden acute headache, and sometimes, vomiting, metal taste in the mouth and general weakness. Often the state of euphoria and the decrease of critics can be observed. Sleep is affected (it becomes interrupted and superficial, and is accompanied by numerous nightmares); in the sleep, patients cry, toss around the bed, jump and want to run. During the daytime, patients are gripped by the feeling of unexplained worry and fear. They are suppressed, lost and their memory is worsened.

Disorders in vegetative nervous system can be found: arterial hypotonia, bradycardia, and hypothermia. The degree of severity of intoxication is determined by the expression of these symptoms. Often,

patients are worried by the feeling of formication in some parts of the body – specific paresthesia.

Depending on the character of main symptoms of stage I of acute poisoning, some symptoms can be defined: predeliria, organic and asthenic ones.

In prediliria symptom complex, sleep disorder dominates. Dreams are accompanied by the fear of death. It seems to patients that they are followed, tortured, and that they face death danger.

Later, hypnagogic hallucinations take place when a patient falls asleep, at first, they look like episodic pictures (a patient sees faces and images of animals), and then they get the character of nightmares. Hallucinations in the period of falling asleep are forerunners of the development of psychomotor agitation.

Organic syndrome on the type of encephalopathy in comparison from predeliria is characterized by the more limited psychopathology. On the front plan, there are disorders of fronto-cerebellar system: ataxia, disorder of movement, nystagmus, and dysarthria, trembling of limbs, and sometimes, intention trembling. Acute headaches and insomnia can be observed as well.

The clinical pattern of the initial period of the acute intoxication with tetraethyl lead in its mildest forms is limited by asthenic syndromes (increased fatigability, disorder of attention, headache, and emotional unbalanced state).

At mild forms of acute intoxication, the process is gradually compensated and is over with complete recovery.

Stage II – pre-culminating. In case of acute intoxications with tetraethyl lead in the majority of severe forms, the process can progress, growing into the pre-culminating stage. Delirium develops. Delirium state is the most characteristic manifestation of severe acute poisoning with tetraethyl lead. Feeling of anxiety grows. Patients are depressed, and do not trust those, who surround them. There are visual, hearing and tactile hallucinations, which have threatening character. Everything, what surrounds a patient, is treated by him as an enemy, and targeted against him or her. In the most cases, there is well marked psychomotor agitation, which develops on the background of staggering state of the awareness. Patients become aggressive. They intend to escape from hospital, and jump out of windows. During this period, certain cenestopathy and disorders of the scheme of the body can be observed, what causes the development of hallucination due to physical impact.

Delirium syndrome can grow into the culmination stage.

Stage III – culminating, it develops rather actively. The most characteristic symptoms are well-marked psychomotor excitement on the background of impaired consciousness. It is very difficult to keep patients

in bed; they tear linen apart, and are very aggressive. On the peak of agitation, vegetative and trophic disorders can be found, connected with the affection of higher sectors of the central nervous system. Disorders of thermal regulation are accompanied b significant increase of the body temperature; leukocitosis and lymphocytosis take place. Breathing becomes more frequent and superficial; blood pressure varies from low indicators to the high ones; as well as profuse sweating can be observed. Sometimes there is a collapse, which can lead to the death of a patient.

Those, who underwent toxic psychosis, in future can have defective state of the psyche for a long period of time (emotional unbalanced state, inclination to pathological affects and intellectual degradation).

C h r o n i c p o i s o n i n g. Chronic intoxication with tetraethyl lead can be observed among people, who has been contact with small concentrations of these matters for a long period of time.

In the progress of chronic poisoning, there are three consecutive stages. In general, intoxication with tetraethyl lead is referred mostly to milder ones, as well as those, which have more favorable progressing.

Initial stage (I) is characterized by asthenic complex of symptoms. Patients complain to have increased fatigability, general weakness, loss of appetite, decrease of memory and attention, disturbed sleep, and emotional instability.

On this background, there are characteristic symptoms for this stage: bradycardia, vessel hypotonia, hypothermia, increased salivation, and sweating. Sleep disorders, can be observed; nightmares take place; feeling of fear, suppressed mood, depression, and sudden emotional outbreaks can be noted. In this stage, chronic poisoning has reverse character.

Stage II of chronic intoxication with tetraethyl lead can be met rarely nowadays. The clinical pattern of the disease gets the character of encephalopathy with more stable changes in the nervous system. Trembling of hands, wobbling when walking, positive symptom of Romberg, dysarthria and nystagmus can be observed.

The progress of the disease in this stage is long; patients need prolong treatment, and often, stable consequences can take place: the decrease of the intellect and the disorder of the sleep.

Stage III includes the syndrome of toxic psychosis. More often, it is the reason of the latter is the additional exogenous factor, e.g. administering of alcohol.

Among those, who underwent marked forms of chronic intoxication with tetraethyl lead, residual effects can be observed, like asthenization, sleep disorder, emotional unbalanced state, and the decrease of the ability to work. Often, these patients have fast progressing atherosclerotic process, affecting vessels of the brain and heart. On this background, hypertonic disease often develops, the progressing of which is rather severe.

Treatment. At acute intoxication, it is recommended to have the following: complete rest, hypnotic from the group of barbiturates. At sudden excitement – intramuscular injections of 10 ml of 10 % solution of hexenal are recommended. Then, administering of 2 % solution of barbital in the microenema (50 ml) is recommended. Intramuscular administering of barbamyl (5 – 10 ml of 5 % solution), intravenous or intramuscular administering of 25 % solution of magnesium sulfate (5 to 10 ml). Intravenous administering of 40 % solution of glucose (20 ml) with ascorbic acid (300 to 500 mg) and vitamin B_1 (40 to 50 g) are recommended. Before going to bed, it is recommended to take warm bath and then take in hypnotic preparations. Oxygen, and if necessary caffeine or Cordiamin can be taken in.

When tetraethyl lead gets to the skin, affected areas should be immediately washed with the help of kerosene or petrol, and then with warm water with soap. It is also necessary to change clothes and underwear.

In case of chronic poisoning and if vegeto-asthenic syndrome takes place: intravenous injections of 40 % solution of glucose (20 ml) with ascorbic acid (300 mg), intramuscular injections of 10 ml of 10 % solution of calcium gluconate (10 to 12 injections), and biogenic stimulators are recommended. At vascular hypotonia: vitamin B_1 (40 to 50 mg) intravenously is recommended. At hypertensia: intramuscular injections of magnesium sulfate in the dosage of 5 ml of 25 % solution (15 injections).

Verification of the ability to work. At the initial stage of intoxication – temporary inability to work (occupational sick leave) is issued. Return to work can be only under condition of positive results of treatment and improvement of work conditions.

At moderately marked stage – return to work where contact with ethyl petrol is possible is prohibited. Stable restriction of the ability to work (occupational invalidism) and rational job are recommended.

Marked stage – the ability to work is strictly restricted or lost (invalidism of group II and III).

Preventive measures. Strict keeping to the stated sanitary and hygienic rules: prohibition of filling machines manually with buckets, sucking in benzene through a hose with the help of mouth, washing hands and clothes in ethyl petrol, keeping to rules of personal hygiene and regulation of feeding during the work, as well as keeping clothes and washing uniform only at the enterprise.

Meals should be rich in lecithin. It is necessary to conduct preliminary and periodical medical examinations of workers.

INTOXICATION, WHICH APPEAR UNDER CONDITIONS OF AGRICULTURE (INTOXICATION WITH PESTICIDES)

Pesticides are chemical matters, which are used in order to destroy live organisms – bacteria, viruses, spores, fungi, insects, rodents, as well as plants, which damage agricultural cultures and animals.

The chemical method to protect plants and animals is still one of the most convenient, cheap and effective ones. It conditions a significant growth of production and utilization of pesticides and a big number of people are in contact with it.

Pesticides, which are active biological compounds, can have a negative impact onto useful insects and animals, and have negative impact onto the health of people and cause poisoning.

The committee of WHO experts considers that the danger with pesticide poisoning endangers mostly workers of enterprises, where these compounds are made, also those who are directly involved into pesticide utilizations, as well as those who stay in premises treated with pesticides. However, significant expansion of the production and scales of pesticide utilization led to contamination of soil and water reserves with them, what caused the contact of almost all people all over the world with them.

As pesticides, a significant number of chemical matters are used, which vary as to their chemical structure, action character, etc. With the purpose of convenience at the production utilization of pesticides, development and implementation of means, directed at the prevention of possible intoxications, various classifications of pesticide preparations are used: production, chemical and hygienic.

In the basis of production classification, there are two indicators: purpose of pesticides and the goal of their utilization. In compliance with this classification, pesticides against various insects called insecticides; against bacteria – bactericides; against fungi – fungicides; against weeds – herbicides, etc. This classification includes such preparations, which are used to destroy leaves of plants – defoliants, and herbal drying – desiccants.

Based on the chemical structure, there are chlororganic compounds (COC), phosphororganic compounds (POC), and mercurious organic compounds (MOC), derivatives of aminoformic acid, etc.

Hygienic classification envisages the division of pesticide preparations according to the degree of their toxicity with the consideration of mean-lethal dosage – $ЛД_{50}$ (drastic, high-toxic, meant-toxic and little toxic matters), the degree of volatility, cumulation, durability, etc.

Main ways of pesticides permeating into organism are respiratory organs, gastric system, and skin.

According to the clinical progressing, there are acute, sub-acute and chronic intoxications. Acute intoxications develop in the result of permeation of a great number of pesticides into the organism. The development of acute intoxications there are the following periods: latent (from the moment of permeation of poisoning into the organism to the first indications of intoxications); the period of forerunner, for which it is characteristic to have non-specific, one-type reactions of the organism under the influence of many chemical compounds (nausea, vomiting, general weakness, and headache); period of marked intoxication, when together with changes, general for many chemical matters, specific poisons onto organism can take place. For sub-acute intoxications, not such a vivid reaction of the organism onto the action of poison and longer duration of the progressing of pathological process are characteristic, than with acute poisoning. Chronic intoxication develops in case of prolonged permeation of relatively small amount of pesticides into the organism (sometimes for several years).

Clinical pattern of intoxication with pesticides is mostly made of manifestations of their polytrophic action onto the organism, for which it is characteristic to have the development of pathological changes in various organs and systems. Due to the fact, at the development of both acute and chronic intoxications, it is possible to distinguish a number of clinical syndromes.

Thus, at the development of acute intoxication, it is possible to distinguish neurotoxic syndrome, conditioned by the impact of poison onto the nervous system. It is manifested by the headache, dizziness, and various disorders of the consciousness (agitation, dormancy, and coma). Coma can be accompanied by movement reactions, up to the development of clinical and toxic spasms.

At severe poisoning with COC and POC, as a rule, neurotoxic syndrome appears initially, and the clinics of severe acute intoxication can be manifested by the coma in the result of direct toxic action of poisons onto the central nervous system. Coma can develop secondarily as well, in the result of dysfunctional metabolic disorders. Second on the frequency acute syndrome of intoxication with pesticides – gastroenteric syndrome, this develops initially in case of pre-oral permeation of MOC, COC and POC. The development of this syndrome is accompanied by vomiting, nausea, diarrhea and pain in stomach.

Syndrome of respiratory disorders is conditioned by several indicators. COC, POC, MOC and other pesticides inhibit respiratory centers, located in the medulla. When poisoning with POC, derivatives of aminoformic acid is damaged by the intervention of diaphragm and other muscles, which participate in the act of respiration.

The syndrome of affection of cardiovascular system appears at the action of many pesticides and is the result of both affection of central sectors of nervous system (COC and POC), and of direct action of poisoning onto the cardiomuscle (POC) and vascular system – walls of vessels (MOC, arsenious pesticides). At this, there are various disorders of rhythm (tachycardia, at the action of MOC and COC; bradycardia at the affection of POC), what is often accompanied by worsening of contractive ability of myocardium with the development of deficiency of blood circulation and swelling of lungs. The arterial pressure can be increased up to 200/140 mm of mercury column at poisoning with POC and the decrease up to the development of collapse.

Hepatorenal syndrome, and in severe cases – acute renal and liver insufficiency, can develop initially at direct action of pesticides onto parenchymatous cells of liver and kidneys. Repeated development of this syndrome is possible in the result of toxic shock, prolonged disorder of hemodynamics, which is accompanied by the fall of the arterial pressure and the volume of circulating blood.

In the clinical pattern of c h r o n i c intoxication with pesticides are mostly observed by changes from the side of the nervous system. At the initial stages of intoxication, syndromes can be outlined, which are conditioned by functional disorder of the central nervous system – asthenic and asthenovegetative. In cases of severe intoxication, there is a threat of the development of organic disorders of the cerebrum – toxic encephalopathy.

The impact of Trichlorfon, COC and arsenious pesticides can cause the affection of toxic sensory, and vegetative-sensory polyneuritis. In severe cases of chronic intoxication, mercury and chlororganic pesticides, the development of diffusive affection of the nervous system on the type of encephalomyelopolyradiculoneuritis.

The disorder of the stomach system (chronic gastritis, dyskinesia of bile tracts, cholecystitis, pancreatitis, and colitis) can be often observed at affection with COC, POC, etc.

Many pesticides cause changes in the system of haemopoiesis. Thus, at long impact of COC and POC, anemia and leucopoenia can develop; toxic grain in neutrophils can appear. Metaphos, many carbamates cause anemia, reticulocytosis, and assist to the formation of methemoglobin. Pesticides, which contain copper, can cause hemolytic syndrome, Warfarin-containing ones – hemorrhagic syndrome.

Some pesticides (COC, POC, MOC, and arsenious compounds) have allergic affection of the skin, asthmatic bronchitis, and bronchial asthma, toxic and allergic myocarditis.

Intoxication with Organophosphorus Compounds

Organophosphorus compounds (POC) are widely used in the industry and medicine. Many POC are active multifunctional additive to lubricants. They combine properties of washing, anti-corrosive and antiwear additives and are anti-oxidant and depressants. Besides, POC is used in the industry at flotation of ores, polymerization, in production of solvents, etc.

As medicinal means, POC is used to treat glaucomas, myasthenias, atony of intestines, chemiotherapy, tuberculosis and cancer.

POC can be distinguished by high biological activity, and many of them are the strongest of all the known poisons,

As to the chemical structure, POC is ether of the following:

1) phosphoric acid (dibromide and gardon);
2) thiophosphoric acid (Thiophos, metaphos, and methyl mercaptophos);
3) dithionic phosphoric acid (carbophos, phosphamide and amiphos);
4) phosphonic acid (Trichlorfon);
5) amides of pyrophosphoric acid (oct-methyl).

POC can be used as highly effective insecticides, acaricides, defoliants, as well as pesticides to protect crops of cotton-wool, orchid trees, grain and a number of agricultural crops from pests.

The majority of POC have sharp unpleasant smell. They are unstable in the environment, can be easily ruined in case of thermal treatment. There is data on unfavorable impact of POC onto the process of embryo development.

Mostly, poisons with POC are the result of the violation of work rules with this matter during agricultural work or in the process of the production of these compounds. Poisoning can take place via skin, or when inhaling its vapors. POC can be extracted via urine.

Pathogenesis. The mechanism of the action of POC is based on the inhibitive impact of these compounds onto the ferment of cholinesterase. This ferment plays an important role in the process of synaptic transfer of the nervous impulse in cholinergic formations. The action of POC onto cholinesterase causes the formation of stable of phosphorized ferment. Phosphorized cholinesterase (cholinesterase + the residual of POC, which contains phosphorus) hydrolyzes very slowly, without enabling the ferment to catalyze various reactions.

From the pharmacological point of view, all symptoms of poisoning with POC, which can be considered as effects, caused by acetylcholines, and can be divided into three groups: muscarinic-like, nicotine-like and central.

Muscarinic effect can be clinically characterized by ample, feeling of pressure in chest, bronchospasms, increase of bronchial secretion,

hypersalvation, loss of taste, nausea, vomiting, pain in stomach, diarrhea, narrowing of irises, and bradycardia.

Nicotine effect is conditioned by irritation of choline receptors. It is manifested through twitching of muscles of eyelids, face, neck, tongue, and the increase of the arterial blood tension.

The central effect can be characterized by a headache, feeling of anxiety, sleep disorder, psyche disorder and spasms.

Pathologic and anatomic pattern. At gistologic research, it is morphologically possible to find phenomena of blurred swelling in the liver and heart muscle, atheroma of liver, and changes in the kidney parenchyma. Early changes are phenomena of vacuolization in saliva and mucous tunic and the decrease of mitochondrion in them.

Clinics. A c u t e p o i s o n i n g. Symptoms of acute poisoning take place suddenly, its progressing can be mild, mean and severe.

At *mild form* of poisoning with POC there is general weakness, moderate headache, light dizziness, nausea, and excessive salivation. At clinical research, it is possible to note moderate paleness of skin coverlets and the change of the frequency of the heart contraction, dullness of tones, and single type coarseness in lungs. All these phenomena can stay for several hours, maximum - for a day.

In case of *mean poisoning*, there are marked disorders of the central nervous system. Patients have depression, apathy, headache, speaking disorders, increase of the threshold of analyzers of vision, taste and smell. There can be dystrophy of myocardium, accompanied by the decrease of the arterial blood pressure, some increase and pain in the liver, small proteinuria and microheamturia. The duration of this stage is from several hours to several days.

Severe form of poisoning with POC can be characterized by polysymptom pattern. At first, there is movement disorder, sometimes, psychosis, which is accompanied by vision and auditory hallucinations. There are fibril twitching of eyelids, tongue, face and neck muscles, and with time accompanied by spasms of epileptoforming or clone-tonic character. Then, a patient loses consciousness and deep coma takes place. Specific smell of poisonous chemicals can be smelt from the mouth of a patient. The secretion of salivary and bronchial glands fills in the mouth cavity, and lumen of respiratory tracts. There are signs of respiratory insufficiency: dyspnea, and cyanosis. In addition, the development of pneumonia and lung swelling can be observed.

Well marked dystrophic changes in myocardium can be observed, manifested by the dullness of tones and tachycardia. Stable hypotonia takes place. Together with it, patients have signs of toxic affection of liver and kidneys: increase of sizes ad painfulness, proteinuria and microhematuria. The body temperature at poisoning of POC is normal as a rule, a little

subfebrile, and only at lasting coma, hypertermia can take place. Severe progressing of poisoning can be often accompanied by hyperleukemia and glycosuria.

Thus, symptoms of acute poising with PC are various, conditioned by the agitation of the autonomous nervous system. As a rule, the first are muscarine-like symptoms, and then nicotine-like and then central ones.

C h r o n i c p o i s o n i n g with POC is possible at long contact with small dosages of preparations.

Pathogenesis of chronicpoisonin is maybe more complicated and less studies than the acute one. It is not always possible to determine the meaning of the cholisterase inhibition.

Clinics. In the clinical patter of chronic poisoning, there are mostly vegetative disorders with clear exaggeration of cholinergic effect (hypotonia and bradycardia) or functional disorder of internal organs – liver and heart.

When examining people, who have worked for a long time in the POC production, there are phenomena of vegetative dystonia (stable red dermographism, acrocyanosis, and acutely positive clinostatic reflex).

Sometimes, there is the disorder of the central and peripheric nervous system, language disorders, finger trembling, and spastic paralyses. In rare cases, there are psychological disorders: hallucination and depression. There are characteristic disorders of carbohydrate and protein forming functions of liver (change of sugar curve, decrease of the concentration of albumin and the increase of globulin mostly due to α_1-, α_2-, and β-fractions), as well as inhibition of the secretor function of the stomach.

Treatment. Tactics of provision of the first aid to patients is determined depending on the way of permeation to some pesticide. In case of permeation of pesticide preparation by inhalation, it is necessary to take the patient out of the contaminated zone, change his/her clothes, and remove obstacles for free breathing. If the preparation touched the skin, it is necessary to wash it with warm water and soap, treat with the ammonia solution (5 – 10 %), or chloramines (2 – 5 %). Eyes should be washed with warm water or the solution of sodium hydrocarbonate. When the pesticide gets into the stomach, it is necessary to cause vomiting, flush stomach with warm water, 2 % solution of sodium hydrocarbonate, 0.25 % - 0.5 % solution of potassium permanganate. To remove it from intestines, it is necessary to utilize high siphon enema or saline laxatives: 20 to 30 g of magnesium sulfate or salt cake in a glass of water.

To administer adsorbed pesticide preparation is mostly used as a method of forced diuresis. During 2 to 3 years, patients are given water burden – isotonic solution of sodium chloride is administered intravenously and 5 % glucose solution (1.5 to 2.0 l). A full time catheter is introduced to the urinary bladder for diuresis measuring by the hour, and then

intravenously – 30 % solution of the urine, prepared based on 10 % solution of glucose or 10 % solution of manit. The solution of diuretic matters is introduced in a flow for 10 to 20 min based on the calculation 1 g per 1 kg of the patient's mass. After water burden, it is possible to administer furosemide intravenously in the amount of 40 to 200 mg. Furosemide is also recommended after the introduction of the urea, if diuretic effect was failed to be called. After administering of diuretic preparations, it is necessary to continue water burden, which includes 4.5 g of calcium chloride, 6 g of sodium chloride and 10 g of glucose per 1 l of water. Similar cycle can be repeated in 4 to 5 hours until poison disappears from the blood flow.

Detoxication of organism is recommended by the method of hemosorption.

An important peculiarity of urgent therapy at acute poisoning with POC is to use specific antidote means as soon as possible. They include: anticholinergic drug and cholinesterase reactivator. Effective antidote is first of all atropine sulfate, 0.1 % solution is introduced hypodermically (1 ml) or intravenously (2 to 4 ml) in 5 % solution of glucose, at the necessity - and then (20 to 80 ml and more per day until threatening of symptoms of intoxication to life disappear). Good result is provided by utilization of anticholinergic drug of the central and peripheric action: arpenal (1 to 2 ml of 2 to 5 % solution hypodermically or intramuscularly), aprophen (1 to 2 ml of 1 % solution hypodermically or intramuscularly), benactyzine (0.001 to 0.002 g inside 3 to 6 times a day). Throughout the first three days, it is necessary to join anticholinergic drugs, which remove "muscarinic" and "nicotine" effects, with cholinesterase reactivators. Among preparations of this group, the most widely used are trimedoxime bromide (1 ml single dosage of 15 % solution, the treatment course is from 3-4 to 7-10 ml intramuscularly or intravenously), isonitrosine (3 ml of 40 % solution, for the treatment course - up to 4 g).

To release nicotine-like reaction, it is necessary to administer ganglionic blockers: benzo-hexamethonium (0.5 – 1.5 ml of 2 % solution), pentaime (0.1-0.3 ml of 5 % solution), and hygronium (1 ml of 0.1 % solution).

In case of appearing of POC poisoning, which is accompanied by movement agitation and spasms, it is necessary to use sodium oxybutyrate (40 ml of 10 % solution intramuscularly or intravenously), magnesium sulfate (20 ml of 25 % solution intramuscularly or intravenously).

Urgent therapy at acute POC poisoning should include a complex of intensive therapy, directed at the support of functions of the central nervous system, cardiovascular and respiratory systems, as well as liver, kidney and blood.

Treatment of chronic POC intoxication is mostly symptomatic, with the consideration of main clinical syndromes, through which intoxication is shown in every separate case.

Verification of the ability to work. After having acute POC intoxication in the mild form, as well as in case of weak manifestation of chronic impact (moderate astehnisation, vegetative and vascular dystonia), the ability of patients is kept. In such cases, where there is acute intoxication or marked chronic intoxication, further work in the contact with toxic matters, with much physical loading is prohibited.

Preventive measures. Prevention of POC poisonings includes a complex of hygienic, sanitary and technical, treatment-preventive and other measures.

Among hygienic measures, first of all, it is necessary to note the following: hygienic selection of pesticide preparations, replacing of hazardous pesticides with less hazardous ones, and hygienic reglamentation of POC utilization.

Sanitary and technical measures can include the following: improvement of methods and ways to use pesticides, as well as rational work organization.

Treatment and preventive measures include preventive and periodical medical examinations. It is necessary to remember that teenagers below 18, men over 55, women over 50, pregnant and breast-feeding women, as well as those who underwent infectious diseases or surgeries for the last 12 months.

Medical examinations should include utilization of a complex of laboratory research. When contacting with pesticides of all groups the following should be done: blood research (hemoglobin, leukocytes, and ESR); X-ray; defining of the content of bilirubin, fructose monophosphate-aldolase in the blood serum, and the general urine analysis.

An important diagnostic criterion for those who work in contact with POC is the activity of cholinesterase in the blood serum and erythrocytes. The decrease of the activity of cholinesterase by 25 % of its initial meaning is the reason to terminate the contact with pesticides of this group.

During the period of intensive work in contact with POC, it is recommended to give 0.5 g of pancreatin two or three times a day, which connects POC and has positive impact onto the functions of gastro-intestinal tract.

Intoxication with Chlororganic Compounds

Chlororganic compounds (COC) are referred to the most widely used pesticides. Until now, the following ones are used most often: hexachloran, dichlorodiphenyltrichloroethane, polychloropinene, chlorindan, heptachlor, dildrin and polychlorpinen. The peculiarity of COC is its high resistance in

the environment. This can be explained by the fact that the temperature, humidity, acids, and alkaline do not have any impact onto them and do not ruin their microorganisms. These pesticide organisms get into the organism with water, food, respiratory system, skin and placenta. They can be discharged by kidneys, intestines, and milk glands. Besides, COC can create deposits in the organism, in particular in adipose tissue. From this deposit, they permeate to blood and stay there for a long period. COC are not as toxic as POC, however they are more dangerous as they can cause chronic poisoning. As to their character, they are neurotropic and parenchymatous poisons.

COC is a strong allergen matter. After contact with it, the following diseases can appear: bronchial asthma, hives, allergic rhinitis, dermatitis and eczema. Besides that, COC can cause gonad-toxic and embryo-toxic action; in the experiment under the impact of small concentrations, the duration changes, and the number of estrous cycles decreases, number of fruits decreases as well. Their teratogenic action has been noted as well; vital capacity of born animals is relatively less, their mass gain is slower, and they are behind in their physical development.

Pathogenesis. Mechanism of the action of COC onto the body of a person has been determined completely yet. It is supposed that the initial factor of the action of COC is the inhibition of ferment systems of the organism, which condition conditioned reflex activity, morphological disorders and clinical manifestations.

All COC are strong protoplasmic poisons, which affect the nervous system and parenchymatous organs. Thus, clinical pattern of poisoning with COC can be characterized by significant polymorphism.

Pathologic and anatomic pattern. At acute poisoning with COC, well marked plethora of internal organs and brain, fine nuclear and diffusive hemorrhages into lungs can be observed. As to the histological state, there is swelling of vascular walls, and dystrophic changes of the nervous cells can be observed in the cerebral cortex; single fine nuclear infiltrates from cells of lymphoid type and histiocytes can be noted in the heart muscle; as well as swelling of cells of liver and kidneys.

At chronic action of COC, perivascular swelling with dystrophic changes of nervous cells of the cerebral cortex can be observed. There are areas of hemorrhages and degenerative –inflammatory changes in lungs, liver, kidneys and myocardium.

Clinics. In the clinical pattern of poisons with COC there are acute and chronic poisons.

A c u t e p o i s o n i n g. Their clinics depend on the way of permeation of the pesticide into the organism. In case of permeation through respiratory organs, the pattern appears in the first 1 or 2 years. State of coma develops; salivation and bronchorrhea increase; respiration

is affected; and collapse can be noticed. Death comes due to inhibition of cardiovascular activity and paralysis of the respiratory center. If a patient survives, he/she develops toxic affection of liver and kidneys (acute renohepatic insufficiency).

Mild inhalation poisoning is manifested through the headache, general weakness, irritation of mucous tunic of the upper respiratory ways, and coughing. There can be nausea, vomiting, moderate increase of liver and pain in the right hypochondrium.

In case of permeation of pesticide to the gastrointestinal tract, at first there are gastric disorders, then the disorder of the function of the central nervous system. In severe cases, dyspnea, cyanosis, increased agitation, trembling, ataxia, fits of clonic and tonic spasms, psyche disorder, vision affection, as well as symptoms of the affection of liver, kidneys, heart and lungs, accompanied by signs of acidosis can take place.

When some products permeate through the skin the following symptoms can take place: skin reddening, rash and dermatitis.

C h r o n i c p o i s o n i n g. They can be characterized by a headache, increased fatigability and irritation, sleep disorder and weight loss. There are signs of the affection of the nervous system (vegetative dystonia) and the pathology of internal organs. At the earliest stage of intoxication, neurological disorder can be manifested through non-specific toxic asthenia. Sudden headache with nausea, general weakness and profuse sweating or fit-like dizziness, accompanied by skin paling and bradycardia can take place.

In later stages of chronic intoxication with COC, the pathological progress involves peripheral nervous system (vegetative-sensor polyneuritis).

The disorder of cardio-vascular system can be characterized by mostly vegetative and vascular dystonia with the inclination to the arterial hypotony, as well as extracardial disorders with cardiac rhythm (sinus bradycardia) and functions of myocardium conductivity. Often toxic dystrophy of myocardium or myocarditis of toxic and allergic character can often develop, in particular among those, who have gone through acute COC intoxication.

Already in early stages of chronic intoxication with COC, secretor function of the stomach is in disorder, for more marked stages of the characteristic development of chronic gastritis with the inhibition of the secretor function of the stomach, up to histamine-resistant achylia.

The disorder of the functional state of the liver at chronic intoxication, at first, can be manifested through the increase of the activity of organo-specific ferments in the blood serum, and later it is accompanied by hydrocarbon and anti-toxic functions. At severe cases of intoxication, toxic hepatitis can develop, which develops without jaundice.

As to kidneys, it is characteristic to have some phases in the development of function disorders: at the initial stage, functional activity increases due to the increase of blood circulation in kidneys and glomerular filtration, and at later stages due to the development of toxic nephropathy of kidney functions can be significantly affected, and sings of azotemia takes place.

The analysis of blood shows that the anemia, moderate leucopenia, relative lymphocytosis, and eosiopenia. The number of trombocytes decreases and ESR slows down.

Thus, COC causes the affection, first, of the nervous system and parenchymatous organs, and liver suffers most of it. Due to that, COC is referred to hepatotropic poisons.

Treatment. General principles of the provision of the first aid are analogous by the fact that when POC poisoning. Besides, it is necessary to take into consideration, that at acute poisoning with COC, especially when the pathological process involve kidneys, hemodialysis is recommended. A good result is also given by the utilization of peritoneal dialysis in connection of accumulation of COC in fat deposits, as well as hemosorption.

To treat acute poisoning with COC, it is necessary to use anti-oxidants, which prevent their oxidation, formation of toxic products of their transformation. They include some vitamins and amino acids (α-tocopherol and galascorbin).

Treatment of chronic pesticide intoxications is mostly symptomatic, with the consideration of main clinical syndromes, with which intoxication is manifested in every specific case.

In case of functional disorders of the central nervous system treatment should be complex, with the utilization of medicinal drugs, physiotherapeutic procedures, therapeutic exercises, keeping to the labor schedule, rest and feeding.

In case of vegetative and vascular dystonia it is necessary to have preparations, which have adrenolytic action (ergotamine and dihydroergotamine), as well as anticholinergic drugs (atropine). At the presence of angiospasms and arterial hypertension, electrophoresis with magnesium sulfate is recommended. Good results have radon baths, elecrophoresis with Novocain onto the collar zone.

If the pathological process involves hypothalamic area, depending on the character of crises it is possible to use spasmpolitic, adrenolitic and cholinolitic preparations, as well as ganglioblockators. It is recommended to use piroxane, and antihistamine preparations (Dimedrol and suprastin).

With the purpose of desintoxication, it is advised to use glucose with ascorbic acid, vitamin preparations, glutamine acid, and oxygen hypodermically.

To liquidate pain, it is recommended to utilize analgin; at vegetative character of this feeling, it is recommended to utilize pachycarpin, gangleron and aminazine.

At changes in cardio-vascular system, treatment measures are directed at the normalization of vascular tone – utilization of toning or sedative preparations. Oxygenotherapy, also oxygen, salt and pine needle bath, and nitrogen baths.

In case of dystrophic changes of myocardium to improve exchange processes in cardiac muscle, it is necessary to include vitamins of group B (thiamine, pyridoxine and cyanocobalamin), as well as ascorbic acid and retinal.

Good results onto energetic processes in myocardium are given by riboxine, and potassium salt.

At toxic hepatitis together with action therapy it is recommended to use preparations, which improve the exchange in liver cells, vitamins of group B, antioxidants (acetate tocopherol) unithiol and sirepar. In severe cases, hormonotherapy is recommended.

Considering the fact that toxic hepatitis is complicated with cholicestite, it is widely recommended to use antispastic preparations (no-shpa), antibiotics, “blind” dosage with utilization of cholagogue preparations.

Verification of the ability to work. In case of acute COC poisoning, as a rule, it is recommended to temporarily terminate from work, which are connected with the action of toxic matters.

At the presence of chronic intoxication, patients should be transferred to the work beyond the contact with pesticides; also work with hard physical burden and under conditions of intensive sun irradiation. It is also necessary to terminate any further contact with COC at relapse, organic affection of the nervous system and toxic hepatitis.

Prevention measures. To prevent intoxication with COC the following is important: qualitative render of preliminary and periodical medical examinations; thorough sanitary observation with prevention and utilization of pesticides; utilization of individual protection means when working with them (duration of the work day should be limited by 6 hours when working in contact with hexachloran, heptachloran, etc).

Intoxication with Mercuric Organic Compounds

Mercuric organic compounds (MOC) refer to the most effective pesticides. They are used in industry (e.g. in paper industry to decontaminate woods), they are a portion of paints, which are used to cover bottoms of ships, hydroplane, internal surface of water pipes, and submarine acoustical devices.

Main mercuric organic pesticides: ethyl mercuric phosphate, granosan, and mercuran. All the preparations of this group are highly toxic, with marked cumulative properties.

Boundary permitted concentration of the content of mercury for all the matters is 0.005 mg/m^3.

It permeates into the organism through respiratory organs, gastrointestinal tract and skin. They circulate in blood, can be found in all biosubstrates; and they permeate into blood through placenta. It accumulates in the cerebrum, liver, kidneys, and adrenal glands.

These compounds discharge very slowly, mostly through kidneys and esophagus, as well as with mild, then with bile and saliva. MOC are more toxic than non-organic mercuric compounds. They are referred to enzymatic poisons. Besides, MOC possess well-marked gonado-toxic, embryotoxic and mutagenic effect. It has also been stated that these compounds cause allergenic action as well.

Pathogenesis. MOC acts onto the organism with metal mercury. They are protoplasmic ferment poisons and have impact onto carbothiolic groups of numerous cellular enzymes, which catalyses various types of exchange. It has been known that for normal functioning of ferments of their sulfahydril groups should be free. Bonding with compounds of mercury, they are blocked and in the result, the ferment inactivates, what leads to deep disorders in the tissue exchange. Besides, mercuric compounds stay in the cell membrane, affecting metabolism of cells and decreasing their content in RNA. The development of pathologic changes in various organs is much assisted by capillary-toxic action of MOC.

Pathologic and anatomic pattern. Mercuric organic pesticides cause sharp fatigability, symmetric atrophy of the cortex of frontal lobe and cerebellar hemispheres. Plethora of folds of small intestine, small hemorrhages under pericardium and endocardium of the left heart ventricle, and under the mucous coat of stomach can be observed. Histologically, nervous cells of the grain layer of cerebellar cortex are wrinkled; neuroglia expanding in the layer of Purkinier cells can be noted. Dissolving of mieline can be observed in lateral columns of the spinal cord and myeline coat of cauda equina roots. There are dystrophic changes in the liver, kidneys, and heart muscle.

Clinics. A c u t e p o i s o n i n g. At permeation of pesticides through the respiration organs, clinical manifestation is manifested rather fast, and through gastrointestinal it takes place a bit later. In general some consecution in the dependence on the way of pesticide permeation into the organism can be observed: at inhalation permeation – first of all, changes start in the nervous system and in case of permeation of poisoning through a mouth – dyspeptic phenomena can be observed.

Initial manifestations of poisoning are manifested through illness, general weakness, headache, and dyspeptic disorders. In the mouth, there can be unpleasant “metallic” taste, and in particular cases, gums swell and bleed. Asthenovegetative syndrome develops. This period of the disease lasts for several days, and then the state of the patient worsens.

Some patients have ulcerous gingivitis and stomatitis. Signs of the affection of the central and peripheral nervous system develop. Lumbosacral radiculitis, polyradiculoneuritis, toxic and encephalomyeloneuritis develop; pain and parastesia appear in extremities, mostly in distal sectors; there are also superficial disorders in the form of “gloves”, “socks’ and “stockings”. Distal sectors of extremities become cyanotic and cold.

In severe cases, walking disorders, memory decreases, trembling of stretched upper limbs takes place and speaking is unclear.

Some patients have a clinical picture with features, characteristic for the diencephalons syndrome. The temperature and arterial pressure increase, palpitation, body trembling and constant thirst take place.

Besides, nervous system, gastrointestinal channel and kidneys suffer as well. Appetite decreases, nausea and vomiting, pain in right hypochondrium, liver increases, and gingivitis develops. In kidneys – toxic nephrosis.

Patients have pain in heart and palpitation. The development of toxic myocarditis is possible. Arterial pressure usually decreases.

For the affection of organs of sense are characteristic disorders of scent, decrease of hearing and sight.

For severe poisoning, it is characteristic to have moderate anemia, some toxicosis with the shift to the left, toxic grain of leucocytes, and the decrease of monocytes.

C h r o n i c p o i s o n i n g. Chronic intoxication with MOC develops in the form of more or less outlined clinical stages. At the *initial stage*, there is an asthenovgetative syndrome with elements of erythrism. If the action of pesticides is continued, then trembling of extremities increases, and character that is more marked gets vegetative disorder, including erythrism.

The *second stage* of the MOC chronic intoxication can be characterized by major affection of hypothalamus. In the clinical pattern, there are disorders of sleep, melancholy, unexplained fear, disorder of thermal regulation and cachexy.

At the *third state*, organic nucleuses or diffusive disorders of the central nervous system are brought to the forefront: mostly toxic encephalitis and encephalomyelitis develop.

Together with the development of the pathology of the nervous system on the clinical pattern of the chronic intoxication with MOC there

can be signs of toxic dystrophy of myocardium can develop, as well as toxic hepatitis, for which it is not characteristic to have fast progressing and the development of jaundice.

Often, at the chronic intoxication with MOC, hypochromic anemia develops, anisocytosis and poikilocytosis appear, and the content of reticulocytes increases. The number of leukocytes first increases and then leucopoenia develops. The content of thrombocytes decreases and the ESR increases.

One of the most important signs of the MOC impact onto the organism, there is presence of mercury in biological environments. At intoxications of mild and mean severity, mercury can be determined in blood and urine; its last excretion is usually more than 0.01 mg/l and increases along the increase of intoxication. However, complete dependence between severe intoxications and the content of mercury in the urine is absent,

Treatment. In case of appearing of acute poisoning with MOC, utilization of antidote therapy should be envisaged (substances of the carbothiolic group). Positive result can be achieved by administering of a domestic preparation of unithiol (5 – 10 ml of 5 % solution intramuscularly or intravenously, every 3 to 6 hours). At the course of treatment, it is necessary to have 50, and in severe cases up to 200 ml of 5 % solution of unithiol. In case of peroral poisoning, peroral can be taken in (100 – 150 ml of 5 % solution) with further gastric lavage.

Besides unithiol it is possible to use complexing agents at poisoning with MOC (tatacin-calcium, pentacin, etc), as well as preparations, which are synthesized based on the physiological metabolites (succimer, 0.3 g of alkaline solution intramuscularly on the first and second days every 6 hours; on the third and fifth days – every 8 hours, and on the sixth and seventh day – every 12 hours).

Besides antidotic therapy, symptomatic treatment is conducted, which is targeted at the normalization of the functional state of main organism systems.

Treatment of chronic poisoning with MOC can be conducted at the presence of some poisoning syndromes. To fix the results of the treatment, it is recommended to prescribe hydrogen sulfide baths in sanatoriums.

Verification of the ability to work. In case of the suspicion of the presence of intoxication (mercury presence n urine), it is necessary to relieve the sick temporarily from further contact with mercury. In case of “mild” intoxications, and moreover after acute poisoning with mean and severe phases, it is recommended to terminate any contact with mercury in full. It is also possible to provide a person with occupational invalidism.

Preventive measures. Sealing-in of machines, utilization of individual protecting means (respirators, overalls, protective glasses, and

rubber gloves), and removal of manual operations. In case of necessity, it is recommended to conduct demercurization of the premises, aerosol inhalations with 5 % solution of unithiol in the dosage of 5 ml three ties a week during a month, as well as preliminary and periodical medical examinations of workers who contact with mercury.

Intoxication with Carbamates

Carbamates, which are used as pesticides, are derivatives of aminoformic, thiocarbamic and dithiocarbamic acids. As pesticides, ethers of aniformic acid, oxide salts of alkali element and heavy metals of dithiocarbamic acid.

Preparations of this group are used as herbicides, insecticides, acaricide, fungicides, and bactericides. The majority of carbamates are highly toxic for insects and little or medium toxic for people. It is very important that carbamates have impact onto insects, which are not sensitive to POC and COC.

Together with these carbamates, there are a number of significant drawbacks. Thus, they destroy a number of useful insects, and are very toxic for earthworms.

Carbamades are poisons of parenchymatous and neurotropic actions. Besides, some carbamates are characteristic to have embryotoxic, gonadotropic, teratogenic and mutagenic actions, many of them are active allergens, and some of them are cancerogenic.

Pathogenesis. Carbamates are “direct” inhibitors of cholesterase. In the process of joining of the ferment with carbamates, there is cholesterase carbamilizing with formation of a complex of “carbamate-cholesterase”. It is characteristic that this complex is very unstable. This cholinesterase is capable to spontaneous reactivation.

The mechanism of actions of ditiocarbamates is reduced to inhibition of ferments of oxide-renovating cycle; most likely, in the result of the interaction with their sulfhydryl groups. Besides, ditiocarbamates disturb carbohydrate exchange, by increasing glycolitic processes and inhibit the accumulation of oxygen by tissues. In the process of decomposition of ditiocarbamates, the following is discharged: carbon bisulfide, which is bound with amino groups of amino acids, peptides, and proteins, it blocks them, disturbs protein exchange and causes changes in many organs and systems. Thus, carbon bisulfide, which is formed in the organism endogenically in the process of metabolism of ditiocarbonates, is a significant component of their toxic impact. Besides, it has been stated that ditiocarbonates disturb the exchange of microelements.

Clinics. When getting poisoned with carbamates, there are symptoms of the irritation of parasympatic nervous system: narrowing of pupils, spasm of accommodation, bradycardia, hypersalivvation, fibril twitching of

muscles, there is nausea, vomiting, pain in abdomen, and diarrhea. Further, there are phenomena of bronchorrhea, bronchospasms, and pulmonary swelling is possible. As to the nervous system, at first, agitation takes place, then entanglement of consciousness, and spasms. In the blood, there is a decrease of cholinesterase. Dermatosis, nettle-rash, conjunctivitis, and irritation of the upper respiratory tracts can develop (rhinitis, bronchitis and tracheitis).

The peculiarity of the impact of carbamates onto the organism is their ability to cause changes in the structure and functions of glands, which have no ducts, and first of all, of thyroid gland.

Many carbamates cause the affection of the nervous system and organs of haemopoiesis, have allergenic properties and form methemoglobin. Sevin, cineb and ciram are the most toxic for people.

POISONING WITH SEVIN. Sevin is naphthylmethylcarbamate, and is referred to derivatives of aminoformic acid. This compound with the most marked insecticide properties comparing with all carbamates has anticholesterase activity.

Acute poisoning with sevin is characterized by agitation of M and N – cholinoreceptors. The following takes place: headache, dizziness, nausea, vomiting, salvation, coughing, and complicated breathing. In lungs, there are dry crepitations. The preparation inhibits the immune reactivity of the organism and causes various allergic reactions of respiratory organs and skin.

The earliest symptom of poisoning with Sevin is the decrease of the activity and cholinestase. Based on other indications, some diagnostic meaning is possessed by the decrease of erythrocytes and hemoglobin.

Chronic intoxications with Sevin are not described.

POISONING WITH CINEB. In case of poisoning with cineb, the body temperature can significantly increase, pulse and breathing become more rapid, skin and whites of eyes become bluish. There are cases of hemolytic anemia, sulfhemoglobinemia, which are connected with the impact of carbon bisulfide, formed at the decomposition of the preparation.

POISONING WITH CIRAM. Ciram has strong irritating properties, in particular when getting to the skin, mucous tunic of eyes and upper respiratory ways.

All these preparations inhibit leucociytopoesis. Leucopoenia develops due to the decrease of the content of neutrophil and eosinophilic granulocyte. Together with this, often the content of erythrocytes decreases in blood, and hypochromic anemia develops.

Treatment. At acute poisoning with carbamates, a patient is prescribed to inhale 25 % solution of ammonia. The antidote is atropine (1-2 ml of 0.1 % solution intramuscularly, in case of necessity injections are repeated every 8 to 10 min until state improves). In comparison with

poisoning with POC, overdosage of atropine is dangerous, and utilization of acetyl cholinesterase reactivators is non-expedient (due to fast spontaneous reactivation).

Treatment of patients with chronic poisoning is syndromic (alkali inhalations, disensibilizing preparations, hemostimulators, vitamins E, thiamine and pirodixin) together with amino acids, which contain sulfur and copper preparations.

Verification of the ability to work. Expert issues can be solved with the consideration of the degree of severity of the undergone poisoning. Mild forms of acute intoxications are reverse, and thus the ability to work of such people is not disturbed. In case of more severe poisoning and at chronic intoxications, further work under condition of the action of carbamates is contraindicated.

Preventive measures. To prevent poisoning with carbamates, it is necessary to keep to generally accepting preventive measures. During the period of intensive work in contact with these pesticides, it is necessary to research the activity of cholinesterase in blood. In case of the decrease of the activity of this ferment by 25 %, further contact with pesticides should be terminated until complete renovation of this indicator.

Intoxication with Compounds which Contain Arsenic

Arsenious compounds, which are used in agriculture, include calcium arsenate and Paris green. These pesticides are highly toxic compounds and they are very stable in the environment. These substances permeate into the organism via lungs, gastrointestinal channel and damaged skin. They are discharged with urine, bile, faeces, as well as with excretions sweat-glands, mammary glands, and with exhaled air. These preparations are capable to accumulate in bones, liver, kidneys, and mucous tunic of the stomach, skin, hair and nails. After exceeding the blood-brain barrier, they deposit in hypophysis.

Pathogenesis. Compounds of arsenic lock numerous ferment processes, disturbing carbohydrate and fatty exchange, as well as tissue respiration. Carbothiolic ferments, which form stable toxic compounds with sulfhydryl groups, are the most sensitive to the action of arsenious compounds. Arsenious compounds increase the permeability of walls of capillars and cause hemolysis of erythrocytes. Small dosages of arsenious preparations stimulate haemopoiesis, and big ones – inhibit it, up to the development of hypoplastic anemia.

Pathologic and anatomic pattern. At the action of pesticide preparations with arsenic onto the blood of a human being, plethora of internal organs, small hemorrhages into the pleura and pericardium, fatty degeneration of the liver with the transition to atrophy, as well as the increase of spleen with the decomposition of erythrocytes. In various

sections of the central nervous system, there are hemorrhages, nucleus affection of cells of lateral horn and anterior horn of the spinal cord, and peripheral nerves. And in marrow, hyaline degeneration is up to the atrophy of myeloid elements.

Clinics. A c u t u t e p o i s o n i n g. Clinical manifestation of poisoning with arsenious compounds depends on the way of the poison permeation, its dosage, concentration and individual sensitivity. According to the type of the permeation of the poison to the organism there are three main clinical forms of acute poisoning: gastrointestinal, paralytic and the one which is manifested through the affection of respiratory tracts.

At gastro-intestinal form, patients get metallic taste in their mouth, burning in pharynx, pain and difficulties when swallowing, sharp pain in stomach, irrepressible vomiting, which can last from several hours to several days. Vomiting can be with a mix of bile, and sometimes of green color due to the presence of arsenious compounds. Then it is accompanied by liquid faeces with tenesmus. Emptying looks like rice-water, sometimes with blood. Due to significant loss of water, organism dehydrates fast. Clinical pattern reminds cholera, and this semblance is increased due to joining by the increased general weakness with dizziness and loss of consciousness. Temperature decreases, cramps in gastrocnemius muscles can take place; the number of urine decreases up to the development of anuria. This form of acute poisoning is often lethal.

Among patients with acute poisoning, gastro-intestinal phenomena do not have time to develop, as paralytic form of poisoning takes place rather rapidly, when general weakness, feeling of fear, drowse, dizziness and coma appear and develop fast. During the state of coma, there can be cramps, and sometimes epileptiform fits. Death can develop within a day.

When the crisis is over, a patient will have signs of diffusive affection of the nervous system in 12 to 14 days: headache, dizziness, and possibly comatose state.

However, peripheral nervous system suffers most often. There are polyneuritis and myelopolyneuronitis. They can be characterized by active progressing, significant expression of pain syndrome, expansion of paralyses, and symmetry of the affection. Mostly, they affect radial nerves and lesser occipital nerve. At first, paresthesia takes place, and then weakness appears in upper and lower limbs, which start with distal sectors and then spreads and progresses up to the degree, that walking becomes unstable and weak due to the affection of muscle sensitivity – atactic form of polyneuritis. In other case, parastesia of bones is accompanied by intensive burning pain. It can appear either spontaneously or from touching. However, only subjective feeling of pain stays very soon, hyperesthesia is replaced with dulling of pain, tactile and temperature

sensitivity, and in distal parts of limbs – anesthesia, which is a specific ability of muscle polyneuritis.

Pareses of soles and fingers appear quickly. In the most severe cases, tetraparesis develops. Often, there is pigmentation and peeling of skin, hair falls out and nails are fragile. Approximately in two months after the disease started, white-gray cross Mees's lines appear on nails – impregnation with arcenious acid, pathognomonic symptom of poisoning with arsenic.

In case of permeation of arcenious compound through respiratory system, initial stages of the disease can be characterized by sharp pain in eyes, lacrimation, nosebleed, coughing, hemoptysis and pain in chest. The temperature increases. In more severe cases, it is also accompanied by diarrhea, loss of appetite, pain in stomach, nausea and sometimes vomiting.

C h r o n i c i n t o x i ca t i o n. In case of systematic permeation of poison into the organism, the amount of which increases gradually, accustoming to it can take place. In the majority of cases, at prolong impact of small dosages of poison into the organism, chronic intoxication develops.

Patients have progressive loss of weight, loss of appetite; often have metal taste in the mouth, general weakness, fast fatigability, and decrease of temperature, sharp pain in eyes, coughing, and nosebleed. Dryness of the mucous tunic of a nose and especially of the mouth cavity is a specific feature of intoxication with arsenious compounds, what makes it look like poisoning with chromium and fluorine.

At objective examination of patients, it is possible to find inflammation of the nasal part of gullet and it covering with ulcers, and sometimes breaking of the nose partitioning, laryngitis, tracheitis and bronchitis.

Periodically, there are dyspeptic phenomena: nausea, vomiting, diarrhea, and pain in stomach. Memory and ability to work worsen. They suffer from headaches, fine trembling of upper limbs and language disorder.

Polyneuritis, which appear at chronic intoxication with arsenic, are very painful, and develop with the affection of sensitivity and have inclination to fast development of repeated contractions, pareses with further muscle atrophy.

It is characteristic for chronic intoxication with arsenious compounds to have significant skin pigmentation – arsenious melanoderma, which starts with inguinal folds and palms and then spreads to peripapillary circles breasts, Sergent's white line and then spreads around the face, locating diffusively or in spots. Face and mucous tunic are not affected. Simultaneously, X-disease develops in these areas.

In some cases, chronic intoxication with arsenious compounds develops with carious affections of haemopoiesis: anemia, leucopoenia and, sometimes, agranulocytosis.

At prolong impact of arsenious compounds, skin cancer can develop.

Treatment. Treatment measures envisage termination of the contact with poison, gastric lavage with the solution of slack magnesia (1 – 1 ½ spoons per 5 glasses of boiled water) with further administering of 25 to 30 g of magnesium sulfate with 400 g of water, and antidotes (Antidotum arsenici or Antidotum metallorum) via a probe.

An antidote is also a mixture of non-dissolved ferrous hydroxide with the solution of magnesia sulphite, which is done right before administering by mixing the solution of slat sulphite (100 units per 300 units of water) and slacked magnesia, which is triturated with water (20 units per 300 units of water). The received solution is mixed and is drunk in the amount of 1 spoon in five minutes.

Positive effect is provided by prescription of carbothiolic preparations: unithiol (5 % solution - 10 ml), sodium tiosulphate (30 % solution – 5 – 50 ml), lipoic acid (0.5 % - 2 ml) and BAL.

In case of development of gastro-intestinal form, it is necessary to administer intravenously a solution of glucose (20 ml – 25-40 %) with ascorbic acid (500 mg); at severe forms, corticosteroids and hemodialisis are recommended.

At arsenious polyneuritis, positive result is shown by administering vitamins (B1, B2, B6, B12, ascorbic and glutamine acids), as well as physiotherapeutic procedures (UV irradiation and ozokerite), massage, and curative gymnastics.

Verification of the ability to work. At mild forms of chronic intoxications, it is recommended to transfer a patient to another job for some period of time with the provision of the sick leave for the period of two months. At marked forms of poisoning, it is recommended to get full-time job, and here can be an issue regarding the transfer of the patient to an occupational invalidism group.

Preventive measures. Individual protection when working with dust includes respirators, protective glasses, and gloves, and keeping to measures of personal hygiene. Preliminary and periodical medical examinations with the content of arsenic in urine in the amount should not be more than 0.5 – 1 mg/l, as well as in hair and nails. Treatment and preventive meals, daily administering of 150 mg of ascorbic acid, and mild (intensifies discharge of arsenic from the body).

Intoxication with Pirethroids

Pirethroids are chemical compounds, which are a very perspective group to use against pests in agriculture. They are synthetic analogues of

natural pirethrins, which are contained in chamomile flowers. Mostly widely spread are representatives of this group of pesticides as decis, rovucuritis, phenopropatrin, permetrin, etc.

The majority of perithorids has low volatility, is badly dissolved in water, and destroys fast under the impact of light. Significant advantage of pirethroids is their high insecticide activity, in the result of what the necessary amount to use is very small, what deceases the danger of the development of intoxication.

Pathogenesis. It has been found out that these preparations are capable to inhibit cholinesterase. By changing the activity of cholinesterase in erythrocytes, piretroids can affect the structure of cell membranes, causing inactivation of Na – K - adenosine triphosphate system, which is located in them.

Besides, pirethroids can change the content of free radicals, cytochrome P-450 and metal complexes in tissues. Many of them are moderate inducers of monooxygenase system, at long impact of piretroid, it is induced with cytochorme P-450 and the activity of NADF·H-cytochrome – C-reductase increases.

Clinics. On the pattern of acute poisoning with pirethroids, symptoms of the affection of the nervous system dominate: trembling, affection of movement coordination, clonic-tonic cramps, and extremities paresis.

Together with this, the affection of piretroids due to hepathotoxic action cause changes of the activity of cholinesterase of the liver and blood serum, ferments, alkaline phosphatase, as well as the decrease of protein and urea in blood serum.

Some preparations of this group cause local irritation.

Prevention of Poisoning with Pesticides

Prevention of poisoning with pesticides includes a complex of hygienic, sanitary-technical, treatment-preventive and other measures.

Among hygienic means, first it is necessary to note the following: hygienic selecting of pesticide preparations; replacement of hazardous pesticides with less dangerous ones; as well as hygienic regulation of pesticide utilization.

Sanitary and technical measures include improvement of ways and methods to utilize pesticides, and rational organizational work.

Treatment and preventive measures include preliminary and periodical medical examinations. It is necessary to remember, that the following categories of people are not permitted to work with pesticides: teenagers up to 18, men over 55 and women over 50, as well as pregnant women, breast feeding women, and also those who had suffered infection diseases or surgeries for the last 12 years.

Medical examinations should certainly include utilization of a complex of laboratory research. When contacting with pesticides, the following should be done for all the groups: blood testing (hemoglobin, leukocytes, and ESR); fluorography, determination of the content of bilirubin, fructose diphosphate aldolase in blood serum; and general urine analysis.

At medical examination of those, who contact with COC, it is necessary to research blood with definition of a number of thrombocytes, transferases, as well as COC in blood and urine.

The most important diagnostic criterion of those, who work in contact with POC, is an activity of cholinesterase by 25 % of its outcome and is an indication to termination of the contact with pesticides of this group.

At medical examination of those, who are in contact with POC, it is necessary to research the state of the mouth cavity through a possible development of gingivitis and stomatitis; diagnostic value has the definition of the content of mercury in urine.

Those, who work with carbamates, are recommended to have determined the specific compounds with the help of methods of G.Khokholkova (diptal), L.Oleksandrova (carbin) and M.Klysenko (Sevin) with diagnostic purpose.

Chapter 5

OCCUPATIONAL DISEASES, CAUSED BY THE ACTION OF MINERAL FERTILIZERS

DISEASES IN THE RESULT OF ACTIONS OF PHOSPHORIC FERTILIZERS

This group of mineral fertilizers include superphosphate (simple, granulated, double, ammonized, granulated with phosphorites of Kara Tau, and manganized), precipitate, fertilizing precipitate, phosphorite flour, metaphosphate, teriophosphates (melted, fluorine-free, and fluorine slakes).

The most spread mineral fertilizers of this group – superphosphates – are a complicated chemical compound, utilization and in particular production of which is connected with the impact of dust, phosphorus, anhydrous hydrogen fluoride and salt of hydrofluoric acid, as well as sulfuric anhydride and nitrous oxide. From all these substances, the most toxic ones are anhydrous hydrogen fluoride and other fluorine compounds.

Pathogenesis. The mechanism of the action of fluorine compounds is first of all connected with the action of fluorine, which is a protoplasmic and ferment poison, it disturbs the processes of glycolysis, mineral exchange, and in particular, calcium and phosphorus, form a hard dissolved calcium fluoride in the organism, what makes calcium more biologically inert.

Clinics. Phosphoric poisons can affect the nervous system, internal organs, locomotor system, and cause allergic reactions. Mild and chronic poisoning can be distinguished.

A c u t e p o i s o n i n g is characterized by acute irritation of eyes and upper respiratory tracts, covering of conjunctives with ulcers, mucous tunic of mucous tunic of eyes, nose, and mouth cavities. Nosebleed takes place and mucous tunic of nose is painful and swollen. In case of deeper penetration of anhydrous hydrogen fluoride and fluorine in respiratory tracts, it is possible that bronchitis, bronchiolitis and toxic pneumonia can develop.

When these compounds permeate inside, clinical pattern of severe gastroenteritis develop, which are manifested with vomiting, bloody flux, sharp pain in abdomen, and subcutaneous hemorrhage. Objectively, the following can take place: agitation, cramps, respiration disorder and the disorder of cardio-vascular system; it is also possible to have kidney affection.

C h r o n i c p o i s o n i n i n g. The most frequent and early disorders in the result of the action of phosphorous mineral fertilizers and

their components are changes in the upper respiratory tracts. They often appear in the first months and can be characterized by complaints on having coryza and insignificant dry cough; besides, throat is always scratchy, and at rhinopharyngoscopy, there is passing, and later stable hyperemia of mucous tunic of a nose and the rare wall of the gullet. If the duration of work with these substances is 2 to 3 years, complicated breathing, thick mucous discharges from a nose and coughing with the discharge of viscous phlegm can be observed. Mucous tunic of a nose is hypertrophied. People with a long period of work in contact with these fertilizers have nucleus, and later – scattered preatrophic of mucous tunic of a nose and a gullet with the transition into atrophy. At first, there is mucous tunic of a nose, and then a pathologic process is expanded onto the mucous tunic of a gullet, and later in some cases – of throat as well. Often, there are specific symptoms, which characterize burning action of fluorine compounds onto the mucous tunic of a nasal partition. At first, there are white spots, and then superficial and later deep ulcerations with further perforations of a nasal partition, and sometimes with significant area. Burning action is possessed by sulfuric anhydride.

Together with the affection of mucous tunic of upper respiratory tracts, changes are possible in the front part of the eyeball and on the eyelid skin (blepharitis, conjunctivitis and keratitis). In severe cases, it is possible to have necrobiotic changes in the form of superficial ulcers, which heal with difficulty. at direct permeating of sulfuric anhydride, hydrofluoric acid and its salts, as well as salts of phosphoric acid (in high concentrations). The development of severe ulcerous keratoconjunctivitis is possible.

A pathological process can expand onto the bronchopulmonary apparatus. With the development of chronic bronchitis, toxic bronchiolitis, often with asthmatic component, peribronchitis, and pulmonary fibrosis, there is respiratory lack of mostly obstructing or mixed type. Also, it is necessary to remember of not only about irritating and burning action of toxic substances onto the mucous tunic of respiratory tracts, but also fibrogenic properties of dust, as well as anhydrous hydrogen fluoride, sulfuric anhydride and nitric oxide. Besides local irritation and fibrogenic impact of phosphoric mineral fertilizers can manifest general toxic action, which first of all refers to phtoric compounds which are included to its content. Its manifestation can be vegetative and vascular dysfunctions with prevailing of tonus of parasymptomatic nervous system, and asthenic state of various degrees with the development of vegetative and vascular paroxysm can develop.

Being an active protoplasmic poison, compounds of fluorine and phosphorus cause significant unfavorable impact onto the parenchyma of a liver. In particular cases, clinic pattern of toxic hepatitis can develop. The

pathology of the liver is often accompanied with affection of the function of stomach with clinical symptoms of chronic gastritis. There are some data about the unfavorable action of the mentioned chemical compounds onto kidneys and endocrine glands. People with long period of work often have tendency to anemia, moderate leuko- and thrombocytopenia.

An irritating action of acid salts of superphosphate, especially if they get into cracks and ulcers on its surface, takes place. Neutral salts do not have impact like that.

It is characteristic for Thomas Slake to have both irritating and allergenic properties, what might cause the development of professional dermatitis or eczema. Impact of thermal fluorine into cracks and affected surface of skin can prolong the term of epitalization of epidermis.

Fluorine accumulates in bones, cartilage, and teeth, what causes slowing down of the growth of bones, development of hard movement of spinal cord, depression and fragility of bones. These pathological symptoms are conditioned by the disorder of calcium exchange.

Mostly cortical bone suffers from it, in particular forearm and shin, as well as bones of pelvis, spinal cord, collar-bone, shoulder-blade and cranium. Radiologically, changes in bones can be found, which are met mostly among people who have long period of work involved in superphosphate production and are characterized by thickening of periosteum and endosteum with the further development of theorsclerosis.

In the result of sclerotic and proliferate processes, lumen of channels of bone marrow can be narrowed. At marked forms of intoxications, phenomena of osteosclerosis progresses; liming, hardening of tendons and thickening of places of attaching to bony prominence take place up to the development of generalized osteosclerosis.

Cardinal manifestation of chronic intoxications of phosphoric mineral fertilizers should be considered by the affection of respiratory organs (upper respiratory tracts and bronchopulmonary apparatus), as well as changes of bone tissue. It is necessary to consider the presence of such syndromes, as vegetative-vascular dysfunction, affection of the system of alimentary tract and toxic hepatitis. In severe cases, neuroendocrine dysfunction can be noted.

Treatment. In case of acute poisoning, it is recommended to make thermomoist soda inhalations, in-take of codeine, Dimedrol, calcium preparations, and expectorants. In more severe cases, it is possible to administer calcium chloride intravenously, cardiovascular preparations, oxygen and antibiotics.

Treatment of chronic poisons is syndromic. The following is administered: cocarboxylase, vitamins (ascorbic acid, B_1, and B_{12}), biogenic stimulators, for example, aloes, coniferous and carbon bisulfide bathes.

Verification of the ability to work. At the initial stage of intoxication, it is recommended to provide a worker with a temporary employment with occupational sick leave. In case of the presence polyneuritis, bone fluorosis, marked affection of locomotor system (marked pain syndrome and the affection of function) further work in contact with toxic substances is prohibited (transfer to the invalidism on occupational disease).

Preventive measures. Mechanization and automation of main production processes, hermetization of apparatuses, effective ventilation and utilization of individual protective means for respiratory organs and skin should be done.

It is necessary to keep to personal hygiene measures after the termination of sanation of the mouth cavity (inhalation with 1-2 % solution of sodium bicarbonate). Feed with meals rich in calcium.

Conduct of preliminary and periodical medical examinations.

DISEASES IN THE RESULT OF ACTION OF NITROGEN FERTILIZER

This group of mineral fertilizers includes much spread kinds of niter: natrium, potassium, ammonium and calcic; anhydrous ammonia or in the form of ammonia water), and fusion cake (water solution of ammonium nitrate and urea).

In this group of substances, the biggest threat is presented by ammonia, which is used in the form of mineral fertilizer in the purest form, as well as in the form of the primary product for the synthesis of urea, ammonium nitrate and ammonia sulfate.

Pathogenesis. At the action of ammonia on the tissues of respiratory tracts, inflammatory reaction develops (hyperemia, transudation, and hypersection of mucus), in case of a longer impact – changes of necrobiotic character. At a definite stage of the pathological processes there is a place for infection joining (it is assisted by the affection of the tissue of respiratory tracts with toxic substances and the decrease of the general immune resistance of the organism due to intoxication).

Significant meaning is provided also by the reflecting disorder, as the irritation of receptors of the mucous tunic of respiratory tracts causes spasms of muscle tissue of the trachea, bronchi, gullet, and later reflector changes in the activity of the heart, respiratory and a vasomotor center.

Mechanism of the development of the most severe manifestation of the impact of ammonia onto the organism of a worker (it is possible only at the action of the latter in higher concentrations and at a longer exposition) is much conditioned by the increase of permeability of alveolar-capillar membrane in the result of the affection of toxic substance onto protein

structures of pulmonary tissues with accumulation at these biologically active substances.

Clinics. A c u t e p o i s o n i n g. Giving the estimation of the danger of the development of acute intoxications at the action of ammonia onto workers, it is necessary to pay attention to the narrowness of the zone of its toxic action, irritation of mucous tunics can be observed at the concentration of 100 mg/m^3, dangerous for the life of the disorder – at 350 – 700 mg/m^3, (MPC - maximum permissible concentration of ammonia in air of industrial premises should be 20 mg/m^3). Clinical manifestation of acute poisoning with ammonia is determined by local burning and general resorptive action. Ammonia, dissolving in the water of mucous tunic of respiratory tracts, front part of the eye, digestion, forms alkali solution, which causes burning of mucous tunic with further development of liquefactive necrosis. On permeating to the blood, ammonia interacts with hemoglobin with the formation of methemoglobin.

When inhaling ammonia, acute rhinopharyngitis, rhinopharyngolaryngitis, and sometimes – tracheobronchitis. Patients complain to have a feeling of stuffed nose, pain in throat, coarse voice, and sharp pain in eyes (mild degree).

At the intoxication of mean severity, there are feeling of asphyxia, coughing with bloody sputum, rejection of necrotic areas of the mucous tunic of respiratory tracts, headache, dizziness, lacrimation and salvation, pain in eyes, blepharospasm, irritation of conjunctive, nausea, fits of vomiting, diarrhea, pain in supergastric area, and swelling of gullet with aphonia.

At direct permeating of ammonia into eyes, there is a possibility for the development of keratitis with perforation of cornea and inflammation of lens.

Permeation of ammonia onto the skin causes dermatitis, clinical pattern of which is characteristic for a chemical burning.

In severe cases of poisoning, there are burnings of mucous tunic of respiratory tracts, digestion tract, which can cause the development of pain shock, toxic swelling of lungs, and methemoglobinemia. Patients are agitated, cyanotic; scleras are subecteric; liver increases, and urination delay.

The development of toxic swelling of lungs, affection of respiration and hemodynamics at severe forms of intoxication can cause a lethal outcome, even during the first day. In further terms, it is possible to have the development of aspiration of pneumonia, mass hemorrhages in the result of rejection of necrotizing tissues in upper respiratory tracts and bronchi.

In certain cases, one to two inhaling of ammonia in high concentrations can cause reflector spasms (child-crowing), swelling of glottis and asphyxia.

C h r o n i c i n t o x i c a t i o n. Clinical pattern of poisoning with ammonia is characterized mostly by the affection of respiration in the form of catarrh of upper respiration tracts, and more rarely – tracheitis and chronic bronchitis. Chronic conjunctivitis can be noticed. There is data that shows the possibility if the development of dyspeptic disorders and moderate anemia. The function of the external breathing is disturbed by obstructive or mixed type. In case of prolong action of ammonia in concentrations, which insignificantly exceed the permitted ones, there can be complaints to have reduction of the ability to work, headache, increased irritation, bad sleep, and worsening of appetite. These complaints are accompanied by significant affection in the higher nervous activity, decrease bioelectric activity of brain, tendency to hypotensia and tachycardia. After short stimulation of homopoesis, sometimes, its inhibition and worsening of indications of non-specific resistance of organism (phagocytic activity of leukocytes and titer alexine) can be observed. The frequency of such diseases, as acute respiratory-viral infections (ARVI), angina and tonsillitis increase. There are disorders of fatty and protein exchange; the activity of glutamine-conducting transaminas; discharge of urea decreases; and the necessity in ascorbic acid grows.

People, who have long term of the contact with ammonia, often have dermatitis. In case of the action of urea, of ammonia sulfate and ammonium nitrate, dermatitis mostly localizes on the areas of the backside of a palm. Possibly, there is formation of erosion on the tips of fingers with acute inflammation around them. Marked inflammation of tendencies to formation of ulcerations, which heal for a long time, and appear when niter gets onto the cracks in skin.

Toxic properties of combined nitrogen mineral fertilizers (ammonia, ammonium nitrate and carbamide). **Calcium cyanamide** much differs from other nitrogen mineral fertilizers as to the character of the action onto the organism of a person. It has marked generally resorptive toxicity.

A c u t e i n t o x i c a t i o n often appears on the background of intake of alcohol (even in small amounts). In this case, general sickness, fever in head, shivering, cooling of extremities, tachypnea, tachycardia, and arterial hypotensia can be observed. Specific manifestation of acute intoxication with calcium cyanamide is in acute hyperemia of mucous tunic of eyes, gums and soft palate, and connected with marked hyperemia of face, neck and upper portion of the torso with sharply marked limit on the type of exanthema scarlatinosa. After suffered acute intoxication, there can be residual manifestations in the form of nuclear myelities or polyneuritis.

For c h r o n i c i n t o x i c a t i o n with calcium cyanamide, it is characteristic to have asthenic and dyspeptic of character; it is also possible to have sharpening of latent progressing of digestion system disease. Irritating action of the preparation on mucous tunic can be manifested in the form of system affection of respiratory tracts, frontal portion of the eyeball and mucous tunic of a mouth cavity. If the dust of calcium cyanamide gets to skin, in mild cases, there are more or less marked irritations. At prolong contact there can be eczema with elements of pustulation. Sometimes, rush covers all body, and is accompanied by strong desire to scratch it. Te color of skin gets blackish coloration. Sometimes, there are symptoms of burning action of the preparation in the form of erosion and even ulcerations in interfinger folds of hands, on the bending surfaces, in sectors of axilla, corners of mouth, and on wing of nose. The action of the preparation is increased in case of its permeation onto the damaged and damp skin.

Treatment. In case of permeating of ammonia into eyes, it is necessary to wash with water right away. Then, liquid paraffin or olive oil, Novocain with adrenaline, and sodium albucid. At the affection of skin, it should be washed with water, to apply lotion of 5 % solution of acetic acid or citric acid. It is also recommended to make inhalations of 19 % menthol in chloroform, codeine, dionine and oxygen.

The development of spasms of glottis is an indication to prescribe inhalations, atropine and tracheotomy.

Treatment of patients with swelling of lungs due to poisoning envisages a complex of urgent measures and medical correction of the main syndrome (disease of upper respiratory tracts, chronic bronchitis and chronic pneumonia).

Verification of the ability to work. Depending on the severity of the suffered acute intoxication, it is recommended to stay in hospital for 3 to 5 days and up to 1.5 months. Further, after intoxication of mild stage, it is recommended to have temporary employment at another work with the provision of a sick leave for the term of up to two moths. After the suffered intoxication of mean or severe stage, it is necessary to have full-time rational employment.

At initial manifestations of chronic intoxication after the conducted treatment, it is recommended to have temporary (up to two months) transfer to work beyond the contact with nitrogen mineral fertilizers. The presence of chronic bronchitis, emphysema, pneumosclerosis, and in particular respiratory and cardio-vascular decompensation is the basis to employ at work, which is not connected with the action of toxic substances, hard work load and the impact of unfavorable weather factors.

The ground to determine groups of invalidism is the decrease of the qualification when rationally employing.

Preventive measures. Preventive measures involve sealing-in of machines, utilization of gas-masks, effective ventilation, control over the concentration of toxic substances in the air of industrial premises, as well as conduct of preliminary and periodic medical examinations.

Chapter 6

OCCUPATIONAL DISEASES, WHICH WORKERS HAVE AT PRODUCTION AND PROCESSING OF POLYMERIC MATERIALS

Polymeric materials are highly molecular compounds, which is widely used in national economy, Macromolecular polymeric materials consists of a high number of chains, which are repeated many times.

There are **natural** polymeric materials (biopolymers, e.g. animal and plant proteins, carbohydrate, natural cauchouc, etc) and **synthetic** polymers (those, which are made artificially), which are obtained from low molecular substances (monomers) with the help of reactions of polymerization or polycondensation (for example, polyethylene, kapron and polypropylene). Main polymeric materials are plastic masses, synthetic resins, rubber, lacquers, paints and glue.

Polymeric materials, in particular, from artificial polymers, can be used for replacement or renovation of functions of tissues and organs, production of dressings for wounds and burnings, blood replacement, packages for medicaments, sewing materials, subjects to take care of patients, details for medical equipment. Nowadays, polymeric materials turned into a permanent factor of the environment of the life of a human being, as it is used in food industry, water supply, construction, when making furniture and many consumer goods.

Together with useful properties of polymeric properties, their hazardous action takes place. First of all, it is determined by the peculiarities of chemical structure of polymeric materials, as well as technologies of obtaining, which can cause hazardous situation for people. In case of permeation of polymeric materials into the organism, it contacts with tissues, blood, lymph, and exudation. There are two mutually connected processes: destruction of polymeric under the impact of substances, which have biological activity (tiredness of polymeric metals) and interaction of decay polymeric products with biological environment, i.e. impact of products of their metabolism onto the life activity of the organism. Schematically, interaction of polymeric materials with the organism has the following look:

Polymeric materials			
Biologically compatible		**Biologically incompatible**	
Physiologically active		**Physiologically non-active**	
Which destroy slowly	**Which destroy fast**	**Which destroy slowly**	**Which destroy fast**

Transformation of polymeric materials in the organism. In case of contact of polymeric materials with the organism, they do not only

discharge not only low-polymeric own decay products, but also residual monomers and additives (plastificators, stabilizers, dyes, stuffing, and emulsifiers), which are contained in them. Primary biochemical reaction of the organism onto polymeric materials are characterized by the decrease of partial pressure of oxygen in tissues, decrease of pH in them, concentration of ions of potassium, sodium, calcium and magnesium; accumulation of physiologically active substances, and increase of the permeability into zones of damage. However, it is necessary to remember, that the interaction of polymeric materials with the organism is complicated with some factors. Thus, in case of implantation of any polymeric material, there is a multi-stage process of migration of low-polymeric matters up to the limit of the division with biological environment. The speed of diffusion in the very polymeric material is determined by the chemical structure, composition, and technological conditions of its processing. Some polymeric materials have additives, which are discharged through them and are metabolized in the organism. It has been stated that in low-polymeric substances of a similar structure, there is metabolic transformation of an additive (oxidation, renovation and hydrolysis) with further conjugation. For instance, from phenol-formaldehyde resins, such toxic matters can discharge as phenol and formaldehyde, and from urea-formaldehyde plastic – formaldehyde; from polysterene and polyacrylate – styrene and acrylate; and from polyvinylchloride - softening agents and monomer vinyl chloride.

Polymeric materials produce and process into products at the enterprises of machine-, avia- and ship engineering, chemical, radioelectronic, light and other spheres of industry. In case of obtaining and processing of polymeric materials, workers are impacted by factors, among which chemical substances are main. Careless labor conditions can be determined by physical and chemical, as well as toxic properties of raw materials, and peculiarities of the technological process. In the production process, various chemical matters can be identified – styrene, phenol, formaldehyde, vinyl chloride, residual monomers, additives and products of thermal destruction of polymeric materials, which can cause the development of acute and chronic poisons, skin diseases, as well as such diseases as polyneuritis and bronchial asthma. At some enterprises, contamination of air with dust of polymeric matters, stuffing, stabilizers and other additives.

OCCUPATIONAL PATHOLOGY AMONG WORKERS AT THE PRODUCTION OF SYNTHETIC RESINS

Synthetic resins are high-molecular compounds, which are obtained in the result of interaction of monomers. They serve the basis to obtain plastics, chemical fibers, synthetic glues, lacquers and waterproofing

compounds. Synthetic resins are widely used in the production of medical polymers, and construction materials, in radioelectronics, machine engineering, in light, and chemical industries and the production of furniture.

Depending on the method of obtaining of resins, it can be divided into **polymerization** (polyacryl, polyvinylchloride and polysterene) and **polycondensation** (phenol-formaldehyde, polyester and epoxy ones).

Main types of equipment to produce synthetic resins are reacting agents, centrifuges, and mixing agents. Insufficiently improved technological equipment can be a source of the discharge of dust, gases, vapors and aerosols of outcome monomers, mean and assisting chemical substances and final industrial products. Discharge of hazardous maters into the air of the production zone takes place also due to the absence of special devices for technological sampling and in the result of irrational pump constructions.

In the process of production and utilization of synthetic resins with occupational hazards are outcome products (styrene and vinyl chloride), substances which assist to hardening (hexamethylendiamine and polyethylenepolyamine), softening agents (dibutyl phthalate), solvents (toluene, and chlorinated hydrocarbon). Occupational hazardous factors at the production at the production of synthetic resins also include static electrics, high temperature of air, radiation of heat and noise. Some operations are connected with significant physical burden and nervous and emotional tension. Many operations on the processing of synthetic resins are done in uncomfortable position, with the tension of sign analyzer.

Among polymerization of plastic masses, which are widely spread, there are polyvinylchloride plastic, polyethylene and polystyrene.

Polyvinylchloride plastics are various chemical compounds, obtained based on polyvinylchloride. At production of polyvinylchloride and plastic with it as basis are most hazardous when such chemical substances: monomer vinylchloride; stabilizers, especially lead compounds (yellow lead, silicate and lead carbonate); softening agents (dibutyl phthalate and tricresyl phthalate); solvents, which are used in the production of perchlorovinyl (chlorobenzol and tetrachloroethane). The most spread poisoning among workers is poisoning with **vinyl chloride**.

Mechanism of intoxication is connected with the affection of deep structure of brain and first of all of reticular formation.

A c u t e p o i s o n i n g can be characterized by the development of narcotic state. Patients complain to have dizziness, nausea, loss of orientation, and disorder of coordination of movements. If the action of etiological indicator is terminated and patients are taken outside in fresh air, manifestation of intoxication with vinyl chloride disappears quickly. However, it is necessary to remember that massive and prolong action of a

toxic substance can cause loss of consciousness and even death in the result of termination of respiration. Provided medical assistance, patients get out of state of coma, however, headache, dizziness and general weakness can last for several days.

C h r o n i c p o i s o n i n g. In early stage of chronic poisoning with polyvinyl chloride, vegetative-vascular, neutrophic and sensor disorders of distal sectors of extremities can be observed on the type of Reino, as well as vegetative polyneuritis, scleroderma – like complex of symptoms with phenomena of acrosclerosis. For later stages, it is characteristic to have the development of encephalopathy with mostly affection of column structures. Vegetative-vascular dystonia is joined by the affection in distal sections of extremities with phenomena of osteoporosis and osteosclerosis. Dissolving of bone tissue in distal phalanxes of extremities (acroosteolysis) can be observed in expressed cases, on the background of scleraderma-like changes with the loss of elasticity of skin thickening of fingers, nuclear narrowing of various sizes on the palm surface of a forearm. All these often develop on the background of cerebral changes in the form of pseudoneurotic syndrome (emotional instability, drowsiness and weakness). At chronic poisoning, there can be affection of internal organs in the form of the disorder of secretor and motor function of digestion tract, increase of a liver with insignificant disorder of the function of small pain at palpation, signs of dystrophy of myocardium. There is data on the possibility of changes of blood (reticulo-, erythro- and leukocitosis). Cases of malignant formations have been found in workers, which were in contact with high concentrations of vinyl chloride at the production site.

In the process of production of items from vinyl chloride, air at the production site can be contaminated with the dust of polyvinyl chloride and stabilizers in case of thermal processing of mixtures with carbon oxide, hydrogen chloride, and hydrocarbon. Impact of significant concentrations of dust can cause the development of chronic pneumonia and insignificant fibrosis of lungs.

Products of thermal oxidization destruction of polyvinyl chloride are of much danger to people. Poisons, which appeared in the result of burning of poly vinyl chloride film, are described. Clinical patterns of these states prove to show acute intoxication of substances of irritating and burning action. There can be burnings of mucous tunic of upper respiratory tracts, covering with ulcerations, toxic swelling of lungs, and acute toxic bronchiolitis.

Polyethylene is a product of polymerization of ethylene. It is used to make pipes, parts of machines and films. Polyethylene of high pressure is used most often. And ethylene is a damaging substance in it. Besides it, in the air of the production site there is the following: solvents (benzene and

methanol), catalysts (triethylaluminum, molybdenum oxide, vanadium, chromium – IV), polymer dust and soot.

Particular dangerous are products of thermal oxidizing destruction, which cause irritating and general toxic action: can cause acute chronic affections.

Polystyrene is a product of sterene polymerization, vapors of which contaminate the air of production facilities on all the stages of the technological process. Polystyrene is widely used in various industries of national economy (for thermal isolation of water pipes, refrigerators and railroad carriages). As to the character of toxic action, sterene is closer to benzol and toluene; however it has some peculiarities, in particular a more marked irritating action. MPC of sterene in the air of production sites is 5 mg/m^3.

A c u t e p o i s o n i n g with sterene can be met in case of accidents and is characterized by mostly symptoms of narcotic action, as well as light irritation of mucous tunic of upper respiratory ways and eyes.

Mostly, c h r o n i c p o i s o n i n g with polystyrene can be met in case of action of sterene vapors in concentrations, which exceed BPL. From clinical symptoms, which can be observed then, it is possible to determine the following ones: asthenovegetative, dyspeptic and encephalopathy. At asthenovegetative syndrome, there are functional changes in a cardiovascular system, arterial hypotensia; as to dyspeptic disorder, there is dyskinesia of digestion tract, disorder of stomach secretion and functional changes in liver, and in future, under condition of the continuation of the act of polystyrene of higher concentrations, and they transform into toxic hepatitis. With the action of sterene, the possibility of the development of endocrine disorders can be observed (mild manifestations of hyperthyroidism and disorder of functions of ovary, disorder of menstrual cycle, and hyper menstrual syndrome). Changes on the side of blood are unstable and can easily be reversing (moderate leucopenia, inclination to anemia and thrombocytopenia).

Polymerization of plastic masses also includes fluorocarbon polymer and polyacrylate.

Fluorocarbon polymer is products of polymerization of ethylene derivatives of fluorine (with hexalfuorine propylene). The widest usage has the following types of polyfluoroethylenes: polytetrafluorinethylene, polytrilfuorinechlorethylene. Polyfluoroethylene reminds paraffin. They are used as isolation materials for wires, cables, transformers, as well as for production of chemical machines.

Thermal processing of items from fluorocarbon polymers is done at very high temperatures, which are close to the temperature of thermal destruction of these polymers (360 – 380 °C). Even small increase of the so-called work temperature much increases the output of polymers of

volatile toxic products with the creation of aerosol of secondary polymerization of fluorocarbon polymers.

Pathogenesis. It is considered that portions of fluorocarbon polymers are ingested with cells of alveolar epithelium and leucocytes; in the result of the death of coniophages, protein is discharged, which causes pyrogenic effect.

Clinics. Workers, engaged in the production of items of fluorocarbon polymer, inhaling of products of thermal oxidizing destruction of polymer causes acute occupational disease, which received the name of fluorocarbon polymer fever. Signs of the disease appear after a short hidden period (1 to 6 years): workers have scratchy throats, have fever, dry coughing, and small asphyxia. The temperature of body increases up to 39 – 40 °C. Leukocitosis can be observed in blood; and during auscultation, dry rales can be heard in lungs. In some time, strong sweating takes place; body temperature decreases. General state normalizes in 1 or 2 days. It assists to the appearing of fluorocarbon polymer fever when smoking at work, as they call the decomposition of fluorocarbon polymer dust, which permeated into tobacco.

Polyacrylate are polymers and copolymer of acrylic acids and their derivatives. The widest utilization in the production of plastic masses was obtained by acrylic resin – product of polymerization of methyl alcohol - methyl methacrylate. At polymerization of methyl methacrylate, organic glass can be obtained. Main dangerous fact in these processes is the dust of plastic, as well as a methyl methacrylate monomer. It causes narcotic, general toxic action, has vague irritating and allergic properties. MPC of methyl methacrylate is equal to 20 mg/m^3.

Clinics. Acute poisoning can be characterized by mainly signs of narcotic intoxication. In mild cases, there are general weakness, nausea, vomiting, headache, dizziness, and short loss of consciousness. If a patient is taken out into fresh air, his/her state normalizes fast. In cases of more marked intoxication, there can be short epileptiform fits and longer loss of consciousness. Recovery takes place in 3 to 4 days.

Chronic intoxication. In the clinical pattern, main place is taken by the disorder of the nervous and cardio-vascular system. At the initial stage, there is astheno-neurotic syndrome and signs of vegetative and vascular dystonia. Patients complain to have general weakness, increased fatigability, dizziness, pain in the heart area, and increased irritability. At objective examination, sweating, pulse lability, and arterial blood pressure are increased. Later there are symptoms of vegetative-sensitive polyneuropathy and asthenoneurotic syndrome: pain and parastesia in extremities, decrease of pain sensitivity of the type of gloves and socks, constant headache, memory disorder, acute asthenization, inclination to cerebral angiospastic reactions with the intensification of

headache, dizziness, nausea, vomiting, increased arterial pressure and the decrease of the sensitivity of hearing analyzer.

Patients with chronic poisoning with methyl methacrylate can complain to have dyspeptic phenomena, pain and feelings of heaviness in the right hypochondrium and in supergastric area, disorders of liver functions, and decrease of acid-forming functions of a stomach, changes in peripheral blood in the form of anemia, as well as leucopoenia with lymphopenia. Accumulation of methanol can be used as a diagnostic criterion (one of metabolites of methyl methacrylate).

Among polycondensed polymer and plastic masses, one of the most spread ones is phenol-formaldehyde resins (phenoplasts). They are products of polycondensation of phenols with aldehydes. Industrial importance is possessed by resins, obtained from phenol and phormaldehyde. In the national economy, such phenoplasts as textolite, turbonit, and faolite are rather widely used. Phenol-formaldehyde resins are used when producing veneer, various materials based on wood, abrasive wheel and as a base to make glues, lacquers and enamels. Main dangerous substances when receiving phenol-formaldehyde resins are gases and vapors of phenol and formaldehyde.

According to the character of the toxic action onto the organism, phenol belongs to nervous poisons; however, it also can cause irritating and burning action. In the clinical pattern of acute poisoning, there are symptoms of general action of poison and affection of the nervous system: weakness, headache, dizziness, as well as noise in ears, fits, and loss of consciousness. Death can come in case of severe poisoning (massive permeation of phenol) in the result of the paralysis of respiratory and vasomotor centers.

Formaldehyde has marked irritating action, and is also a general toxic poison, which impacts the central nervous system. There is data, which proves embryotoxic, gonadotropic and mutagenic action of formaldehyde.

At acute poisoning with formaldehyde, patients get burning in eyes, epiphora, symptoms of acute affection of upper respiratory tracts, and bronchitis. There is general weakness, headache, dizziness and unsteady walking. At the action of very high concentrations of gas, necrosis of the mucous tunic of the upper respiratory tracts can be observed.

Occupational affections of skin, in particular at the presence of direct contact of skin with synthetic resins and polymer materials can appear. Action of high temperature and gas-like products assists to the appearing of skin diseases. Here, patients complain to have skin rush and itch. Manifestation of dermatitis or eczema is localized mainly in open areas of skin – on face, neck and arms. People, who have a prolong contact with

phenoplasts, undergo thickening of cornea, change of sweating and desensibilizing of organism.

Particular attention should be paid to the affection of respiratory system among workers, who work in contact with phenoplasts. Patients complain to have dry rales, dryness and pain in a nose, gullet, stable running nose, nosebleed, coarse voice and dyspnea. Workers, who have been working for a long time in the production, mostly have subatrohpic and atrophic changes of mucous tunic of the nose, gullet and larynx; in some cases there are chronic bronchitis, with bronchiospastic component, and in case of the development of sensibilization – and bronchial asthma.

Those who work in the production of press powders might have a slightly marked black-lung disease of the interstitial form, which is characterized by non-malignant progressing.

Before manifestation, impacts of phenol-formaldehyde resins onto the organism of workers can also include the affection of the central nervous system (astheno-vegetative syndrome with vascular reactions), dyspeptic phenomena, functional stomach diseases, gastritis and liver functions disorder.

In various sectors of industry, epoxy resins are widely used, certainly only in various combinations with substances, which cause their hardening, with the so-called compounds – hardeners, plastifiers and stuffs. Based on epoxy resins, plastic masses can be obtained. When utilizing epoxy resins, the main production hazardous factors are resins and vapors of volatile substances, which are contained in them, as well as vapors of hardeners, plastifiers and solvents. MPC of volatile substances for various types of epoxy resins are within 0.2 – 1 mg/m^3.

Outcome products and hardeners of epoxy resins have mostly irritating action onto the skin, mucous tunic of respiratory tracts and eyes. However, some of them cause toxic impact onto the nervous and cardiovascular system, affection of parenchymatous organs, inhibit haemopoiesis and cause sensitization.

Clinics. Workers who work with epoxy resins most often have occupational diseases of skin – contact and allergic dermatitis and eczema. They develop dryness of skin, its reddening, itching, and later swelling, peeling, spotty and fine – nodular rashes. Epoxy resins manifest their sensitizing properties, when they get onto not only skin, but also by inhalation and other ways. At this, there are signs of the affection of the respiratory, urine, nervous and bile-extracting systems. There are conjunctivitis, hypertrophic, subatrophic or atrophic rhinitis, faringitis, bronchitis with asthmatic component, bronchial asthma, hepatitis, hypochromic anemia and arthralgia. At general resorptive action, there are signs of affection of the nervous system, which are characterized by the syndrome of vegetative and vascular dystonia. Patients complain to have

headaches, dizziness, increased sweating, and pain in some areas of heart. At the objective examination there is inequality of palpebral fissures, anisocoria, increased tendon reflexes, hyperhidrosis, cyanotic-marble coloration of skin of hands and feet, and red spread dermographism. In case of inhalation permeation of products of thermal destruction of epoxy resins, lung swelling can develop.

OCCUPATIONAL PATHOLOGY AMONG PATIENTS AT THE PRODUCTION OF SYNTHETIC RUBBER AND RUBBER PRODUCTS

Synthetic rubbers are high-polymeric compounds, which are capable to reverse deformations. They are utilized mostly to produce rubbers and rubber items; they are used in production of medical devices (tubes for blood transfusion, catheters and gloves), as well as for sanitary and hygienic measures (hot-water bottles, bottles for ice and nipples). Also, silicon organic rubber is used, for example, dimerhyl xyloxane, properties of which satisfy many medical requirements. It can be used to produce prosthetic devices for various parts of organism, for example, heart valve, mammary gland, as well as an implant for a nose, and finger phalanxes.

Rubbers can be divided into natural and synthetic. Synthetic ones can be divided depending on the dependence of the sphere of their utilization: rubbers of general designation, which are used in mass production of items where the main property of a rubber is used – elasticity; rubbers of specific designation, used for production, should be not only elastic, but also resistant to the acts of various solving agents, acids and alkali.

It is also possible to utilize a classification of synthetic rubbers depending on the chemical composition. The following sorts of rubbers are most widely used, like butadiene, butadiene-styrene rubbers, polyisoprene and copolymer of ethylene with polypropilene.

The main occupational hazard when obtaining any synthetic rubber is the contamination of the air of shops and gases of chemical substances, which participate in the synthesis of polymer (monomers, emulsifier and catalyzes), among which the most hygienic place is taken by monomers. At a number of production lines, there is noise, presence of unfavorable microclimatic conditions.

Butadiene sodium synthetic rubber is received in the result of polymerization of butadiene in the presence of sodium. Butadiene is a gas, which causes irritation of mucous tunic of upper respiratory tracts and eyes. In high concentrations, it acts as a narcotic substance. BPL in the air of the work zone should be 100 mg/m^3.

A c u t e p o i s o n i n g with butadiene is manifested through pain in eyes and throat, sweet taste in mouth, headache, dizziness, noise in ears, general weakness and sometimes the feeling of alcohol intoxication,

paleness of skin coverlets, tachycardia, and dyspnea; it is possible to have vomiting, loss of consciousness and narcotic state. These disorders are not stable and in case of immediate evacuation of a sufferer into the fresh air, they disappear fast.

For c h r o n i c p o i s o n i n g, it is characteristic to have the affection of the nervous system in the form of neurasthenic syndrome (headache, dizziness, general weakness, apathy, insomnia, increased irascibility, and weakening of memory); cardiovascular (palpitation, "congestion" into a head and arterial hypotension) and gastro-intestinal systems (nausea, burning, heart burn and pain in a stomach). There can be irritation of mucous tunic of mucous tunic of eyes, affection of skin (dermatitis) and decrease of the number of leukocytes in the peripheral; blood.

Butadiene nitrile rubber is a product of copolymerization of butadiene and nitrile of acrylic acid. Nitrile acrylic acid is a colorless liquid with weak smell. Incase of getting onto the skin, it can cause chemical burning and resorptive toxic action.

Chronic intoxication can be characterized by mostly affection on the side of the nervous system (asthenic syndrome), vegetative dysfunction and arterial hypotension. There are signs of dystrophy of myocardium, moderate manifestations of toxic hepatitis, dyskinesia of a gastric tract, anemia, leuko and neutropenia. It is also possible to have allergic diseases of the skin and respiratory system.

Polychloroprene rubber (Nairit) is a product of polymerization of chloropene. It has narcotic, general toxic action, as well as irritating, allergenic, cytotoxic and mutagenic properties. BPL in calculation into chloropene is 2 mg/m^3.

A c u t e p o i s o n i n g. In case of mild acute poisoning, there is a headache, dizziness, nausea and epiphora; in more marked cases, loss of consciousness, weak frequent pulse, dullness of heart tones and repeated vomiting.

C h r o n i c i n t o x i c a t i o n is manifested through a change of the side of nervous, cardio-vascular system and liver. Changes on the side of the nervous system are characterized by the marked asthenic state, vegetative dysfunction with the disorder of a cardiovascular regulation; there is vascular hypotension, and tendencies to bradycardia; in later stages, there is encephalopathy with epileptic forming fits; and often there is toxic hepatitis.

For chronic poisoning, it is characteristic to have trophic disorders: hemorrhagic diathesis and expansion of gums, fragility and laying of nails, and hair falling. In case of a local action of the chloropene, there are dryness of skin, peeling, multiple surface cracks, and rash. There are numerous proofs of the unfavorable action of chloropene. Besides,

chloropene has blastomogenic properties (both in the place of the contact with skin, and also in internal organs).

Main consumers of synthetic rubbers are the production of rubber items. Technological process of the production of rubber items consists of two stages: preparing operation and the process of vulcanization. At the stage of preparatory work, where preparation of rubber substance takes place, workers are subjects to high concentrations of dust of mixed character as a rule. This can cause the development of bronchitis and black-lung disease. There are interstitial and nodular forms of black-lung disease (I – II stages) with non-malignant progress.

The process of vulcanization is carried out in hydraulic presses and autoclaves at the temperature of 120 – 190 °C. At this, in the air of the work zone there is a complex vapor-gas mixture of chemical substances, which contain residual monomers of rubber, products of thermal-oxidizing destruction of components of the rubber mixture. Workers can observe hypertrophic and subtrophic changes of the mucous tunic of upper respiratory tracts. There can be functional changes of the nervous system, liver, inclination to arterial hypotension, cracks and other changes, connected with the action of hot rubber.

In the production of rubber, dangerous factors are also chlorinated hydrocarbon, which are rather widely used as solvents of glues and petrol. At long contact with the latter, there is neurasthenic syndrome, and the impact of chlorinated hydrocarbon causes manifestations of gastric tract (dyspeptic complaints, decrease of stomach secretion and disorders of liver functions).

Besides, it is necessary to remember that a big number of manual operations, connected with the given tension of muscles of the upper shoulder-girdle, it can cause the development of diseases of peripheral nervous system and resistance apparatus.

OCCUPATIONAL PATHOLOGY AMONG WORKERS AT PRODUCTION WHERE SYNTHETIC FIBERS ARE OBTAINED AND PROCESSED

Synthetic fibers are produced based on polymer compounds of various classes. There are polyamide (kapron and nylon, polyester (lavsan and polyacrylonitrile), polyvinylchloride (chlorine) and some other types of synthetic fibers. The technology of obtaining of synthetic fibers consists of three main stages: synthesis of polymer, formation and textile processing of fibers. Work at this production is connected with the impact of mostly toxic factors in connection with the increased air temperature. Particularly dangerous operations are as follows: when loading and cleaning of machines and filters. When obtaining fibers from the melt of polymer in spinning room, monomers and other chemical substances can be

discharged which participated in the synthesis of polymer. An important hygienic peculiarity of chemical and spinning workshops can be contamination of hand skin of workers with monomers and solvent, which can cause skin affection, as well as absorbing of toxic matters through skin.

The most spread are the production of polyamide (kapron), polyester (lavsan) and polyacrylonitrile (nitron) synthetic fibers.

Kapron fibers are received from polycaproamide – a product of polymerization of caprolactam monomer. Air environment in chemical and spinning shows when obtaining kapron fiber is contaminated with vapors and aerosol of caprolactam and vapors of dinyl thermofor, which is a mixture of biphenyl and biphenyl ether. BPL for each of them is equal to 10 mg/m^3. Caprolactam is a nervous poison, which causes fits, as well as affection of parenchymatous organs. Dinyl vapors cause toxic action onto the central nervous system, parenchymatous organs and cause irritation of mucous tunic of upper respiratory tracts and eyes. In the result of combined action of dinyl vapors, vapors and aerosol of caprolactam can cause the development of subatrophic and atrophic changes from the side of the mucous tunic of a nose and a gullet, functional disorders of the nervous system (vegetative-vascular dystonia, and vegetative-sensatory neuropathy), and toxic affection of a liver. Besides, there can be a disorder of menstrual functions, pathology of pregnancy and labors among women who work at this production.

Polyester fibers (lavsan) is received from polymer polyethylene terephthalate – a product of polycondensation of dimethyl ether of terephthalic acid and glycol. As heat carriers, dinyl is used.

Dimethyl terephthalate is a colorless solid substance. It is powder with irritating and general resorptive action. BPL in the air of the work zone for each of these substances (dimethyl terephthalate and terephthalic acid) is equal to 0.1 mg/m^3.

At prolong period of work at the production of lavsan, changes of upper respiratory tracts are possible (sub- and atrophic rhinitis and laryngotracheopharyngitis), nervous systems in the form of functional disorders, cardiovascular system (vegetative-vascular dysfunction with the tendency to arterial hypotension), disorder of the functional state of the liver, some changes in peripheral blood (decrease of the number of erythrocytes, leukocytes and the content of hemoglobin).

Polyacrylonitrile fivers (Nitron) are synthetic fibers, which are obtained from polyacrylonitriles or copolymer of nitril of acrylic acid. Conditions of work can be characterized by the contamination of air environment with acrylate vapors.

Clinic. A c u t e p o i s o n i n g. They are manifested through disorders of digestion tract (nausea, vomiting, and pain in stomach),

functional disorders of the nervous system, cardio-vascular system and liver.

C h r o n i c p o i s o n i n g with acrylate is manifested through functional disorders of various systems of the organism. Mostly, there are disorders of the nervous system – vegetative-vascular dystonia and asthenovegetative syndrome. Workers, who have worked for a long time at the mentioned enterprises, have micro-organic symptoms (nystagmus, decrease of smelling, gullet reflex, and anisocoria) and signs of hypothalamic pathology. These changes of the central and vegetative nervous system is joined with the functional disorder of the cardio-vascular system (arterial hypotension and sinus bradycardia), and sometimes there are signs of myocardosis, which can be found on the electric cardiogram. Often, there are cases of dyskinesia of a digestion tract, decrease of a secretor function of a stomach and a chronic gastritis, as well as disorders of renal functions, and in some cases, clinical signs of hepatitis. There are also changes in peripheral blood in the form of anemia, leucopenia and relative lymphocitosis, disorder of porphyrin exchange, as well as earlier and more frequent manifestation of pathological climax among workers of this production.

Among workers involved in the production of nitron, also sensibilization to acrylates with the development of allergic diseases of respiratory organs and skin can be observed.

When producing synthetic fibers, workers contact also with dimethylformamide, which is used as a solvent. This matter has narcotic, general toxic and irritating action. It can permeate into the organism through inhalation and through undamaged skin.

During textile processing of various types of fibers, one of the most unfavorable factors of a productive environment there is noise and stress on vision organs. Among workers, which have great work period under these conditions, and also such pathology as occupational partial deafness and vision disorder can be observed. Besides, at enterprises where synthetic fibers are produces, workers are impacted by the following such unfavorable factors of the production environment, as dust of polymer materials, which is capable to cause weak toxic and fibrogenous impact, and dust of kapron and lavsan has also allergic action. Processes, connected with thermal processing of various types of synthetic fibers, are accompanied by the impact onto works of ammonia, formaldehyde, and ethylene oxide, that in case of prolong term of work, it can cause functional disorders in the nervous system (asthenovegtative syndrome), skin diseases (dermatitis and eczema) and conjunctivitis.

PRINCIPLES OF DIAGNOSTICS, TREATMENT, VERIFICATION OF THE ABILITY OF WORK AND PREVENTION OF OCCUPATIONAL DISEASES, WHICH APPEAR UNDER CONDITIONS OF PRODUCTION OF POLYMER MATERIALS

When conducting diagnostics of a c u t e o c c u p a t i o n a l p o i s o n i n g under conditions of production of polymer materials, it is necessary to consider peculiarities of clinical symptoms and poison progressing, as well as to reveal group character of poisons with similar clinical pattern among those who work under the same conditions. Most often, a patient comes across the manifestation of narcotic action of some type of polymer, clinical signs of which are as follows: euphoria state, headache, dizziness, unstable walking, nausea and vomiting. In more severe cases, there can be loss of consciousness, and comatose state. It is necessary to remember about possible combination of narcotic action of monomers with weak irritating effect. It might be that in case of impact of high concentrations of some monomers (formaldehyde and epichlorohydrin), laryngotracheitis, bronchiospasm, toxic pneumonia and toxic swelling (perphorhizobutylene) can develop.

Clinical symptoms of acute poisoning of products of burning of polymer materials can be determined by chemical composition of the mixture, which is discharged when burning. Thus, in case of poisoning with products of PVC burning in the clinical pattern, there is mostly pathology of respiratory organs (marked burning of respiratory tracts, acute bronchiolitis, toxic swelling of lungs). In case of the action of burning product of foam-rubber, clinical complex of symptoms an be observed, which includes the affection of the central nervous system together with acute bronchitis, and toxic swelling of lungs. Progressing of acute poisons can also vary. Thus, in some cases (at mild poisons), manifestation of narcotic and irritating action of toxic matters pass fast (it is enough to get to the fresh air), and in other normalization of the state takes place only after a prolong stable treatment. Sometimes after this can be stable consequences after the undertaken intoxication (encephalopathy, chronic toxic hepatitis and chronic pathology of a respiratory system).

When conducting diagnostics of c h r o n i c i n t o x i c a t i o n s under production conditions of polymer materials, it is necessary to remember about the presence of specific signs of these poisons and the possibility of their combining in case of action of specific toxic matters. There are such signs as toxic angioneurosis, syndrome of Reino, scleroderma-like narrowing of skin, osteolysis of distant phalanges of fingers, vegetative neuropathy, which takes place when poisoning with vinyl chloride; marked asthenic syndrome or encephalopathy in combination with epileptiform attacks, toxic hepatitis, and trophic

disorders in the form of hair losing – when getting poisoned with chloroprene.

More often, it takes place that chronic intoxication among whose who work on production of polymer materials, progresses with little clinic symptoms; manifestations of the nervous system (vegetative-vascular dystonia, asthenovegetative syndrome, and sensitive polyneuropathy); functional disorder of liver; and moderate changes in peripheral blood. In similar cases, stating of the connection between a disease and the conditions of work should be based on determining such syndromes, which are characteristic for the action of some poisons and are pathogenically conditioned by their impact. It is necessary to remember about the presence of some period of work under this conditions, increase of BPL of some toxic matters, as well an exceptions of appearing of other etiologic indices.

Treatment. In case of acute occupational poisoning, therapy should be urgent, active and purposeful. In such cases, main principles of provision of urgent aid should include the following:

1) termination of poison permeation into the body;
2) excretion of poison and products of its transformation from the body;
3) neutralization of poison in the organism (antidote therapy);
4) renewal of affected functions, first of all those, which are important for the vital activity of a human being (heart and vessel and respiratory ones); and,
5) intensifying of protection forces of the organism (renewal and support of internal environment; water-salt, electrolytic, vitamin, and hormonal balance as well as acid-base equilibrium).

In various stages, these principles should contain specific activities. For example, in case of treatment of patients with fluoroplastic fever, it is advised to use intakes of calcium chloride and glucose with ascorbic acid (prevention of lung swelling), acetylsalicylic acid, and oxygen inhalations. cyanic-like manifestations at intoxication with nitrile of acrylic acid can be treated with inhalation of 0.5 ml of amyl nitrile, intravenous introduction of 50 ml of “Chromasmone” and 30 to 50 ml of 30 % solution of sodium thiosulfate.

Victims with mild degree of acute poisoning should be treated as outpatients, and in more sever cases of intoxication manifestation, hospitalization might be necessary.

Etiotropic treatment of chronic occupational intoxications and diseases of workers involved in production of polymer materials are absent. Various means of pathogenic and symptomatic therapy are used at specific syndromes with the consideration of their clinical form and the degree of expression.

Verification of the ability to work of workers, engaged in the production of polymer materials, at chronic occupational intoxications are conducted individually with the consideration of the character and the degree of the found disorders and their reversibility.

In case of chronic intoxication, which is manifested through functional changes of the nervous system and liver, it is recommended to have an appropriate treatment and follow-up. In more marked cases (stable functional changes on the side of the nervous system, partial deafness, encephalopathy, toxic hepatitis, and changes of blood as well) of a patient, it is necessary to temporarily dismiss a patient from work for the period of the treatment or to transfer to the full position beyond the contact of toxic substances.

Patients, where there are cases of intoxication in respiratory tracts, mild bronchitis and are subject to the supervision of a doctor and treatment.

If in the clinical pattern, there is chronic atrophic laryngitis with functional disorders and ulceration on mucous tunic of a nose, patients should be transferred to a full time position (rational employment). Stable marked chronic bronchitis, bronchial asthma is a contraindication for further work under conditions of the action of dust, toxic substances of irritating and sensibilizing action.

Patients with black-lung disease of the 1st stage (*interstitial form*) without phenomena of bronchitis and insufficiency of respiration can stay at their wok under conditions of dynamic medical observation over them. In more marked and complicated cases of black-lung disease, it is necessary to terminate any contact with dust.

Patients with manifestation of intoxication in the form of dermatitis and eczema need treatment, and in case of more marked clinical pattern – temporary conduct to another job beyond the action of an etiological factor. Rational employment is indicated at the stable or relapsing eczema.

In case of decrease of qualification in the result of conduct to another job due to the presence of stable occupational disease, patients are sent to the Expert Commission.

Preventive measures. Preventive measures for enhancement of work conditions should be aimed at standardization of prescription mixtures and raw material with the purpose to limit the content of hazardous elements, replace of highly toxic components with less toxic ones, as well as perfection of technological equipment. For struggle against dust and elimination of manual operations, it is necessary to use more widely pneumatic transport and transportation of raw materials with the help of a conveyor. Raw materials should be supplied to the enterprise in the form of granules and briquettes. It is necessary to maximally limit the contact of workers, especially open parts of body and arms with dangerous substances, as well as to use protective uniform, pastes and respirators.

Specific meaning is possessed by heating isolation of warmed up equipment and communication, as well as correct utilization and operation of ventilation equipment. Workers, who are employed, should undergo preliminary medical examination and periodical examinations in the future. A list of contraindications for hiring, as well as periodicity of medical examinations, depends on the type of production. Educating of all employees to rules of safety measures and personal hygiene when working with chemical compounds are important.

Chapter 7

OCCUPATIONAL DISEASES, CAUSED BY PHYSICAL FACTORS

Vibration disease

Vibration disease is a professional disease, caused by vibration. For the first time, this pathology was described by Loring in 1911 as a syndrome of "dead fingers" among scabblers, and in 1955, it got the name of "vibration disease".

The main factor, which causes the development of the disease, is vibration. From the physical point of view, vibration is a mechanical oscillation, which is repeated at definite periods.

There is a l o c a l vibration, which impacts mostly onto hands of a worker when working with vibroinstruments, and a g e n e r a l vibration, which impact the whole organism. Local vibration takes place when workers use pneumatic and electric instruments (chisel hammers, riveting hammers and chopping hammers). The impact of the general vibration can be observed in case if a worker works with the vibrating equipment (vibroplatform and automatic concrete distributors), as well as in case of transfer of vibration from working engines, machines and equipment to the floor.

Expression and the time for the development of the disease is determined by the zone of the amount of oscillation energy, which is transferred to the body of a human being or his/her limited part, as well as factors, which assist to the development of vibration disease: forced body position, cooling and noise.

Among occupational diseases, the vibration disease still holds a leading place and is mostly encountered among those, who work in metal processing, machine engineering, metallurgic, construction, aircraft manufacturing, mineral resource industry, agriculture, transportation and many other spheres of national economy.

In Ukraine, vibration disease appears mostly among workers of such professions as cutters, drillers, fettlers, face-workers (impact of low frequency local vibration), riveters, polishers, tool dressers (impact of high frequency local vibration), as well as drivers of heavy earth-moving machines (impact of general vibration).

Pathogenesis. In the basis of the vibration disease is a complicated mechanism of nervous and reflector disorders, which cause the development of nucleuses of stagnant agitation and to further stable changes both in receptor, as well as in various sectors of the central nervous system. A significant meaning in pathogenesis of the vibration disease is played by specific and non-specific reactions, which reflect adaptation and compensatory processes of the organism. It is considered that the vibration disease is a specific angiotrophoneurosis, when spasm of fine and bigger vessels can be observed. There are also thoughts that

an angiotrophoneurous syndrome at this disease, connected with the affection of laminated bodies (Fater – Paccini).

Recent data proves that in the pathogenesis of vessel changes at the vibration disease takes place in the following:

- disorder of mechanisms of membrane transportation of calcium with the increase of basal concentration of the latter in unstriped muscular cells of blood vessels;
- increase of the speed of both active and passive transportation of potassium;
- replace of properties of pre-membrane spectrin and actinic complex, decrease of urgent skeleton proteins of erythrocytes – spectrin and actin;
- accumulation of primary and secondary products of lipid peroxidation and inactivation of ferments of antioxidant protection.

In the genesis of trophic disorders, which develop at this disease, a significant role is played by changes in microcirculation, rheological properties of blood, disorders in obtaining and utilization of oxygen. There are also disorders in hypothalamic-pituitary-adrenal system, changes in correlation of vasoactive substances of rennin-angiotensin-aldosteronogenic system and hormones of pituitary-thyroidcompex, content of nucleotide, increase of prostaglandins in blood, affection of vitamin and microelement balance, as well as change of immune indicators. Besides that, neurohumoral and nervous-reflector disorders have phase character. They depend on the degree of the expression of vibration pathology. Thus, in initial stages of the disease, there is an increase of the functional activity of sympathyco-adrenal system due to activation of mechanisms of adaptation and overexcitation of peripheral vegetative formations. Further, in case of progressing of pathology, this state changes by the normalization of excretion of catecholamines, and then inhibition of sympathico-adrenal mechanisms correspondingly to the decrease of adaptation possibilities of the organism.

Clinics. Due to the fact that manifestation of vibration disease is many-sided and polymorphous, and until now the classification of E.Andreyeva-Galanian and V.Artamonova is still pressing; it considers its various forms, conditioned by the action of local and general vibration. Prolong study of this pathology enabled to state various variants of its progressing with mainly the manifestation of neuro-vascular disorders or pathology of a locomotor system. E.Drogichyna and N.Metlina isolated seven syndromes of the disease: angiodistonic, angiospastic, syndrome of vegetative polyneuritis, neuritis, vegetomyofascitis, vestibular, and diencephal with neurocircular disorders. Isolation of these syndromes is conditioned by the fact that the impact of additional production factors together with vibration (cooling down, microtraumatism, and physical tension) enabled to isolate some syndrome of the disease in the clinical pattern. At the same time, the progress of the disease has

changed lately, what conditioned the necessity of the consideration of this classification, taking into account the action of local vibration of various intensiveness.

The clinic of the vibration disease is complicated, and it is distinguished by its variability and is not always specific. The disease develops gradually, thus for a long time patients might not go to the doctor due to seeming improvement of their state when working. Only an active medical examination can help to make a diagnosis. Depending on the character of the work, physical parameters of the acting vibration, total amount of time of its impact, place and area of collision with the source of vibration, symptoms manifest differently and peculiarly.

The clinical symptoms of any form of the vibration disease consist of the neurovascular disorder, disorders in neuro-muscular system, locomotor system, and metabolism. The main place in the clinics is possessed by angiodistonic syndrome with phenomena of angiospasms of peripheral vessels. At the same time depending on the form if their manifestation is a localization of vascular and nervous – muscular disorders can be conditioned by the character of the action of vibration, in the range of which there are high and low frequencies. Thus, complaints of patients in the result of the action of low frequency vibration and significant “recoil” of a vibrating instrument can be numerous in the first stage already. These patients develop a vibration disease with mostly the affection of muscles, bone changes, angiodistonic manifestation, vascular hypotonia, atony of lesser vessels, pattern of polyneuritis, mostly vegetative, and a rather marked pain syndrome.

The vibration disease in the result of vibration, in the range of which high frequencies prevail, differs by the peculiarity of vascular disorders, and more marked cardiovascular syndrome.

Vibration disease in the result of local vibration

In compliance with the existing classification of this form of the vibration disease, it has three forms of severity:

I – *initial manifestations*: 1) peripheral angiodistonic syndrome of upper extremities, including rare angiospasms of fingers; 2) syndrome of sensor (vegetative-sensor) polyneuropathy of upper extremities.

II – *moderately marked manifestation*: 1) peripheral angiodistonic syndrome of upper extremities often with angiospasmic fingers; 2) syndrome of vegetative-sensor of polyneuropathy of upper extremities: a) often with angiospasms of fingers; b) with stable vegetative-trophic disorders of hands; c) with distrophic disorders of a locomotor system for upper extremities and their belt (myofibrosis, periathrosis and arthrosis); d) with cervicobrachial plexopathy; and e) with cerebral angiodistonic syndrome.

III – *marked manifestations*: 1) syndrome of sensomotor polyneuropathy of upper extremities; 2) syndrome of encephalopolyneuropathy; and 3) syndrome of polyneuropathy with generalized acroangiospasms.

Initial manifestations of the disease progress in the form of peripheral angiodistonic syndrome or the syndrome of sensor polyneuropathy hands. The disease starts slowly with passing paresthesia. Patients complain to have numbness, pricking, feeling of butterflies in the stomach, ache in distal parts of arms, increased sensitivity to cold in fingertips. Pain and paresthesia can be noted only in calm state, after work and at night. Besides, manifestations take place when cooling, and in case of the change of atmosphere pressure, when doing heavy physical work. After prolong breaks in work, unpleasant sensations in hands disappear.

Patients with vibration disease in this stage the following symptoms can be observed: cyanosis, hypothermia of hands, hyperhidrosis, sometimes, palm dryness, and mottled skin. All these show the disorder of peripheral blood circulation in palms; these symptoms are not stable. Peripheral angiodistonic syndrome can be accompanied by finger whitening at general or local cooling. Acroangiospasms develop either on both palms simultaneously, or at first on the hand, which suffers from the vibration impact. At first they usually appear during cold periods of the year at general cooling down: suddenly there might be sudden whitening of tips of one or several fingers (except the first one) and lasts for several minutes, and then are replaced with cyanosis (stage of angiohypotonia), which can be accompanied by paresthesia. If the process develops, angiospasms can be spread onto other phalanxes, and then appear on the other hand. In the initial stage of the disease, Reino syndrome takes place rarely (about 1 or 2 times a month).

The perception of vibration and pain sensitivity decreases. At initial manifestations of vibration disease, there can be hyperstesia of fingers, which is replaced with hypoesthesia. Zone of the decrease of sensitivity gradually spreads onto palms and forearms. Trophic disorders in this stage of the disease are limited by the worn out pattern of fingers and X-disease of palms.

Peripheral angiodistonic syndrome is actually a clinical manifestation of vegetative polyneuropathy of upper extremities. With intensified pain and paresthesia in distal sectors of arms, expanded zones of hypoesthesia beyond palms diagnose the syndrome of vegetative and sensor polyneuropathy of upper extremities.

Moderate marked manifestations of the disease can be characterized by more marked intensiveness of pain and paresthesia in arms and intensification of the frequency of the development of acroangiospasms. Pain and paresthesia in distal sectors of upper extremities become more stable during a day. After work and at night they are intensified; sleep is affected; patients toss in bed and rub hands at night. During vacations or treatment, this unpleasant sensitivity in hands usually decreases, however it does not pass completely.

The growth of expression of peripheral vegetative and vascular, sensor and trophic disorders takes place. In the morning, patients have swelling fingers, slow moving of fingers, which together with pain and paresthesia usually disappear or decrease soon after the work is commenced. Cold angiospasm of fingers develops after washing hands with cold water, or sometimes spontaneously. Fits of angiospasms can involve all the fingers; its duration grows up to 30 to 40 minutes. It is over with angiohypotonia with pain reaction and prolong ones (1 to 2 hours) with cyanosis of fingers. Here, the spasm of capillaries changes with their atony. Peripheral vegetative and vascular disorders at this stage do not develop independently, but are a part of the syndrome of vegetative and sensor polyneuropathy of upper extremities. Further increase of the threshold of vibration sensitivity, decrease of superficial sensitivity not only in distal, but also in proximal sectors of extremities can be observed. Sometimes, the zone of hypoesthesia expands to the chest and head.

In case of presence of stable swelling of fingers and palms, slow moving and moderately marked bending contracture of fingers, deformation of interphalanxes, limbs, X-disease of palms, changes of form and feeding of nails, a diagnosis can be made as to the syndrome of stable vegetative and trophic disorders. Here, nail plates can be in the form of watch crystalline lens, often they are thickened or thinned and dim.

Distrophic disorders in tissue of a locomotor upper extremities and their belt, and also can be manifested in the form of myalgia and myositis of extensors of palms and fingers, suprascapular muscle, periarthrosis and deforming of arthoris of elbow, shoulder and interphalanxes limbs.

From the side of the central nervous system, there is a neurosis-like syndrome, with mild vegetative dysfunction. In the measure of the development of the disease, patients have intensified irritability, fatigability, headache, sleep disorder, cardialgia, dizziness, lability of pulse and arterial blood pressure.

Thus, if to sum up, it is characteristic for Stage II of the vibration disease to have deepening of clinical manifestations of the syndrome of a vegetative and sensor polyneuropathy of upper limbs with more diffused decrease of superficial sensitivity, more marked peripheral angiodistonic syndrome, stable vegetative and trophic disorder of bones, frequent angiospams of fingers, as well as the development of myofibrodistrophic syndrome.

Nowadays, *marked manifestations* can be met very seldom. Such patients have a syndrome of sensomotor polyneuropathy of upper extremities with the intensification of pain and paresthesia, manifestation of weakness in hands, as well as decrease of force in them. Here, there is hypotrophy of bone muscles, forearms, and the decrease of tendinous reflexes.

Some patients might have generalization of angiospasms and manifestation of the latter on toes as well. In such cases, a syndrome of vegetative and sensor polyneuropathy with generalized acroangiospasms can be diagnosed.

It is also necessary to make a stress, which is in the basis of clinical pattern of these forms of the vibration disease, which can be encountered today, initial manifestations of the disease and symptoms are put in the basis of the local vibration, which can be considered as a transitional one from initial to moderately marked manifestations. Besides that, there are symptoms of visceral pathology, e.g. change of the level of arterial pressure with overbalancing of hypertensia, functional disorder of the activity of alimentary gland, dyskenesia of a stomach, intestines, gall tracts, disorder of vitamin balance, as well as carbohydrate, protein, and mineral exchange. It is considered that these changes have reflector character and are conditioned by the disorder of endocrine and vegetative regulation.

Vibration Disease, Caused by the Impact of General Vibration

Classification. There are three stages of the severity of a pathological process:

I – initial manifestations: 1) angiodistonic syndrome (cerebral or peripheral); 2) vegetative-vestibular syndrome; and 3) syndrome of sensor (vegetative-sensor) polyneuropathy of lower extremities.

II – moderately marked manifestations: 1) cerebral-peripheral angiodistonic syndrome; 2) syndrome of sensor (vegetative-sensor) polyneuropathy together a) with polyradicular disorder (syndrome of polyradiculaoneuropathy); b) with secondary lumbosacral plexus syndrome (due to osteochondrosis of the lumbar sector of the spinal cord); and c) with functional disorders of the nervous system (syndrome of neurasthenia).

III – marked manifestations: 1) syndrome of sensormotor polyneuropathy; and 2) the syndrome of dyscylindar encephalopathy together with peripheral neuropathy (syndrome of encephalopolyneuropathy).

Clinics. Patients in *the initial stage* of the disease complain to have headache, irritability, fatigability, and sleep disorder. Here, we can observe the lability of pulse and arterial pressure, though predominant signs are hypertensia, hyperhidrosis, and affection of dermographic reaction. All these prove the development of cerebral angiodistonic syndrome. Together with this syndrome, there are manifestations of peripheral angiodistonic syndrome, for which it is characteristic to have unstable and moderately expressed paresthesia and pain in lower limbs, sometimes cramps of sural muscles. During the examination, it is possible to observe cyanosis or mottled skin, hypothermia of feet, hyperhidrosis of soles, reduction of perception of vibration and pain sensitivity on toes. If there is intensification of pain and paresthesia in feet, expression of peripheral vegetative and vascular disorders, decrease of superficial sensitivity on the polyneurotic type, mostly in distal sections of lower extremities prove the presence of the syndrome of vegetative and sensor polyneuropathy of lower extremities.

In case of the development of the disease of Stage II, there are *moderately marked* symptoms of the syndrome of vegetative and sensor polyneuropathy of lower extremities. In some cases, similar symptoms may take place in hands as well. Together with vegetative and sensor polyneuropathy, secondary lumbosacral syndrome develops in the result of osteochondrosis of the lumbar part of the spinal cord.

Marked manifestation (Stage III) of the disease can be observed very rarely. For this stage of the disease, it is characteristic to have sensomotor polyneuropathy (pain and weakness in lower limbs when walking, reduction of force and hypotrophy of specific muscles of shins and feet, pain in nerve trunks when palpating).

Vibration Disease, Caused by Combined Affection of General and Local Vibration

Classification. There are stages of three stages of the disease:

I – *initial*;

II – *functional*;

III – *marked manifestation*.

Clinics. The disease starts gradually. Patients complain to have headache, dizziness, increased irritability, general sickness, and fast fatigability, ache in lower extremities, as well as their numbness and paresthesia. There can be fits of whitening of toes. The disease at this stage (initial) is manifested through neurasthenic syndrome with phenomena of vegetative dysfunction. The disease has compensated character, and after the termination of contact with vibration, the ability of the sick to work is renovated.

Further, headache becomes constant, agitation increases, and desire to whine appears. Periodically, there are “vegetative crises”: nausea, short-term lapse of memory, dizziness, and increased general hyperhidrosis. Skin coverlets become pale, eyes become brighter, pupils widen, muscles are tensed, and the body temperature increases. Fits end up with profuse hidrosis with further development of prostration. This stage (*functional*) can be characterized by the development of general angiodistonic phenomena with “vegetative crises”, lability of cardio-vascular system, and vegetative-sensor polyneuropathy of extremities. There is a marked asthenic syndrome.

At later stages of the disease, there are following sings: worsening of memory, sleep disorder, formation of diencephal syndrome (weight loss, anorexia, acute asthenia, microorganic symptoms of the affection of stem portion of brain and hypothalamic sector), there are changes in the cardio-vascular system (bradycardia, and arterial hypotensia). This stage (marked manifestation) has a number of peculiarities: headaches become more permanent. Crises with short-term loss of consciousness become more frequent, vegetative and sensitive polyneuropathy of extremities develops, as well as encephalopathy and diencephal syndrome.

Differential diagnostics. Differential diagnostics of the vibration disease is conducted to determine such diseases as Reino syndrome, syringomyelia, vegetative polyneuritis and myositis.

Reino disease mostly develops at women; its development does not depend on the occupation. Clinically, it can be characterized by marked angiodistonic syndrome of peripheral vessels, presence of disorders of vibration, pain, temperature and tactile sensitivity, as well as changes in the internal organs, and locomotor system.

Syringomyelia starts gradually. It is characteristic for it to have segmental disorder of sensitivity, atrophy, pareses, and paralyses; also finger whitening can sometimes take place. Together with disorders of pain temperature sensitivity according to the segmental type, there is tactile and muscle sensitivity.

Vegetative polyneuritis can be characterized by disorders of sensitivity on polyneuritic type; disorders on polyneurotic type; it is not characteristic for it to have affection of vibration sensitivity and fits of angiospasms. In addition, affection of trophism, temperature and tactile sensitivity decrease can take place.

Myalgia and myositis have definite connection with the occupation. They can be characterized by pain when palpating muscles, absence of peripheral vessels, specific disorders of sensitivity at plexitis (disorder of pain sensitivity at the absence of disorders of vibration, temperature and tactile sensitivity takes place).

Treatment. Etiological principles of treatment of patients with vibration disease involve the principles of elimination (temporary for the period of treatment or full-time in case of absence of therapeutic effect) from the work under conditions of the impact of vibration and other unfavorable factors of production environment.

Among generally strengthening and treatment/preventive measures, it is necessary to consider aero-, gelio and hydrotherapy with the utilization of natural factors of the external environment: air baths, dosed sun irradiation, and swimming in open pools in summer.

As to special treatment – preventive measures, it is necessary to recommend vitamin therapy (ascorbic acid, B_1, PP and B_{12}), irradiation with UV rays, preparations, which increase non-specific reactivity of organism.

To conduct pathogenic therapy, it is recommended to use anticholinergic drugs, ganglionic blockers and acupuncture. Among anticholinergic drugs, good results are shown by spasmolytin and benactyzine; and ganglionic blockers - pachykarpin, benxohexamethonium and hexamethon. More positive result is given by combining ganglioblockers and anticholinergic drugs with preparations, which are capable to expand vessels (nicotine acid and papaverine). Nowadays, new data as to positive effect of the recommended calcium channel blocking agents, first of all, the group of nifedipine (corinfar and cordafen, 10 mg three times a day for three weeks), and particularly, corinfar and unithiol (5 ml of 5 % solution, 10 injections), show fast

improvement of the condition of patients, which is accompanied by weakening of pain, acroparaesthesia in hands, disappearing of angiospasticattacks, earlier appearing of the feeling of warmth in hands, and sleep improvement. Together with this, structural and functional state of membranes of erythrocytes, indications of peripheral and central hemodynamics, and rheological properties of blood get normal.

Among physical methods of treatment, it is recommended to have iontophoresis of 5 %solution of Novocain onto hands; diathermy on cervical ganglions; UV irradiation of cervical ganglions; and utilization of two or four chamber galvanic baths.

It is recommended to conduct a spinal blockade 0.25 % solution of diphacyl together with Novocain, UV irradiation on the level of segments C_3-C_4 and $Д_5$ and $Д_6$, starting with 2 or 3 biodoses, increasing it to 3 or 4; course is 7 to 8 sessions. It is also recommended to undergo hydrogen sulfide, nitric-thermal, rhodon baths and mud cure (37 – 38 °C) as well as rational meals.

Verification of the ability to work. At the disease of Stage I for patients, they are temporarily (for one month) employed at work beyond the action of vibration (with the provision of a leave on occupational inability to work in case of the decrease of earnings). When qualification of a worker at change of employment is decreased much, then a percentage of the loss of the ability to work for the period of re-qualification is set by the decision of a treatment – expert commission (one year).

Similarly, issues can be solved in case of vibration disease of I to II stages. Only to achieve a stable effect of such diseases, patients are transferred to work beyond the action of vibration for the period of two months.

Treatment of patients with Stage II of the disease should be done in hospital with further transfer to work, which is not connected with the impact of vibration or cooling down to fix results of treatment for 1 or 2 months. In case of acute decrease of qualification at the change of work, thy can be assigned to undergo expert examination to determine the degree of the loss of the ability to work for the period of re-qualification (1 – 2 years).

As a rule, patients with vibration disease of Stage III have limited ability to work. They obtain a percentage of the loss due to occupational inability to work or an invalidism group (III) due to the occupational disease.

Patients with vibration disease of the 1^{st} degree do not lose their ability to work due to general vibration. They undergo treatment, and then to stabilize its results, they are transferred for a month or two to work that is not connected with the impact of vibration, intensive noise, and receive a leave as to their inability to work.

At well-marked pathological changes of the 2^{nd} degree, it is necessary to undergo rational employment with the definition of the degree of the loss of the ability to work (for one year).

For patients with vibration disease of Stage III, it is characteristic to have the decrease of occupational and general ability to work. They can obtain 2nd or 3rd group of invalidism in the result of the occupational disease.

Preventive measures. Technical measures – decrease of vibration in the source of their formation, utilization of carious shock-absorption means, provision of normal microclimatic conditions in premises, where work on vibration instruments and equipment is carried out; hygienic formation of the level of vibration; as well as organization of the regime of labor at minimal contact with those, who work with vibrating instruments.

It is recommended to conduct hydraulic procedures – bathes for hands with the temperature of water 37 °C together with self-massaging; UV irradiation sub erythematous dosages of mostly cervical areas; gymnastics and regular medical check-ups.

OCCUPATIONAL PATHOLOGY, CONDITIONED BY THE IMPACT OF NOISE

Noise is a chaotic combination of sounds, i.e. mechanical vibrations in the zone of frequency from 20 Hz to 16 kHz, which are perceived by a hearing analyzer.

Under conditions of the production of the noise impact, there are engine testers, riveters, cutters, copper-smiths, weaver and spinners. Noise is an unfavorable factor of production environment impacts mechanization experts in agriculture, as well as repair shop workers.

A hearing apparatus of a human being can perceive a sound with the frequency from 16 to 20 000 vibrations a second. As to the spectral composition, it is possible to distinguish high frequency noise with the predomination of levels on the frequencies higher than 800 Hz, and low frequency – with most levels on frequencies lower than 300 Hz and mean frequency, which cover an intermediate diapason of frequency (300 to 800 Hz). The character and degree of expression of the action of noise onto the hearing organ is determined by its intensiveness, key, periodicity, as well as joining of noise with other occupational factors, in particular with vibration.

Pathogenesis. Until 1960’s, it was considered that noise causes affection on only a hearing analyzer. It was stated that in the basis of occupational partial deafness, there are destructive changes of both hair cells of cochlea, and also in spiral ganglion and in hairs of a cochlear nerves. And only for the last twenty years, a possibility was proved of a non-specific action of noise onto an organism was proved, which manifested itself in the affection of the functional state onto the organism, which manifested itself in assumption in disorder of the functional state of the nervous and cardio-vascular systems.

At first, hair cells of the lower cochlea of helix, which perceive sounds of high tones. If further impact takes place, support cells of Deiterse and internal hair cells are involved. A number of nervous fibers of external hair cells

decreases. Cells of spiral ganglion look pressed together and their number is decreased. At the occupational partial deafness, sound-perceiving apparatus is affected (a spiral organ and a plexus of fibers of a helix of vestibulocochlear nerve around hair cells), i.e. occupational worsening of hearing belongs to perceptive partial hearing.

It is necessary to remember that in the development of pathology of the vestibulocochlear nerve, a significant role is played by the affection of nervous and cardio-vascular system, conditioned by disorders of blood circulation and tissue trophism. Under the impact of intensive and prolong noise, agitation of the hearing center is transferred to the mesh substance and reticular formation.

High frequency noise is transferred subjectively worse and it has more dangerous impact onto the organism. Impulse noise is considered harmful than constant one.

Clinics. With the development of occupational partial deafness, there are four stages of loss of hearing (Table 9). Occupational partial deafness develops according to the type of cochlear neuritis and can be characterized by gradual development. At first, there is noise in ears, which becomes more intensive and stable gradually. At research with a tuning fork or with audiometry already at early stages, there is a decrease of perception of high frequencies (4000 – 6000 Hz) and the reduction of bone conduction. Gradually, worsening of hearing, there are also other tones, and the level of perception of whispering reduces as well. Bad hearing of whisper attracts attention as well, though hearing of speech is still good. The latter is affected only in case of presence of a very large work period under conditions of the impact of noises (20 years and more). Otoscopic pattern goes without changes.

Table 9

Criteria of Evaluation of Hearing Function for People, Who Work under Conditions of Impact of Noise and Vibration (According to V.Ostapkovych and N.Ponomariov)

Disease Degree	Indications	Tonal Threshold Audiometry		Distance, whisper is perceived at
		loss of hearing at audio frequency 0.5; 1 and 2 kHz, dB	Loss of hearing at 4 kHz and limits for possible oscillation, dB	
I	Signs of noise impact onto a hearing organ	up to 10	50 ± 20	5 ± 1
	Cochlear neuritis			
II	with mild degree of hearing worsening	11 – 20	60 ± 20	4 ± 1
III	with mean degree of hearing worsening	21 – 30	65 ± 20	2 ± 1
IV	with severe degree of hearing worsening	31 - 45	70 ± 20	1 ± 0.5

Thus, for patients with occupational partial deafness of the *1st degree*, whisper can be perceived at the distance of 5 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 50 dB.

For the *2nd degree* (cochlear neuritis with a mild degree of hearing worsening) – whisper can be perceived at the distance of 4 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 60 dB.

For the *3rd degree* (cochlear neuritis with a mean degree of hearing worsening) – whisper can be perceived at the distance of 2 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 65 dB.

For the *4th degree* (cochlear neuritis with a severe degree of hearing worsening) – whisper can be perceived at the distance of 1 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 70 dB and more.

Thus, at the initial stage of the disease, perception of whisper (diapason of frequencies within limits of up to 2.5 kHz) does not almost change, and workers do not notice the decrease of their hearing. However, special checking with the help of audiometer at frequencies of 4 to 6 kHz demonstrates the decrease of hearing well. At this, both air and bone perception is affected on the same level; the process has symmetrical character, affecting both right and left ear. Along with progressing of the disease under the impact of noise, perception in the area of audio frequencies of 2, 1 and 0.5 kHz decreases; it usually develops gradually and slowly, and it increases with the period of work on the position, what curves of hearing threshold are shown (Fig. 6 – 9).

Complaints to have complaints on general sickness, increased irritability, bad sleep, headache, dizziness, and sound of noise or ringing in ears take place. Some patients complain to have pain in hear, often of complaining character with the irradiation under the left shoulder-blade. In future, there are complaints to have the decrease of hearing of both ears. At objective examination, there are vegetative disorders on the general neurotic background, which takes place in the form of instability in the position of Romberg, trembling of stretched our arms, red stable dermographism, as well changing of a reflector sphere.

At noise pathology, there are disorders in metabolic process. Change in protein exchange is manifested through the increase of general protein and globuline.

Clinical observations show the change of heart activity among those, who are subject to noise impact. Thus, on electric cardiograms, there is lability of pulse and slowing down of intra-ventricle and intra-atrial heart conductivity.

The majority of researchers consider that under the impact of a prolong and systematic noise, arterial blood pressure is increased, thus noise can be a factor of risk in the development of hypertonic disease.

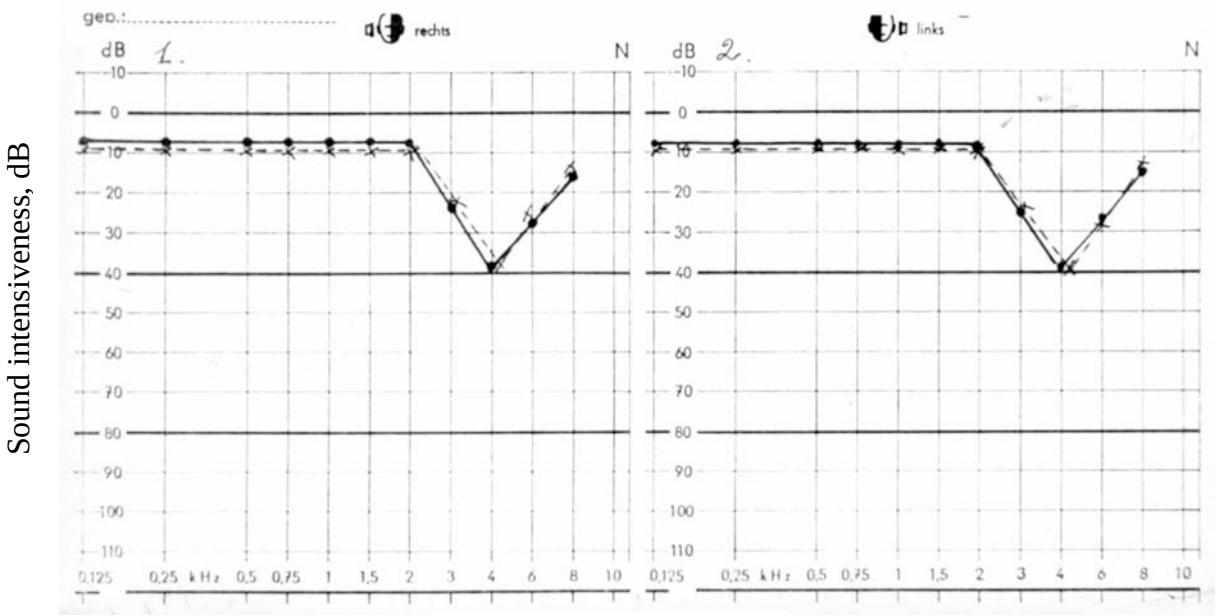


Fig. 6. Audiogram at initial sings of the impact of noise onto hearing organs

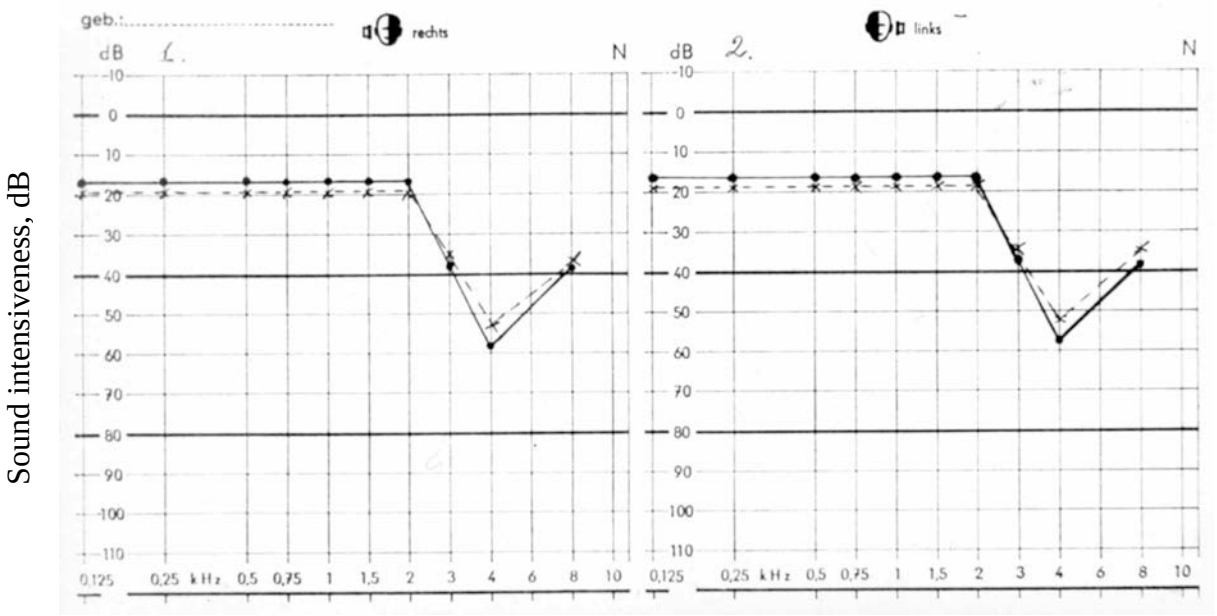


Fig. 7. Audiogram at cochlear neuritis with a mild degree of hearing worsening

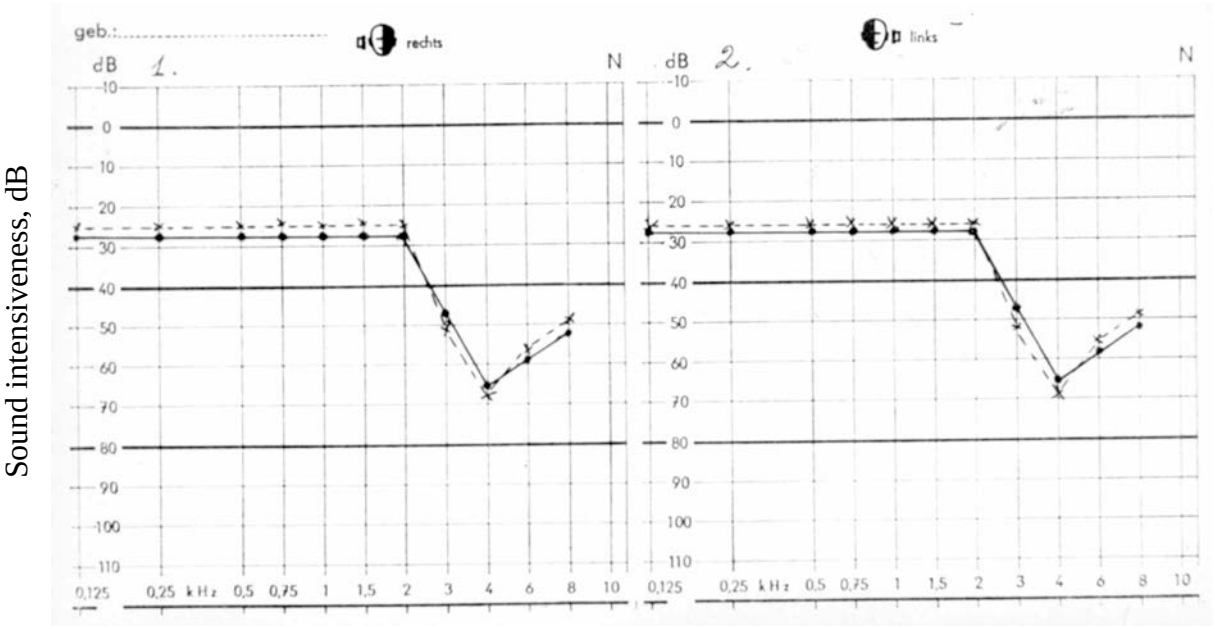


Fig. 8. Audiogram at cochlear neuritis with a mean degree of hearing worsening

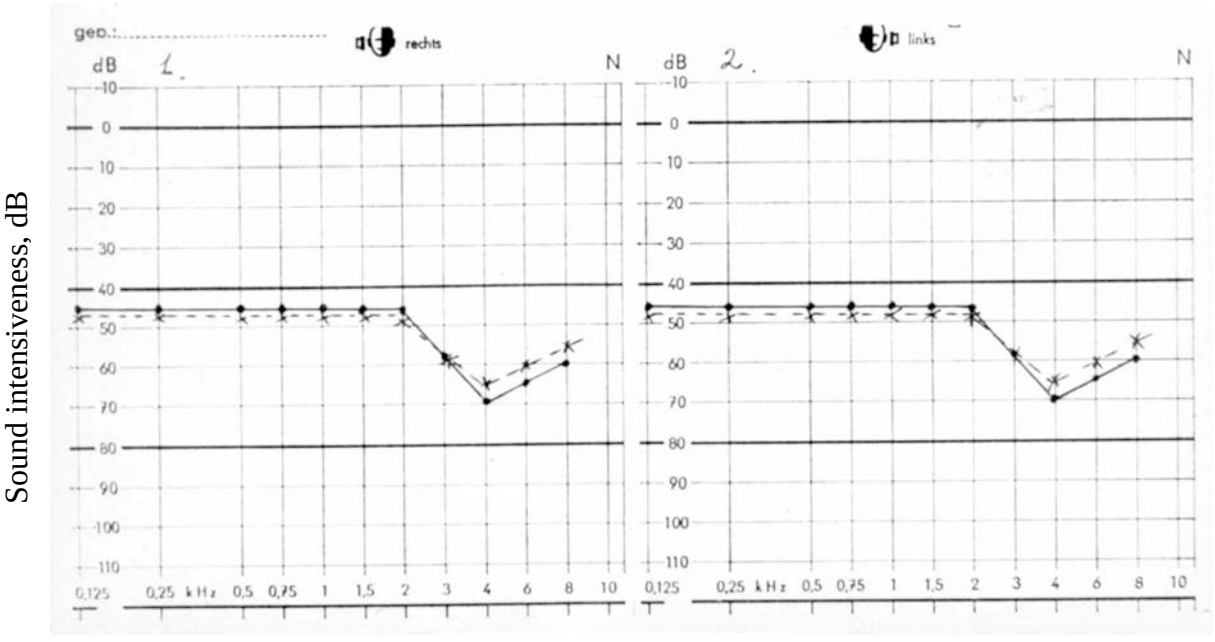


Fig. 9. Audiogram at cochlear neuritis with a severe degree of hearing worsening

Diagnostics. The diagnose of cochlear neuritis is made based on occupational guidelines, sanitary and hygienic characteristics of labor conditions, study of complaints of a patient, anamnesis of the disease and life, instrumental examination of hearing and vestibular analyzers.

For occupational partial deafness, it is characteristic to have the following: gradual development of the process; presence of correlation between disorders of the function of the central nervous system and a number of other organs and systems with changes of hearing sensitivity; presence of similar cases of the disease among workers of the professional group; absence of indications in the anamnesis as to the beginning of this disease during the period, which was prior the contact with occupational hazardous factors; and loss of hearing takes place in the result of affection of air and bone conductivity; whereas deafness is stable and permanent even when the source of noise is eliminated.

Treatment. Considering the peculiarity of the clinical manifestation of syndromes, conditioned by the action of noise, it is necessary to approach selection of therapeutic means in a differentiated way. First, it concerns generally strengthening therapy, organization of regular meals, sleep regime, obligatory staying in the fresh air for 1 to 2 hours, every day.

Among medical grudges, it is recommended to use bromide, elenium, trioxazine, benactyzine, as well as glutamine acid and rutin, depending on the expression of an accompanying syndrome and cochlear neuritis.

With the purpose of generally strengthening action, it is recommended to use ascorbic acid in dosage of 300 to 500 mg, as well as a complex of vitamins of group B.

At the presence of angiodistonic syndrome, it is recommended to use spasmolitic means and ganglionic blockers (bromides, aminazine, meprotane together with bensohexamethonium and penthamine).

In case of development of neurocirculatory dystonia of hypertonic type at the patient, it is recommended to prescribe bromide, valerian, and diazepam in combination with spasmolytics. As neurotropic drugs, it is recommended to prescribe reserpine and rhaunatin, which initiates processes of the connection of noradrenaline and dopamine in depositing granules of ends of post-angliar simpatico fibers, and do not let to deposit noradrenaline, which circulates in blood, also has tranquilizing and neuroleptic action.

Among physical methods of treatment, galvanization on the method of Scherbak, darsonvalism, and UVF onto carotid sinus zone are used. To conduct balneotherapy, it is recommended to take salt-coniferous, carbonic acid gas, and hydrogen sulfide baths.

To treat neuritis of hearing nerves, it is necessary to use diabasol, nicotine acid, sulfate atropine and tropacine.

Among physiotherapeutical methods, good results are shown by diathermy onto the zone of mammiform process, and mud application onto the ear area.

Verification of the ability to work. At the neuritis with mild decrease of hearing, the work ability of a patient is saved as a rule. It is necessary to conduct dynamic doctor observation, and conduct of outpatient treatment with utilization of sanatorium-preventoriums.

At cochlear neuritis with a mean degree of the decrease of hearing of a qualified worker with long occupational period of work, it is also possible for them to keep their work under thorough observation and conduct of outpatient treatment. In a number of cases, if general disorders prevail, in compliance with occupational medical leave, it is expedient to transfer a patient to another occupation, which is not connected with the impact of sound. However, young people with a short period of work, as well as unqualified workers and people who are subject to impulse noise, especially in case of fast progressing of the process, it is necessary to recommend rational employment beyond noise.

At cochlear neuritis with a sever degree of hearing worsening, it is recommended to have rational employment, which is not connected with the impact of noise. In all these cases, if rational employment is impossible without the demotion of qualification, patients are to be sent to the expert commission to determine a group of invalidism of an occupational character.

Preventive measures. Introduction of various earplugs and cotton wool; anti-noise bushes of the type of “Earplug”; as well as utilization of earphones and helmets. Preliminary and periodical medical examinations are recommended.

OCCUPATIONAL DISEASES, CONDITIONED BY THE IMPACT OF ELECTROMAGNETIC IRRADIATION OF RADIO-WAVES

Electromagnetic irradiation as a totality of electric and magnetic fields is one of the main factors of the impact of factors of the impact of the environment. Electric and magnetic field do not exist separately from each other, and their mutual transformation condition appearing of a unified electromagnetic field, which expands in the surrounding environment in the form of electromagnetic waves with the speed of $3 \cdot 10^8$ m $\cdot$ c^{-1}.

Electromagnetic irradiation can be characterized by the oscillation frequency and the length of waves. The frequency is measured in Hertz (Hz) (1 Hz is equal to one oscillation a second), and the unit to measure waives is a meter (m). Derivatives of these units are correspondingly kilohertz (1 kHz = 10^3 Hz), a megahertz (1 MHz = 10^6 Hz), and also a kilometer (km), a centimeter (cm), etc.

Based on biological action of various range of action of the irradiation (Table 10), in hygienic and medical practice, it is recommended to use a simplified classification of main ranges of radiation.

The range of electromagnetic irradiation is very wide: from infrasound with the frequency of up to 3 Hz and the length of a wave – over 10^8 m and to ionizing (X-ray and γ-irradiation) with the frequency over $3 \cdot 10^8 – 3 \cdot 10^{11}$ Hz,

the intensiveness of electromagnetic irradiation can be characterized by surface density of energy current, i.e. the amount of energy, which goes through the plane, the area of which is equal to one unit, located in perpendicular to the direction, where electromagnetic waves spread. A unit of measuring of the density of the energy current is a Watt per a square meter (W/m^2). Boundary permitted level (BPL) of the density of the flow of energy of radiation in the range of low frequencies at irradiating throughout the whole workday – $0.1/m^2$, at irradiation for not more than 2 years – 1 W/m^2, and not more than 15 to 20 min – 10 W/m^2 under condition of obligatory utilization of protective glasses.

Table 10

Characteristics of Main Ranges of Radiation

Range of Frequencies, Hz	Range of Waves, m
High (HF) $3 \cdot 10^4 – 3 \cdot 10^5$ $3 \cdot 10^5 – 3 \cdot 10^6$ $3 \cdot 10^6 – 3 \cdot 10^7$	 Long (LW) - $10^4 –10^3$ Medium (MW) $10^3 –10^2$ Short (SW) – $10^2 – 10^1$
Ultra high (UHF): $3 \cdot 10^7 – 3 \cdot 10^8$	Ultra short (USW) – 10^1 –1 (meter)
Superhigh (SHW) $3 \cdot 10^8 – 3 \cdot 10^9$ $3 \cdot 10^9 – 3 \cdot 10^{10}$ $3 \cdot 10^{10} – 3 \cdot 10^{11}$	Microwaves: Decimeter - $1 –10^{-1}$ Centimeter – $10^{-1} – 10^{-2}$ Millimeter – $10^{-2} – 10^{-3}$

Electromagnetic irradiation is utilized for thermal processing of metals, semiconductors and dielectrics. Induction heating up of metals and semi-conductors is carried out mainly by magnetic field in the range of HF and UHF.

HF and UHF equipment is used to dry various materials (wood, paper and leather), to warm up plastics, welding of synthetic materials (production of book covers, folders, bags and toys), as well as sterilization of food.

Such rages of electromagnetic radiation as HF, UHF and SHF are most widely used in radio broadcasting and TV broadcasting, and SHF range – for microwave-link equipment, radiolocation, radio navigation and radiodefectoscopy. Active implementation of radiation in physiotherapy needs particular attention. Properties of radiation to warm up tissues in organism are used in such procedures as low frequency magnetic therapy (devices “Polus-1”, and “Polus 101”), inductometering (devices DKV-2 and 1KV-4), microwave therapy with centimeter (such devices as “Luch-2”, “Luch-3” and “Luch-58”) and decimeter waves (devices “Volna-2” and “Ranet”).

The main source for artificial electromagnetic radiation is radio and TV stations, radiolocators, and high-tension lines. It is necessary to remember that together with radiation. It is necessary to remember, that together with radiation, servicing personnel is often subject to other hazardous production factors. In the areas of induction heating and at processing of electronic schemes with utilization of soldering, in cabinets of intercity relay stations, it is possible to

observe contamination of air environment with aerosol of lead, tin, carbohydrate, and nitric oxide. In cabinets of intercity relay stations, premises of radio- and TV stations, at the areas of induction heating up, there is high temperature of air; the level of noise can be up to 75 – 99 dB. The work of operators of intercity relay stations, as well as personnel of radio and TV stations is accompanied by significant nervous, emotional and sight tension.

Pathogenesis. The mechanism of the action of radiation onto a human being is very complicated and is not comprehended in full. It has been stated that electromagnetic irradiation causes radio wave and heating impact onto biological objects. Heating action of microwaves is reduced to the fact that at every change of the direction of an electromagnetic field, there are relaxation oscillations and transition of ions in organism tissues, which electromagnetic irradiation is aimed at, and it is accompanied by the discharge of heat and increase of the tissue temperature. Blood, lymph glands, parenchymal organs, muscles and crystalline lens warm up most of all.

Thus, in the basis of a heating action of electromagnetic irradiation there are primary processes of interaction of electromagnetic waves with tissue molecules. Electromagnetic energy in a biological environment transforms engulfing molecules into kinetic energy, what causes tissue heating. The degree of increase of the temperature is determined by field strength, duration and frequency of irradiation, depends on the fact what part of body is subject to irradiation, as well as effectiveness of thermal regulation and some other indications.

The mechanism of the action of radio radiation of a small (less then heating) intensiveness is realized mostly through its reflector action onto the central nervous system. Hypothalamus is the most sensitive to the impact of radio waves, where highest vegetative centers are accumulated. It has been stated that parasympatic portion of vegetative nervous system is the most sensitive to the action of radio radiation, than a sympatic one.

The action of radio radiation onto the brain is realized by a complex group of biophysical, physical and chemical, and quantum-biological effects. On the cell and subcell levels, there are changes of potassium-sodium gradients in cells, polarization of biological membranes with the affection of their permeation, deformation of structures of water systems, change of activity of ferments, disorder of oxide processes, etc.

Conditionally, there are mechanisms of biological action of electromagnetic fields as follows: a) direct action onto tissues and organs, providing the change of functions of the central nervous system, as well as neurohumoral regulation connected with it; b) reflector changes of neuroumoral regulation and c) joining of main mechanisms of the pathogenesis of the action of electromagnetic irradiation with mostly disorder of the exchange of matters, and activity of ferments. Probably, all three mechanisms are rightful, and a role of each of them is determined by physical and biological changes.

Clinics. Clinical symptoms of the impact of radio irradiation depend on its range, intensiveness and duration, and possibly on the regime of irradiation.

Research show that the most biologically active ones are SHF- waves, then UHF range and the less active one is HF range.

A c u t e f o r m of pathological impact of electromagnetic irradiation is divided into three stages: *mild, mean* and *severe*. Acute form of the affection takes place during accidents or in case of gross violation of the safety measures, thus in this case, if the intensiveness of irradiation much exceeds the thermal threshold. People, who suffered from the impact of intensive SHF irradiation, the body temperature increases by 1 to 2 °C, general weakness, sickness, pain in extremities and muscles, headache, state of worrying, thirst, dyspnea, face reddening, hyperhidrosis, lability of pulse and arterial pressure and nose bleeding take place, and leucocytosis can be observed. Sometimes, there are simpatico-adrenal crises, and fits of paroxysmal tachycardia.

After acute affection with electromagnetic irradiation, there are functional disorders of the nervous system in the form of vegetative-vascular dystonia or asthenoneurotic syndrome. These stages last for 2 to 3 months and then pass.

C r o n i c f o r m of the affection takes place in the result of prolong action of electromagnetic irradiation, intensiveness of which exceeds BPL, however it is lower that the thermal threshold.

In this case, reaction – reply of the organism is both in adaptation of reconstruction of the nervous and cardio-vascular system, and in the development of cumulative effect, which is manifested through the intensification of pathological reactions in the organism with the period of work. In the first plan, there is disorder of functions of the nervous and cardio-vascular system. People, who worked under conditions of the action of electromagnetic field, adrenocorticotropic activity of the hypophysis, the activity of sex glands is inhibited, enzymopathy appears; neurocircular dystonia develops on hyper- or hypotensive type; immune and biological reaction of the organ is changes; and trophic disorders can be observed.

Under conditions of modern production, the development of symptom complex of chronic SHF affections is possible only at the period of work not less than 10 to 15 years. Women are more sensitive to the action of electromagnetic irradiation, than men; general health state, previous infections and overfatigue of a person are very important as well. In spite of general polymorphic clinical pattern of the disease, the main role of action of electromagnetic fields onto a human being is played by functional disorder of the central nervous system and the affection of cardio-vascular system.

Changes in the central nervous system can be characterized frost of all by the development of asthenoneurotic and vegetative symptom complex.

Asthenic syndrome develops in the initial stage of the disease. Patients complain to have increased fatigability, irritation, and headache without clear

localization, as well as sleep disorder, unpleasant feelings in the heart area, and hyperhidrosis.

In case of growing of the expression of asthenic reactions and joining of vagotonicsigns, the so-called astehenovegtative syndrome develops, which clinically manifests itself through growing of general weakness, further decrease of the ability to work, memory and concentration. Dizziness and stable arterial blood hypotensia with bradycardia cause the development of consciousness losing.

Further in the clinical symptoms, the change of the direction of vegetative reactions with further transition to the arterial hypotensia and bradycardia into the state, which can be characterized by the lability of the arterial pressure and pulse, and into a neurocircular dystonia of the hypertonic type can take place. Increase of tonus of the sympatic part of a vegetative nervous system can be characterized by the presence of frequent and long headache, dizziness, pain in the heart area, sleep disorder, memory disorder and emotional lability. At examination, such patients have the increase of reflexes, finger and eyelid tremor, general hyperdrosis and bright red dermographism. In some cases, in case of prolong impact of SHF irradiation, when a patient has fit-like intensive headache, dizziness, general weakness, cardialgia, tachycardia, arterial hypertensia, body temperature increases, and a patient has a fixed idea about the death threat. On this background, there can be present scattered organic symptoms, which prove the development of dysciruclatory encephalopathy.

On the background of the given above neurological syndromes, there are also functional disorders in the cardio-vascular system. This can be manifested through unpleasant feeling in the heart and pain in the heart area. There are also lability of pulse and arterial pressure, shift of the limits of heart to the left, and sometimes, there can be systolic noise to the top of the chest.

At electrocardiograph research, there is often a change of rhythm of heart contractions (sinus arrhythmia) and amplitude of T wave.

People, who suffer the action of electromagnetic irradiation, often have endocrine-metabolic disorders. First of all, it is manifested through the increase of the functional activity of a thyroid gland, disorder of the activity of sex glands, increase of the secretion of adrenalin and noradrenaline with urine, loss of weight, hair loss and nail fragility.

As to the peripheral blood, there is a tendency to lymphocytosis and thrombocytopenia. There is a big number of eosinophiles, monocytes, reticulocytes, the content of general protein at the expense of the level of globulin, histamine, cholesterol, as well as the decrease of albuminic-globulin and potassium-calcium ration, and the level of chlorides.

In addition, digestion system suffers from it. It is manifested through dyspeptic disorder, vaguely marked pain syndrome. There are insignificant changes in extractor and inserter function of pancreas.

People, who have worked with the source of the SHF irradiation for a long time, might have the development of dystrophic changes of a crystalline lens – cataract, a characteristic peculiarity of which is the localization of a pathologic process in the area of a rear pole.

Depending on the expression of changes in various organs and systems, there are three stages of the disease.

Stage I – it is characterized by the development of asthenic syndrome, which is often combined with vaguely marked vagotonic symptoms. Functional activity of a thyroid gland increases. These changes have functional character and do not affect much work abilities of patients.

Stage II of the pathological process can be characterized by the development of asthenovegetative syndrome with a stable bradycardia and arterial hypotensia. However, there can be vegetative-vascular dystonia with the lability of pulse and arterial pressure. There are deeper dystrophic changes in myocardium, changes can be found in peripheral blood, and there are moderate endocrine-exchange disorders.

Stage III of the disease can be met very rarely. Hypothalamic syndrome develops; sympathico-adrenal crises become more stable. There is a fit-like headache, shivering, seizing pain in heart, acute weakness, and arterial hypotensia. At much tense of electromagnetic field, encephalopathy with psycho disorders can develop accompanied by memory worsening, depression and mitochondria state.

Diagnostics. To state a diagnosis of the occupational disease in the result of the impact of electromagnetic irradiation, it is necessary to conduct detailed sanitary and hygienic characteristics with defining frequency range of oscillations, intensiveness of irradiation, term of contact, period of work under harmful conditions of the production. It is also necessary to consider non-specifics of manifestations of the disease and on considering it, to eliminate other general diseases, which can condition the development of asthenia or cause neurocirculatory disorders.

Characteristic manifestations of the action of electromagnetic irradiation onto the organism of a human being is asthenic or asthenovegetative syndrome with a vagotnoic direction of disorders, which further are replaced by the syndrome of vegetative and sensor dystonia with prevailing sympathic reactions, appearing of endocrine-exchange disorders, changes of indicators of blood, presence of a cataract. Fast reverse progress, in particular, in initial stages, under the impact of treatment and in the result of normalization of work conditions are the proof of the diagnosis.

Treatment. Asthenic states are indicators to prescribe tranquillizers (trioxazin 0.3 g, diazepam 5 mg two or three times a day); generally strengthening preparations (injections 5 ml of 5 % solution of ascorbic acid with 20 ml of 40 % solution of glucose intravenously once a day, and the course is 15 injections; 1 ml of 6 % solution of thiamine bromide; 1 ml of 5 % solution of

pyrodixine hydrochloride intramuscularly once a day, course – 15 to 20 injections); tonic preparations (saparal 0.05 g two or three times a day, ginseng tincture 25 drops three times a day).

At parasympathic – toning direction of vegetative disorders, it is recommended to use anticholinergic drugs (ergotamine hydrotartrate – beloid, belataminal – 1 pill two or three times a day); antihistamine medical preparations (Dimedrol 0.05 g and suprastin – 0.25 g).

In case of the presence of hyperkinetic syndrome in cardio-vascular system (tachycardia, palpitation, tendency to increased arterial blood pressure), it is recommended to have small dosages of β-blockers: propranolol 0.02 g two or three times a day; preparations, which widen vessels and have hypotensive action (rhaunatine – 2 mg, cynarizin – 25 mg, caviton – 5 mg three times a day, no-shpa or papaverine hydrochloride – 2 ml 2 % solution intramuscularly once a day for 10 to 15 days).

Complex therapy includes curative gymnastics, reflexo- and psycho therapy, diet with small food value, but high content of proteins, rhodon and coniferous baths.

Verification of the ability to work. At the initial stage of the disease, the ability to work is not affected. After active treatment, patients should be transferred to work beyond the contact with electromagnetic irradiation for the term of one month and in case of positive progress of the disease, they can do usual work.

People who underwent diseases in moderately marked stage, it is necessary to conduct treatment under conditions of specialized hospital, after what to fix results of treatment and for dynamic work, they should be transferred to work, which is not connected with the action of electromagnetic irradiation, for the term of 1 to 2 months. Return to work can take place only under condition of complete return of functions.

In case of absence of a real treatment effect, as well as in case of presence of severe stage of affection, patients need rational employment beyond the action of electromagnetic oscillations. Acute decrease of qualification is the basis for assigning patients to the Expert Commission and to determine the stage of loss of the work ability for the period of re-training (one year). In case of a cataract, further work in contact with radio irradiation is prohibited.

Preventive measures. The following methods are recommended and methods of protection for the impact of electromagnetic field: organizational, technological, sanitary and technical, individual and treatment/preventive ones.

Main organizational measures, which allow improving the state of the environment in places of location of sources of electromagnetic irradiation, there is the reduction of the duration of the action and increase of the distance from the source to a worker.

Technological measures envision the provision of mechanization and automation of production processes, utilization of manipulators and remote control.

Sanitary and technical measures involve screening of all sources of electromagnetic field. Among measures of individual protection, there are radioprotecting clothes and glasses.

Treatment and preventive measures include the conduct of preliminary and periodical medical examinations, during which it is important to do research of systems of an organism, which suffer the most from the action of electromagnetic irradiation.

OCCUPATIONAL DISEASES, CAUSED BY LASER IRRADIATION

Irradiation of optic quantum generators (lasers) is comparatively new factors of the production environment. Lasers are completely new sources of powerful directed electromagnetic irradiation. The range of their utilization is wide, and rate to implement it in various spheres of science and technology is rather fast. As in the result of concentration of high energy of irradiation in a relatively small volume, lasers enable to carry out melting, welding and cutting of various hard metals, to create high temperature plasmas, to conduct thermal nuclear reactions and to initiate chemical ones. Nowadays, laser irradiation is used in geodesic work, in systems of information transfer and guidance systems, in various scientific research in medicine, when solving complex medical and biological problems, to do surgeries in oncology, ophthalmology, dermatology, etc.

Main physical values of laser of irradiation (and its units) are as follows: the length of a wave (mkm), radiating power (watt), density of radiation current (watt $\cdot$ m^{-2}), radiant energy (joule), density of stream energy (joule $\cdot$ m^{-2}). As to their degree, they are divided into four classes: the first one– lasers, irradiation of which Is not hazardous for eyes and skin; the second one – lasers, irradiation of which is hazardous when irradiating eyes with direct or mirror reflection, as well as in case of scattered irradiation on the distance of 10 cm from a diffusively reflected surface on the distance of 10 cm from a diffusively reflected surface.

Pathogenesis. Biological action of a laser irradiation is determined by such main characteristics as wave length; radiating power; irradiation duration; frequency of pulse advancing; anatomic and functional peculiarities of tissues, which are under the impact of irradiation; and the area of irradiation of surfaces. There is a thermal and non-thermal, local action of laser irradiation.

Thermal action of laser irradiation of a non-stop action has much in common with usual warming up. Under the action of impulse laser irradiation, tissue warms up immediately with immediate boiling up of tissue liquid, what causes mechanical affection of the tissue. At high energy of irradiation (100

Joule and more) in the result of ruining and evaporating of cell elements, necrosis area with crater-like deformation appears on the skin. A characteristic feature of laser irradiation in this case will be a clear border of the affected area from the intact one.

Non-thermal action is conditioned by mostly electric and photochemical effects, as well as absorption of electromagnetic energy by tissues.

Local action of laser irradiation can cause affection of eyes and those organs, which selectively react to this irradiation.

Clinics. Organs, which are the first to be affected in case of impact of laser irradiation, are eyes and skin. The action of laser irradiation depends on pigmentation of eye-ground and the range of irradiation. At this, visible range of irradiation acts mostly onto the photosensor layer of the retina, causing loss of sight in the area of vision space.

In ultraviolet range (240 – 450 nm) of laser irradiation, energy is absorbed by all protein structures of an eye, including cornea and lens. In the result of burn, mucous tunic of an eye is the first one to be affected. At a high level of energy of irradiation, coagulation of protein of a retina causes irreversible and full loss of vision. In infrared range (close and middle areas – 820 – 1500 nm) of a laser irradiation, energy is absorbed by iris, lens and vitreous body. Iris warms up quickly, and protein coagulation in lens takes place. Subjectively, warming up of iris causes the feeling of irritation and wink/opticofacial reflex. At high level of energy irradiation, lens darkening takes place due to temperature increase. Affection of lens with laser irradiation of this range usually takes place after a long action. The range of closer area of an infrared range (1000 to 1600 nm) is the least dangerous for eyes, as there is temporary superficial affection even at high level of energy irradiation.

People, who work much with lasers, complain that their eyes are tired by the end of the day, and they have dull or sharp pain in eyeballs, impossibility to look at bright light, as well as epiphora or dryness. Vision acuteness, as a rule, does not change, however, the threshold of color recognition, increase of the durability of adaptation in darkness, and sometimes, narrowing of vision poles.

Affection of skin at the action of direct of diffusively reflected laser irradiation can have various characters – from erythema to burning. In mild cases, the action of laser irradiation onto the skin shows functional affection in the activity of intra-skin ferments, and changes of skin electric conductivity.

Laser irradiation of the prolong action causes the affection of a function of nervous and cardiovascular system; it causes changes of hematologic and immune indicators, as well as the activity of some ferments and mediators. In the majority of cases, they are joined into asthenic and asthenovegetative syndromes, which are accompanied by compensatory and adaptation reactions. The clinical symptoms, caused by the impact of laser irradiation, do not have specific character and are a result of a complex of unfavorable production factors, which take place during the violation of laser operation.

Treatment. In case of affection of eyes or skin, the character of medical assistance is determined by the progress of the affection, which depends on the length of a radiation wave. In case of affection with ultraviolet irradiation, it is recommended to use cold wash on eyelids. It is also recommended to apply by droppings 0.25 % solution of dicain or 2.5 % solution of Novocain into conjunctival sacs. In case of burning of iris, caused by irradiation of visible or close infrared area of the range, it is advised to apply by droppings a 0.1 % solution of atropine sulfate into conjunctival sacs; aseptic wound dressing onto an affected eye, and the suffered should be immediately seen by an ophthalmologist.

Verification of the ability to work. Solution of expert issues depends on the degree of the affection of an organ. If affected retina, patients should terminate temporarily the work for the period of the treatment (1 to 2 weeks). Changes of lens and retina need longer treatment (to one month) with further transfer to work, which is not connected with the impact of laser irradiation (up to two months). If a patient has progressing of the disease, this person should terminate working with lasers and for the period of re-qualification, this person is given group III of invalidism.

Preventive measures. When working with lasers, levels of hazardous production factors should not go beyond the indicators, stated by the State Standards and reference indications.

Laser should be placed in separate premises (lasers of III – IV classes), or to have screens and fences (lasers of II – III classes). Laser device should have screening shields, screens, or curtains, and to protect workers from the affection with electric current, it is necessary to use remote control and blocking; to protect hands – woolen gloves, and eyes – protective glasses.

Personnel, permitted to work with lasers, should have preliminary and periodical training and medical examination.

OCCUPATIONAL DISEASES CAUSED BY INFLUENCE OF ULTRASOUND UPON A PERSON'S ORGANISM

Ultrasound is mechanical vibrations of elastic medium, which differ from sound with vibrations of higher frequency (above 20 kHz) and cannot be perceived with person's ear. Ultrasound vibrations like sound vibrations spread out in form of alternating thickening and discharging, and are characterized with length of wave, frequency and speed of spread. The higher is frequency of ultrasound vibrations the more is degree of their absorption by medium and the smaller is depth of their penetration into person's tissue. Absorption of ultrasound is accompanied by heating of medium.

Range of ultrasonic frequency is divided on vibrations of low frequency (from $1.12 \cdot 10^4$ to $1.0 \cdot 10^5$ Hz), which spread through air and

contact, and vibrations of high frequency (from $1.0 \cdot 10^5$ to $1.0 \cdot 10^9$ Hz), which spread only through contact.

Ultrasound is applied in different spheres of economy: metallurgy, machinery construction, apparatus building, radio engineering, chemical and light industry, medicine and so on. Wide application of ultrasound stipulates increase of quantity of workers, which are under its influence. Main occupational groups, which are under influence of ultrasound, are as follows: inspectors, fitters, supervisors of sewage disposal plants, welders, tinsmith, doctors and nurses, who operate therapeutic and diagnostic ultrasonic apparatus, surgery units and sterilization tools.

Pathogenesis. Three kinds of ultrasound are distinguished depending upon intensity of ultrasonic waves and their influence on live tissue:

1. Ultrasound of small intensity (below 1.5 W/cm^2), which is considered as physical catalyser. It causes some changes of physicochemical organism response, exchange process speeding up, tissue light heating, micro massage, and does not cause morphological changes at cells.
2. Ultrasound of medium intensity (1.5 till 3.0 W/cm^2) causing reaction of oppression in nervous tissue. Speed of function renewal depends from ultrasound influence intensity and duration.
3. Ultrasound of high intensity can cause irreversible oppression even up to total tissue destruction.

Ultrasound biological action consists of violation of functional state of receptor apparatus and peripheral vegetative formations (excitation of temperature, tactile, and pain vibroreceptors); transition of mechanical vibrations energy to heating energy with dilatation of vessels those changes into spasm later. This is accompanied with reinforcement of oxygen absorption by cells and lowering of carbonic acid concentration, accumulation of dross, which influence large toxic action on central and peripheral nervous system, cause injury of cell membranes.

Besides, development of photo- and spectral chemical processes, which evolve in cavity, has meaning.

Clinic. Functional changes from side of nervous, cardiovascular and endocrine systems, auditory and vestibular analysers may be observed in case of systematically influence of ultrasound, which intensity and contact duration exceeds MPC.

Persons who long time serve ultrasonic equipment complain to have headache, blackout, general weakness, quick fatigability, sleep disorders, irritability, memory worsening, heightened sensibility to sounds, and fear against bright light; sometimes complaints can have dyspeptic nature.

Bradycardia and hypotonia are marked for the workers up to the end of a working day; bradysystolia, disorder of intracardiac and intrastomach conductivity are determined with the help of ECG. Monocytos, eosinophilia that later turn into eosinophinia are marked in blood. Quite often lowering

of sugar content in blood, hyperproteinemia is determined. All this developments have non-persistent character.

Mentioned symptomatology is expressed strongly in those cases when ultrasound is transmitted not only though air (this concerns low-frequency sound), but through contact also (high-frequency sound).

Vegetative syndrome of asthenia is determined during clinical observance, sometimes diencephalitic abnormalities - loss of mass, subfebrile temperature, paroxysm of visceral attack type, increasing of muscles mechanical excitation, itch – are observed.

Disorders in the form of angiodystonic syndrome, vegetative polyneuritis, vegetative myofascitis of arms, and vegetative vessel dysfunction can develop in operators during a long-period operation with ultrasonic defectoscopes.

As a rule, general cerebral disorders unite with development of vegetative polyneuritis of arms in different extent of expression. This becomes apparent as acrocyanosis, intumescense, hyperhidrosis, lowing of all kinds of sensitivity of short and long gloves type.

Treatment. When asthenic syndrome is present in clinical picture, a prescription of tranquillisers - 0.2 g meprobamat 1-2 times a day, 0.3 g trioxazine 2 times a day - is recommended to patients. 0.05 g ascorbic acid 3 times a day, warm shower, coniferous baths, one hour walks before sleep are recommended parallel with that.

Group B vitamin (B_1 1 ml 6% solution intramuscularly, riboflavin 0.005- 0.01 g two-three times a day during 15 days, injections of cocarboxylase intramuscularly 0.05 g a day during 20-25 days) is necessary to use in parallel with tranquilliser receptions (three times a day) for persons with more marked symptomatology: continuous complaints of asthenic kind, development of neurocircular dystonia.

Vegetative polyneuritis with sensible and trophic disorders needs longer treatment. Massage, ozokerite applications, rhodon baths and all this in complex with intravenous injections of 10 ml 0.5 % Novocain solution, 15-20 injections in total, are recommended for such patients. Treatment in sanatoria and health resorts (Odessa, Khmelnik) gives good results.

Veification of the ability to work. Ability to work in the presence of early, acute marked appearance of asthenisation and vegetative-vessel changes remains on the assumption of regular observation and out-patient treatment. In individual cases a temporary (1 – 2 months) move of patient to work, which is not connected with influence of ultrasound, is recommended.

Reasonable placing in a job that excludes influence of working environment vibroacoustic factors is recommended in case of development of obvious stable neuro-dynamic and neuro-circular dysfunctions, disorders of hearing and vestibular apparatus parallel with adequate treatment in out-patient or hospital conditions.

Preventive measures. It is necessary to check a permissible ultrasound level on working places (110 dB or 0.1 W/cm^2 at the most in the frequency range from 0.1 till 10 MHz), to follow general requirements after methods of check-up and protection from ultrasound influence (designing of automated ultrasonic equipment and units with remote control; providing of ultrasonic equipment with casings and screens; use of individual protective means: noise shields, gloves; 10-15 minutes stoppages over every 1.5-2 working years) for the purpose of prophylaxis of negative ultrasound influence on persons serving ultrasound units.

Persons older 18 years can be permitted to work with ultrasonic equipment. It is necessary to conduct preventive and periodical medical inspections when taking on; 1 time a year with participation of doctors-specialists: neuropathologist, physician, and otolaryngologist. Hearing organ (audiometry) and speech in a whisper are subject to obligatory study. Persons with an impaired hearing or with a threshold-volute organ dysfunction should be transferred to another work.

INFLUENCING OF IONISING RADIATION ON A PERSON'S ORGANISM (RADIATION SICKNESS)

Radiation sickness is a disease that develops as result of ionising radiation in doses exceeding permissible.

Acute and chronic forms of radiation sickness of various levels of weight (with predominance of local or general changes) are distinguished depending upon kind of influence (occurring once and massive or long repeated in relatively small doses) correspondingly.

For the first time, radiation injuries with general and local displays were described for patients who had experienced influence of ionising radiation for medical aims as well as by rontgenologists and radiologists. With time cases of health condition disturbance of people, which made radium preparations, and of those who worked with ionising radiation sources in industry became known. Mass radiation injuries took place after the USA use nuclear weapon in Japan in 1945.

Acute radiation conditions owing to casualties, mainly as a final output of violation of work rules with different ionising radiation sources are described. And today energy of ionising radiation is widely used in different areas of industry, biology, medicine, and agriculture.

Electromagnetic vibrations with small wavelengths, X-rays and beta-radiation, and also flows of alpha and beta particles (electrons), protons, positrons, neutrons and other charged particles can be referred to ionising radiations. This problem became particularly urgent after the emergency on Chernobyl nuclear power station in 1986 due to influence so-caused small doses of ionising radiation on a person's organism. Their ingress on skin or in more deep tissues is possible depending upon a penetrating power of

these particles during external irradiation. Alpha rays and x-rays have the greatest penetrating power; beta rays have smaller penetrating power.

Organism experiences influence of external irradiation only during presence a person in a sphere of radiation influence. External influence ceases in case of radiation disappearance also, and changes in organism can develop as consequences of radiation. Various radioactive materials can appear in organism as a result of external influence of neutron radiation, for example, radio nuclides of natrium, phosphorus etc. In similar cases an organism for some period of time becomes a carrier of radioactive materials owing to what there can begin its internal radiation in it.

Ionising radiation arises also in working process with various radioactive materials — natural (uranium, radium, thorium) and isotopes. Atomic nucleus in radioactive isotopes is unstable. They are capable to disintegrate, to transform into nucleuses of other elements, at that their physico-chemical properties change. This phenomenon is accompanied by a nuclear radiation and is caused a radioactivity, and these elements are radioactive. Radioactive decay is characterized by energy release in the form of gamma-radiation and corpuscular particles of alpha and beta radiations.

The radioactive materials can permeate into organisms of workers through lungs or gastrointestinal channel, as well as through an uninjured skin. Works connected with mining of radioactive ores are in particular dangerous in this respect.

Radioactive radiation not only causes ionisation of air and also results in similar process in tissues of an organism changing them considerably. The expressiveness of possible biological changes depends on a penetrating power of radiation, its ionising effect, dose, radiation time and condition of organism.

Radioactive materials after getting into organism are spread with blood in various tissues and organs and become a source of internal irradiation. Isotopes, which during all life of a suffered person can be source of ionising radiation, are especial threat for organism. Radioactive substances are brought out basically through a gastrointestinal channel, kidneys and organs of breathing. The various kinds of radiation have their own features, unequal biological potency and, thus, unequal degree of safety for people working in contact to them. So, workers can experience influence of X-rays while servicing X-ray apparatuses in medical organizations and technical labs. X-rays are electromagnetic radiation with a very short wavelength and high penetrating power.

People working with x-rays and gamma rays during implementation of gamma-flaw detections on industrial enterprises, maintenance staff of accelerating installations and nuclear reactors, as well as workers exploring and mining mineral resources and so on can experience influence of ionising radiation too. The basic problems of radiation safety are now resolved.

However, ionising radiation under certain circumstances and at violations of safety precautions can cause development of radiation illness (acute and chronic).

Pathogenesis. The basic feature of ionising radiation effect is ionisation of atoms and molecules of living matter. This process is considered as an initial stage of biological influence of radiation, which in future produces functional and organic lesions of tissues, organs and systems. Complicated mechanisms of direct and indirect influence of ionising radiation on organism lie in the basis of radiation illness genesis.

Direct radiation effect (of large doses) on protein molecules results in their denaturation. In result a protein molecule is coagulated and settles from colloidal solution experiencing influence of proteolytic ferments of decay later on. At that a disturbance of physico-chemical processes in cell with a depolymerization of nucleic acids is observed, which is accompanied by structure change of cells surfaces and membranes permeability.

According to a target theory it is assumed that not a whole cell is sensitive to radiation.

In each cell there is a sensitive segment or "target", which accepts effect of ionising radiation. It is determined that especially sensitive to radiation effect are chromosomes of nucleases and cytoplasm.

Indirect effect of ionising radiation is explained by mechanism of water radiolysis. As it is known water makes about 80 % of weight of all organs and tissues of a person body. "Radicals", which have both oxidative and reducing properties, arise during ionisation of water. Atomic hydrogen (H), hydroxide (HO_2), hydrogen dioxide (H_2O_2) has the greatest importance among them. Free oxidizing radicals react with ferments, which contain sulphurhydrilic groups (SH), which transforms in inactive disulphide combinations (S = S). Catalytic activity of important carbothiolic ferment systems taking active participation in synthesis of nucleoproteides and nucleic acids, which play a considerable role in process of organism vital functions, becomes disturbed as a result of these reactions and transformations. Quantity of DNA and RNA in nucleases of cells is sharply reduced, a process of their restoration starts. At that, nuclear biochemistry changes are morphologically expressed by various violations of chromosomes structure and, consequently, of whole genetic system.

Radio toxins that are formed and changes of neurohumoral and hormonal regulation of tissues and cells also make certain influence on a course of biochemical processes in nucleuses of tissues affected by radioactive radiation. Exchange processes upset and that leads to accumulation of such substance unrepresentative for organism, as histamine-like toxic amino acids. All this strengthens biological effect of ionising radiation and assists an intoxication of organism. Tissue intoxication becomes apparent with clinical signs of nervous activity lesion, change of internal

organs functions (achylosis, myocardial dystrophy, hepatopathy, endocrinopathy, and disturbance of haemopoiesis).

One of the leading places in pathogenesis of radiation illness takes a lesion of hemogenic organs. Hemogenic tissue is the most sensitive to radiation, in particular blast cells of bone marrow. Therefore aplasia of bone marrow, which develops under influence of radiation, is a consequence of depressing of mitotic activity of haematogenic tissue and mass destruction of marrow cells.

Abrupt lowering of haemopoiesis activity predetermines development of haemorrhagic syndrome.

In formation of radiation illness a certain meaning has the fact that ionising radiation creates specific injurious effect on radiosensitive tissues and organs (marrow cells of hemogenic tissue, epithelium of testicles, thin intestine and skin) and non-specific irritant effect on neuroendocrine and nervous systems. It is proved that a nervous system has high functional sensitivity even to small radiation doses.

Irritation of external and internal receptors results in malfunction of central nervous system and its highest sectors in particular, that can cause reflex changes of the activity of internal organs and tissues. Thus, a certain meaning is given to endocrine glands, and first of all to hypophysis, adrenal and thyroid glands etc. Capability of arising of reparation-regenerative processes in injured organs beginning from the first hours of radiation attracts attention to itself.

Pathologic and anatomic pattern. Pathological anatomy of the so-called medullar form of radiation illness with prevailing lesion of hemogenic tissue, which develops during effect of ionising radiation in doses up to 100 rad, is studied mostly full. Morphological changes, which are characteristic for this form of acute radiation illness are shown in a stage of latent period yet and become expressed in a period of disease height. Signs of haemorrhagic diathesis are determined macroscopically: haemorrhages in skin, in serous tunic and mucous tunic, as well as in parenchymatous organs. Degree of expressiveness of haemorrhagic diathesis oscillates over a wide range depending on lesion seriousness: additional traumas strengthen phenomena of haemorrhage. Excessive haemorrhages in stomach and intestine, in lungs, adrenal glands and their destruction, large haemorrhages in myocardium that seize a leading system of heart, can have crucial meaning in development of disease. Bone marrow loses a customary consistence and becomes fluid, its colour is determined by presence of blood; lymph nodes look like increased due to haemorrhages tissue retresection. Deep lesions in a hemogenic system determine tendency to bleeding and intensity of development of infectious complications, which as a rule show themselves in period of height of the disease. Ulcer-necrotic

gingivitis, necrotic anginas, pneumonias, inflammatory changes of thin and thick intestine concern them.

In other organs there are signs of lesion of blood circulation and dystrophic changes. Lesions of skin (loss of hairs, and radiation burns) can be shown in case of considerable irradiation: they are united as a rule with thermal burns for persons, who have got within a zone of nuclear explosion.

Outcomes of microscopic examinations testify that hemogenic organs experience the most characteristic changes. They are found out yet in a latency stage: it is possible to see a decay of lymphocytes in lymph nodes during the first hours after radiation in particular in a central part of follicles, that is in a zone of disposition of B-lymphocytes; a little bit later changes arise in a zone of T-lymphocytes. In a period of disease height it is possible in the main to recognize elements of gland stroma and plasma cells on a background of sharp hyperemia. Analogous changes are seen in tonsils and liver. Aplasia develops fast in marrow: to the end of the third day about 10 % of the cell structure remains in it. This is explained by active throwing out of mature forms in a circumferential channel and by termination of division and disintegration of cells. Later mitotic activity renews for some period of time, though the cells, which are divided, perish during mitosis. The fast and considerable exhausting of cells is accompanied as if a vicarious plethora of marrow vessels with gaps of vascular walls and formation of hemorrhage sites. A customary hemogenic tissue almost at all does not remain in a marrow in period of height of an acute radiation illness, elements of stroma and plasma cells prevail. Lesion of a lymphoid tissue and marrow results in decrease of organism immunobiological reactivity and predetermines favourable conditions for development of self-infection complications.

Characteristic changes in sex glands in particular in men develop in a course of radiation illness. In a stage of latency a mitotic division ceases, the epithelium of testicles disintegrates and separate large and colossal degenerated cells arise; there are only separate spermatogones and cells of Sertolli in testicles channels in a period of disease height.

Early changes in mucosa of small intestine, which is highly sensitive to action of ionising radiation, show destruction and depression of cells crypts epithelium mitotic activity. Pathological forms of mitosis appear.

Terminal changes are connected with distresses of blood and lymph circulation, self-infection processes: mucous mucosa is swallen up, there are areas of necrosis and open ulcers, on a surface of which mass of fibrin, slime and colonies of microorganisms are visible; and leukocyte infiltration is almost absent.

Radical changes in a cardiovascular system during an acute radiation illness will be localized mainly in small-sized vessels that matter in development of haemorrhagic syndrome. Morphological characters of

increased vessels permeability (walls swelling, endothelium separation, increasing pervascular swelling) are found out even long before arising of haemorrhages. Diapedesis haemorrhages, diffuse infiltration of erythrocytes and phenomenon of plasmorrhagia through walls of vessels are noticeable in a period of disease height.

Changes, which are connected with violation of blood circulation and inflectional complications, are watched in lungs during acute radiation illness. Among them a so-caused agranulocyte pneumonia, which is accompanied with loss of serous-fibrinogenous-haemorrhagic exudation, formation of sections of necrosis with colonies of microorganisms without perifocal inflammatory reaction, deserves a special attention.

Violation of blood circulation and high permeability of vessels are mainly watched in kidneys during an acute radiation illness: haemorrhages are visible in their tissue; dystrophy changes of epithelium twisted channels can be found out.

Violation of activity of internal secretion glands, which is at first considered as a development of an intensified function, is characteristic for an acute radiation illness; further a relative normalization comes, and signs of a functional exhaustion of glands are shown in a period of disease height.

Classification. A. Radiation illness caused by effect of general external irradiation or radio-isotopes with their even distribution at an organism:

1. Stages: formation of a phase, or I, II, III and IV degrees of weight, recovery of consequences (hypoplastic conditions; hyperplastic processes — leucoses; accelerated involutory processes in vascular, nervous and endocrine systems).

2. Forms: transitional, marrow, intestinal, and toxaemic.

3. Phase or degree of weight: I — primary general reaction; II — latent; III — height of illness; IV — renovation.

B. Radiation illness conditioned by influence of radio-isotopes with selective depositing or local external irradiation:

1. Period of formation of pathological process (preclinical stage).

2. Stage of clinical manifestations and exit of disease (dystrophic and hypoplastic conditions - hypoplastic anaemia; involutory processes - accelerated ageing, pneumosclerosis; hyperplastic processes - leucosis, swellings, canceroid).

The given classification foresees a possibility of both acute, and chronic radiation influence.

Acute radiation illness

The acute radiation illness is a group of clinical syndromes, which develop after short-lived (from several seconds till three days) influence of

penetrating radiation in doses exceeding an average permissible dose for a body, equal 1 Gy (100 Rads).

Acute radiation illness depending on a total level of doses, power and distribution on a body can take place with prevailing lesions of haemopoiesis (1-10 Gy), intestine (10-20 Gy), general haemodynamic and toxaemia developments (20 — 100 Gy) and cerebral violations (100 Gy).

Etiological factors of acute radiation illness are: gamma - neutron, x-ray and gamma-, beta-radiation of atomic explosions, change of regimen of work or violations of rules of work on nuclear energy stationary plants.

An single-stage traumatizing of all organs and systems of an organism takes place in case of ionising radiation operation, and first of all: acute lesion of cells hereditary structures; marrow haemogenic cells; a lymphatic system; epithelium of gastrointestinal channel and skin; cells of liver, lungs and other organs.

Radiation lesion is essentially a trauma of biological structures. Its characteristic depends upon an energy quantity factor that is influence of small doses can be unnoticeable, and ones of large doses can call fatal consequents. Dose power of radioactive influence plays an essential role also: the same quantity of a radiation energy, which is absorbed by a cell, causes the greater lesion of biological structures than more short duration of irradiation.

The large doses of the influence, which are stretched in time, will effect much smaller lesions than the same doses, which were absorbed for a short time.

Thus, basic characteristics of a radiation injury are two factors: *biological* and *clinical*. On the one hand, they are characterized by a radiation dose (“dose-effect”), and on the other hand, this effect is conditioned also by power of dose (“power of dose-effect”).

A clinical picture happens very poorly noticeable; sometimes symptomatology is not shown at all directly after irradiation of a person. Therefore, knowing of a radiation dose plays a central role in diagnostic and early forecasting of acute radiation illness motion, in definition of therapeutic tactics before a moment of development of the main signs of disease.

Clinics. In clinical motion of an acute radiation illness (mainly, marrow forms are distinguished four phases: I — a primary general reaction; II — an imaginary clinical health (latent); III — expressed clinical manifestations (height of illness); IV — renovation.

Phase of the primary general reaction continues from several hours till one - two day; it is characterized by predominance of nervous-regulatory shifts, mainly reflexive (dyspeptic syndrome); by re-distributive shifts in composition of blood (more often by a neutrophyle leukocitosis); by violations of analysing systems activity. Symptoms of direct harmful

influence of ionising radiation on lymphoid tissue and marrow are displayed: fall of lymphocytes quantity, destruction of young elements in cells, appearance of chromosome aberrations in cells of marrow and lymphocytes. There are typical clinical symptoms in this period, such as: nausea, vomiting, headache, increase of temperature, general weakness, and erythema. If the radiation dose is less 1.5 Gy, these phenomena can be absent; they appear under effect of higher doses, and degree of their expressiveness will be as greater, as the dose is more. Nausea that the primary reaction can be limited with in case of appearance of a light degree of the illness is replaced by vomiting; attacks of vomiting repeat many times after increase of the dose irradiation. This dependence is a little upset in case of incorporating of radio nuclides owing to radiation from a radioactive cloud: attacks of vomiting become repeated and steady even at a dose close to 2 Gy. Sometimes sufferers complain of metallic smacks in a mouth.

If doses of external radiation are higher then 4-6 Gy, transient hyperaemia of skin and mucosa, cheeks and tongue mucosa hypostasis appears. Early appearance of rhinopharyngitis, conjunctivitis, and radiation erythema is possible even in course of radiation illness of light degree at radiation from radioactive cloud when hamma- and beta-components effect upon skin and mucosa simultaneously.

Gradually in several hours manifestations of primary reactions fall down: vomiting is ended, headache is finished, hyperemia of skin and mucosas disappears. Patients' feeling becomes better, but an expressed asthenia and a very fast getting tired remain.

Phase of imaginary clinical health continues from 10-15 days till four-five weeks and is characterised with gradual arising of pathology changes (continuing empting of marrow, changes on mucosa of intestines, suppressing of spermatogenesis, progress of skin changes, baldness) on background of general nervous - regulatory violations damping and patients' health satisfactory state.

Phase of expressed clinical manifestations is characterized by deep lesion of blood system, depression of immunity, development of infection complications and haemorrhagic manifestations. Death of ill man from deep violation of haemopoiesis, infection complications (haemorrhagic-necrotic pneumonia), bleeding in this phase of pathological process development is possible. Duration of the III-rd phase in cases of convalescence does not exceed 2-3 weeks. Appearance of young cell forms in marrow punctates is a testimony of a favourable prognosis.

In a period of a *renovation phase* the' general condition of patient becomes better, temperature reduces, haemorrhagic manifestations disappear, tearing away of necrotic mass and suppuration of erosion surfaces on skin and mucosa takes place; a hair growth renews from the 2 - 5-th

months, hidrosis is normalized. As a whole this phase lasts 3-6 months (sometimes 1-3 years).

If the external irradiation was integrated with infiltration of radionuclides inside (and they directly affect mucosa of respiratory routes and intestine), so in the first days after irradiation a fluid defecation can took place (several times a day).

All these phenomena cease within the nearest days, but arise again after a certain period of time.

At that there is another phenomenon connected with radiation injuries besides quantitative interconnections between dose and effect, between dose power and effect: the more is dose, the earlier a specific biological effect comes. This phenomenon means that vomiting, specific to primary reaction, at effect of a higher dose comes earlier; main signs of the illness - radiation stomatitis, enteritis, decrease of leucocytes, thrombocytes, reticulocytes quantity with all their regularities, epilation, skin lesion and so on – comes up as earlier, as the dose is higher. The described phenomenon received a name of relation «dose - time of effect», it plays an important role in biological dosimetry.

During the first days of illness a transient increase of liver can be found out in many sufferers without direct relation to the dose. Disintegration of marrow red cells can cause small icteritiousness of scleras and increase of an indirect bilirubin level in blood, but these phenomena shortly disappear.

Directly after irradiation a neutrophylic leukocitosis, which expressiveness does not depend upon a dose, is marked in a majority of sufferers. Further changes in a picture of blood evidently depend on a radiation dose, first of all dynamics of leucocytes content is rather peculiar. This can be imagined in view of a curve: at doses less 5 Gy quantity of leucocytes step-by-step drops to 7-12-th day (the higher is a dose, the earlier the curve reaches minimum primary fall), and then it is again increases remaining below a normal level as usual. Term of increase depends on the dose: the dose is higher, and the term is shorter. This growth of leucocytes received a title of abortive rise, it is finished by the main fall of leucocytes quantity, when an agranulocytosis comes on the 1^{st} - 2^{nd} week, fall of leucocytes quantity makes below than 1000 in 1 mcl. Then haemopoiesis renews at radiation doses smaller than 6 Gy. Content dynamics of both thrombocytes, and reticulocytes is similar with a leukocyte curve. Parameters of the leukocyte curve have a great importance in a biological dosimetry. The fall of lymphocytes quantity has evident significance only in the first 2-3 days after radiation, and then this dependence becomes less visible.

A clinical picture of an acute radiation illness, which is caused by external radiation, consists not of only from a lesion of blood system and secondary pathological processes mainly infectional-inflammatory caused by depression of haemopoiesis.

Lesion of epithelial covers conducts to the responsible violations: stomatitis, enteritis, gastritis, proctitis, and hepatitis appears.

Dryness in a mouth comes before the first week ending after irradiation with dose 4-5 Gy, spit becomes viscous and is hardly separated, cracks and then necrotic raids appears on mucosa of an oral cavity.

Lesion of oral cavity mucosas develops in itself and can precede to agranulocytosis, as at these doses the main and deep decrease of quantity of leucocytes is realized approximately on 12-20-th day (the higher dose, the earlier). With development of agranulocytosis a condition of mucosas gets worth, recovery is detained, infection complications - pneumonia, angina and so on - develop. Weight of condition is complicated by a haemorrhagic syndrome caused by a deep thrombocytopenia, development of syndrome of disseminated intravascular conglutination. Changes in mucosas of oral cavity, nasopharynx are conditioned not only by radiation injury directly them, but also by radiation injury of salivary glands, which the first signs develop in case of irradiation of submandibular site with a dose above 5 Gy. Due to this lesion a salivation almost absolutely ceases, a sharp dryness of oral cavity mucosas appears, xerostomia, which lasts some days and very disturbs ill persons. Xerostomia becomes irreversible in case of salivary glands irradiation with a dose of 10 Gy and more.

Signs of radiation necrotic enteropathy appear in period of agranulocytosis with doses 4-5 Gy: temperature of a body is increased up to febrile, often up to 40 °C, infrequent fluid defecation occurs, abdominal distention, and capotement and babble at palpation are determined in ileocecal zone. Necrotic enteropathy in grave cases can be accompanied by high-gravity diarrhea, invagination, rupture of intestines and peritonitis. Necrotic enteropathy develops before agranulocytosis, if under any cause a radiation dose on intestine has exceeded the general dose given above.

Sensitivity of various segments of gastrointestinal tract to ionising radiation influence is unequal: ileocecal department of intestine is easily struck, empty intestine suffers less. Radiation gastritis as against enteropathy arises in case of the above mentioned doses operating in 1.5-2 months after irradiation, when agranulocytosis has already passed for a long time, remained inflammatory processes have stopped, and temperature became normalized.

Radiation proctitis with such clinical symptomatology as tenesmuses during normal defecation, standard temperature, and absence of pain in anus zone can appear for a short time approximately at the end of the second month. In several days tenesmuses pass. Radiation esophagitis, which can

be developed at the end of the second month, is characterized by availability of difficulties during swallowing, pain during passing of food in particular hard.

All these lesions are referred to categories of primary ones, in other words conditioned directly by radiation factor operation. On a background of these primary violations there are various secondary processes: phlegmons, infected erosions, acute tonsillitis, pneumonia, inflammation of other organs. Radiation hepatitis develops approximately after three months. It is characterized by such features: temperate hyperbilirubinemia, high activity of aminotransferases, and regimenrate increase of liver and often expressed itch of skin.

Symptomatology of progressing lesion of central nervous system characterizing by loss of consciousness can arise in case of doses irradiation larger than 10 Gy. Patients perish at phenomena of cerebral coma. Thus, if тanausea occurs and single vomiting attack is probable at acute radiation illness of light degree in some hours after irradiation, then expressed primary reaction, which is shown with vomiting in 1-3 h after irradiation at acute radiation illness of a medium degree of weight, is already marked. Term of appearance of primary reaction manifestations decreases in process of illness progress. So, the primary reaction at acute radiation illness of high weight degree comes already in 30 minutes, and at a very high weight degree it comes less than in 30 minutes and has high weight, irresistible nature. This stage of acute radiation illness is manifested in several clinical forms depending on doses level: transient, intestinal, toxaemic and nervous.

Transient form: the primary reaction lasts 3-4 days; enterocolitis, enteritis, fever can appear from the 6-8-th day. General course of the disease is weight; convalescence is possible only at case of treatment started in proper time.

Intestinal form: the primary reaction is grave and continuous; development of erythema and fluid defection is watched; expressed changes of oral cavity and pharynx mucosa appear during the first week, temperature is subfebrile, defecation is normalized; acute aggravation of state of health comes on 6-8-th day of the disease: fever (up to 40 ºC), grave enteritis, dehydration, haemorrhage, infection complications.

Toxaemic form: the primary reaction arises directly after ionising factor action, short-lived collaptoid condition without consciousness loss is possible; acute intoxication, hemodynamic violation (weakness, arterial hypotonia, tachycardia, oliguria, azotemia) are developed on the 3-4-th days, general brain and meningitis symptoms (brain hypostasis) appear from the 3-5-th days.

Nervous form: collapse with a loss of consciousness directly after irradiation is possible, after return of consciousness wearisome vomiting and diarrhea with tenesmuses appear in the first minutes after influence (in

case of collapse absence); consciousness is upset later, signs of brain hypostasis arise, arterial hypotonia and anuria progresses; death comes on the 1-3-rd day at phenomena of brain hypostasis.

Treatment and measures for rendering of the first help. Radiation injury by its own is not a subject of emergency treatment since it is impossible to influence a little effectively on process of acute radiation illness, when all processes are already started up by effect of irradiation. However, it is necessary to perceive that various emergency conditions determine various kinds of lesions, and various protective measures against further irradiation of people.

In case of emergencies arising on experimental reactor blocks when irradiation is distinguished with a very fast formation of critical mass, a large neutron flux and gamma-radiation, when irradiation of organism of sufferer lasts a very short time, staff must immediately leave the reactor block. Everyone who was in the room must be sent to a first-aid post or medical unit (if it is located in this territory) irrespective of state of health. In case of a rather high-gravity degree of lesion weight a vomiting attack can begin in some minutes after irradiation and moving in a car will instigate its originating. Taking this into consideration, if a hospital is placed far from emergency location, it is necessary to move sufferers there after a primary reaction termination, having kept them in cabinets of the medical unit for a period of vomiting. Sufferers with lesions of a high-gravity degree must be placed in separate cabinets aiming do not allow a view of vomiting of one sufferer could instigate vomiting of others.

All sufferers should be transported in specialized clinic after vomiting ending.

Actions of medical staff in case of emergency originated at industrial units with emission of radioactive gases and aerosols should be some others. At first, all staff of the hall and nearest rooms should as soon as possible abandoned by them, as each superfluous second of stuff stay in a cloud of aerosols and gas radiation influences on sharp increase of the dose. It is a lot of isotopes of radioactive gases and aerosols, which a half-life makes some seconds. It explains various degrees of injuries of persons, who were close one to other, but had a small difference in duration of staying there. All staff should know about danger to take in hands any things, which are in emergency room, and to sit there on something. Contact with things, which are very much contaminated with alpha-, beta-radiation, can result in local radiation burn.

All staff of an emergency room in case of emergency must immediately put respirators, immediately accept a tablet of potassium iodide (or drink three drops of tincture of iodine diluted in glass of water), as a considerable activity of radiation is a share of radio iodine. Customary iodine goes in a thyroid gland and is absorbed by its cells. The cells

saturated with the customary iodine do not accept a radio iodine any more. After sufferers have left an emergency room they must be carefully washed with soap under douche also dressed in other clothing. Clothing, which was on them, should be accepted and given to radiation monitoring. Problem of duration of washing and shearing hair should be decided depending on results of the radiation monitoring. Adsorbent should be given to all radiated. Occurrence of diarrhea in a short time after emergency is connected with a reception of potassium iodide (it can provoke diarrhea for some people). However, as a rule, diarrhea during the first days after irradiation from a radioactive cloud is produced by radiation injury of mucosa of gastrointestinal tract.

The first aid at radiation injury of a high-gravity and rather high-gravity degrees can be necessary due to appearance of primary reaction, which expression of manifestations is not peculiar to primary reaction in case of operation general irradiation of the light and medium degree of gravity. Repeated attacks of vomiting starting every 15-30 minutes after irradiation refer to such manifestations first of all. They are needed to be interrupted by injection 2 ml (10 mg) of Cerucalum (Reglanum) intramuscularly or intravenously.

Medicine should be injected intravenously or through dropper, or very slowly (10-30 minutes), that increases its efficiency. 0.5 ml of 0.1 % of atropine solution should be injected subcutaneously or intramuscularly to reduce vomiting attacks. If vomiting becomes irresistible due to hypochloremia, which develops on this background, 30-50 (up to 100) ml of 10 % hypertonic solution of chloride natrium salt should be injected intravenously through dropper. After that it is necessary to prohibit sufferer a reception of any liquid for some hours. Next saline solutions should be prescribed with the purpose of dehydration elimination caused by repeated vomiting: isotonic solution of sodium salt (500-1000 ml) intravenously or as a last resort subcutaneously, or 500-1000 ml of solution “Threesol” (5 g of sodium salt, 4 g of soda and 1 g of potassium chloride per 1 litre water; sometimes it is caused solution 5:4:1), or 1000 ml of 5% solution of glucose with 1.5 g of chloride of potassium and 4 g bicarbonate natrium.

Neuroleptics and sedative drugs should be used in case of total irradiation by a dose 10 Гр for reduction a vomiting and тanausea, which are developed even at low power irradiation. Aminazine and fenobarbitalum are applied more often. These drugs are injected repeatedly. Nevertheless it is necessary to remember that application of the said drugs outside of a hospital is excluded as a constant check after a level of arterial pressure, which can be lowered without their application, is required.

Liquids in this period are injected each 4 h till 1 l, then each 8 h after 24 h of such regimen, alternating solution “Threesalt” and 5 % solution of glucose with chloride of potassium and soda.

Injection of liquids reduces intoxication caused by massive decay of cells. With the same purpose plasmapheresis with replacement of plasma for saline solutions is applied at a rather high-gravity degree of primary reaction.

Cell decay can cause a syndrome of intravessel coagulation, that is a blood clotting, its quick conglutination in a needle during fulfilment of a vein puncture or appearance of haemorrhagic eruptions in a hypodermic cellular tissue despite of an initial normal level of thrombocytes. In this case a jet injection of chilled plasma (60 drops per one minute) 600-1000 ml is recommended, injection of heparin (intravenous in drops at a rate of 500-1000 units/h or 5000 units under a skin of an abdominal wall triply per day), as well as plasmapheresis.

The rather high-gravity degree of acute radiation illness can be accompanied by development of collapse or shock, gloominess of consciousness owing to brain hypostasis. Sometimes a forced injection of liquid can be enough at collapse caused by reallocating of liquid in tissues and by hypervolemia, for example, saline solution or 5 % glucose solution at the rate of 125 ml / minutes (only 1-2 l), and intramuscular injection of cordiamine (2 ml). 0.5 mls 0.1 % atropine solution are injected in case of bradycardia. Rheopolyglucin can also be used for elimination of hypervolemia; it as disaggregant reduces hypercoagulation also. However rheopolyglucin at brain hypostasis should be used carefully, as it can enlarge hypostasis. Diuretic drugs (40-80 mg of lasix intravenously or intramuscularly) are used in case of brain hypostasis presence; the drug is injected under arterial pressure watch. For elimination of brain hypostasis an intravenously injection of 60-80 mg prednisolon is allowed. Hypertonic glucose (40 %) solution should be used with this purpose carefully, it can enlarge brain hypostasis (at the expense of hypervolemia increasing).

It is necessary to conduct antishock measures in case of shock development: intravenous injection of large doses of prednisolon up to 10 mg/kg or hydrocortizone up to 100 mg/kg, antishock liquids under a central venous pressure watch (norm is 50-120 mm of water column), 5-10 % albumin solution from 200 up to 600 ml, dopamine (under arterial pressure watch).

The main care in treatment of sepsis and septic shock is to neutralize microflora that caused it. During the first some days it is necessary to inject parenterally large doses of high active antibiotics of a broad spectrum action (from a group of semi-synthetic penicillin or cephalosporins and aminoglycosides), then when an exciter will be detected, drugs of directional effect; large doses of penicilline in case of appearance of pneumococcal sepsis; at a pyocyanic sepsis: carbenecilline (30 g a day) tgether with aminoglicosides (gentamicin or kanamycin 240 mg or 3 g a day accordingly); at a staphylococcal sepsis: cephamesin till 4-6 g a day. At the

same time it is necessary to inject intravenously gamma globulin in a dose of 1 g/10 kg single time during 7-10 days. Plasmapheresis is used for treatment of sepsis as it is capable to actuate phagocytosis.

Local abscesses, more often segments of necrosis, can be arrested with use of applications of 10-20 % solution of dimexid with antibiotics 4 times a day, first of all for those patients, whom microflora extracted from necrosis segments is sensitive for.

First of all it is necessary to adhere to a regimen of full starvation in case of development of necrotic enteropathy as complication of agranulocytosis or as independent process - intestinal syndrome - caused by radiation injury of small intestine. Saline solutions are introduced intravenously with help of dropper. Intensive parenteral antibiotic therapy is carried out for of infection contamination suppression.

At haemorrhagic syndrome a thrombocyte mass is introduced in four doses (dose is $0.7 \cdot 10^{11}$ cells), total about $3 \cdot 10^{11}$ cells per one procedure two times a week and more often, if necessary. If a bleeding have appeared, it is necessary with jet (60 drips one minute under watch of central venous pressure) to inject 600 - 1000 ml of fresh frozened plasma, and to apply transfusion of thrombocytes also.

Radionuclides thrown out in air, mainly caesium, plutonium, strontium, radium, that can result in lesion of gastrointestinal tract, liver and in development of tumoral process in these tissues (far consequences) in case of arise of acute radiation illness owing to emergency on reactors or atomic bomb burst effect on sufferer side by side to external beta-radiation or neutron radiation. It is necessary for warning consequences of these matters penetration into tissue, in blood for reduction of irradiation, which they will cause at delay in tissue, to apply such methods as lavage of lungs, and also injection of substances that bind radionuclides of the so-called chelates. The chelates are applied as follow: pentacyn for plutonium, cerium; ferrik ferricyanide for cesium.

Urgent measures in case of acute irradiation are as follow: it is necessary to take a sufferer from a contaminated room or from a hypocenter of irradiation to a distance exceeding 2000 m, to prohibit to sit on subjects, which are placed in room contaminated by radionuclides, or to touch them, to exchange clothing, to wash his/her body, if influence radioactive materials was with beta- or alpha - activity.

Term of discharge from hospital in case of absence of focal lesions, as a rule, does not exceed 2-3 months from a moment of irradiation; return to labour at with acute radiation illness of the II – III-rd degrees of gravity is possible in 4-6 months. Stay in sanatoriums and subsequent dispensary observation after treatment in hospital is recommended.

Chronic radiation illness

The chronic radiation illness is a complicated clinical syndrome, which develops in case of continuous influence of irradiation in doses exceeding permissible.

Its characteristic signs are:

- duration and wave-likeness of its process;
- availability in a clinical picture both signs of organism lesion from irradiation action, and manifestations of recovering and adaptation reactions.

Three terms are distinguished in development of a chronic radiation illness according to existing classification: 1 - term of formation, or exactly chronic radiation illness; 2 - term of recovery; 3 - term of consequences of radiation illness.

The disease is characterized by slow development; leading symptoms are changes in nervous, cardiovascular and endocrine systems, hemopoietic system, gastrointestinal tract, liver, kidneys; violation of exchange processes arises.

Development of chronic radiation illness is possible depending upon routes of entry: 1 - caused by influence of general external radiation or radio-isotopes with their even distribution in organism (4H, 37Cs, 24Na etc.); 2 - caused by influence of isotopes with selective deposition 226Ra, 86Sr, 210Po etc.) or local external irradiation.

Chronic radiation illness caused by general irradiation is met in persons, who have experienced influence of ionizing radiation during three-five years and have received one-time and summary doses that exceed permissible.

Clinic. The term of formation of chronic radiation illness passes in the form of series phases that are reflected by concept of a gravity degree. Four gravity degrees of chronic radiation illness are distinguished.

Chronic radiation illness of the I degree is characterized by early development of functional coming back violations that have not specific nature. Main complains of patients include general weakness, poor health condition, headache, fall of capacity for work, deterioration of appetite, violation of dream. Patients in this stage are emotionally labile; stable red dermographism, trembling of fingers and general hyperhydrosis are marked.

Symptoms of neurocirculatory dystonia after hypotonic type, tachycardia, sinus arrhythmia are shown on a background of astenovegetative syndrome.

One of constant symptoms is functional violation in gastrointestinal tract in view of dyspeptic phenomena, intestine dyskinesia and gall channels, chronic gastritis with lowering of secretory and motor functions of ventriculus. Signs of capillary tubes penetration increase can also be watched. That is determined by positive Nesterov test. Violation of

endocrine glands function takes place, impotency is marked in men, violation of ovarial-menstrual function is marked in women. Quantity of leucocytes in blood is changed with tendency to leucopoenia, toxic granity of neutrophils, and thrombocytopenia is watched.

Chronic radiation illness of the II degree is showed through further development of astenovegetative violations and vascular dystonia, depression of function of hemopoietic system and haemorrhagic manifestations. At patients an expressed asthenic syndrome, which is accompanied with headache, temporary insanity, hyperexcitability and emotional lability, decrease of memory, lowering of sexual feeling and potency, is marked. Trophic violations become more expressive: dermatitis, exfoliation of nails.

Steady hypotonia with prevailing decrease of diastolic pressure, low voice of cardiac tones is marked on a part of cardiovascular system; phenomenon of myocardial dystrophy is marked on electric cardiogram. Haemorrhages in skin and mucosa, haemorrhagic gingivitis and stomatites, nose bleedings are watched.

Rough fall of leucocytes level (up to $2.0 \cdot 10^9$/l) that is accompanied by neutropenia and lymphocytopenia takes place on the part of peripheric blood. Hypoplasia of all kinds of haemopoiesis is marked in bone marrow.

Chronic radiation illness of the III degree is characterized by high-gravity, irreversible changes in organism with full loss of tissue regeneration capabilities. Patients are very asthenic, they complains on considerable general weakness, permanent headache, nausea, vomiting, poor sleep, often bleedings, memory impairment. Haemorrhagic phenomena, necrotic processes on mucosas arise. Massive loss of hair, and signs of high-gravity necrotic gingivitis are observed.

Patients complain on short wind, palpitation attacks and dull aches in heart zone. Heart borders are increased, dead tones are listened.

On ECG, there are deep dystrophic changes in a heart muscle. Appetite badly becomes worse, and that is tied up with dyspeptic distresses. Quantity of leucocytes (up to $1.0 \cdot 10^9$/l) in blood badly fall with absolute neutropenia and lymphocytopenia. Quantity of thrombocytes (up to 3 g/l) is considerably reduced. All blood cells are degeneratively changed. High-gravity hyperchromatic anemia develops. Addition of inflammation process can result in development of panmyelophthisis that creates a capability of development of a high-gravity sepsis.

As to the IV degree of the disease, it represents a terminal period.

Term of recovery starts after termination of irradiation; it lasts 2-3 years.

Changes on the part of blood in this term are met seldom, are expressed faintly. Secretory function of digestive glands renews; recovery of functions of cardiovascular and nervous systems takes place during more

continuous term (4-6 years after termination of contact with radiation). Late organic lesions of nervous system and accelerated lens age involution, as well as eyes vessels (angiosclerosis of retina and cataract) are sometimes possible.

The most dangerous consequents are: hypoplastic conditions both dystrophias; hyperplastic and blastomatous processes - leukoses, swellings; accelerated involutory processes in vascular, nervous and endocrine systems.

Clinic of chronic radiation illness caused by radioactive isotopes entry in organism. Symptoms of asthenization, as well as signs of haemorrhagic diathesis and changes in a haemopoiesis system are showed first of all in a clinical picture at presence of incorporated substances in organism. In general a clinical symptomatology of this form of radiation illness differs on a background of organism asthenization, arterial hypotonia and moderate leukopenia with singularity of vegetative - vascular violations.

Research of small radiation doses influence on a man organism has taken a large importance today. Topicality of this problem is illustrated by the fact that owing to action of small doses there is a rather definite group of diseases, which demand directional medical measures, and also it gives possibility to evaluate degree of safety of medical irradiations: diagnostic and medical. Irradiation in according with outcomes of scientific researches strikes more tissues, than it was possible to conclude on ground of clinical picture of an acute radiation illness.

Development of swellings in some tissues testifies their radiation sensitiveness only in many years after radiation influence.

Diagnostics. Diagnostics of chronic radiation illness is difficult, in particular, at initial symptoms, as any of available symptoms has no specific features. At diagnosis statement a large attention should be given to sanitary - hygienic characteristics of working conditions, occupational anamnesis, development and features of clinical picture.

Treatment. Staying in open air, medical gymnastics, valuable meal, vitaminization are prescribed in case of availability of precursory symptoms of illness. Bromum, calcium, Glycerophosphatum, Phytinum, Pantocrinum, and also means, which stimulate haemopoiesis (hepatoflavin, Natrium nucleinicum or Leucogenum) are recommended from medicinal means. Stimulators of haemopoiesis (hepatoflavin, Pentoxylum, Natrium nucleinicum), antihaemorrhage means (Acidum ascorbinicum, vitamines B6, P, K; and calcium drugs), anabolic hormones (Nerobolum) are used at radiation illness of the II degree. Main attention is given to struggle with hypoplactic conditions of haemopoiesis (repeated hemotransfusions and transplantation of osteal brain), infection complications, trophic and exchange lesions (hormonal drugs, vitamins and blood substitutions).

Removal of radioactive incorporated matters remains very complex. So, alkali, diuretic and adsorbing drugs are used in the case of availability of uranium wreckages in organism. In addition, special diets are recommended: alkaline one - in a case of uranium incorporation, magnesium one - in a case of strontium incorporation. Complexones (calcium tetacinum and pentacinum) are prescribed for isotopes linkage and acceleration of removing.

Expertise on capacity for work. Temporary discharge from work bound with influence of ionizing radiation is recommended for about one year at initial developments of the disease. Assigning of patient to medical experts commission to establish a degree of loss of occupational capacity for work and labor recommendations is given in case of availability of more expressed illness manifestations. Further labor activity in contact to this factor is counterindicative.

Preventive measures. Rational organization of work, observance of radiation safety norms is necessary. All kinds of work should have an effective screening. It is necessary to adhere to storage instructions and carry of ampoules using containers, manipulators with work with protected radiation sources. Radiation monitoring, realization of preventive and periodic medical examinations, and also observance of medical contraindications concerning people, which are taken to work with radioactive materials, are of great importance.

OCCUPATIONAL DISEASES BOUND WITH ATMOSPHERIC PRESSURE CHANGES

Altitude sickness

The altitude sickness is a disease that results from a considerable and fast decrease of partial pressure of oxygen ($p0_2$) in ambient gas medium.

In 1918 Schneider had offered to aggregate pathologic conditions that arise in time of flight and climb up an altitude in a unified nosological unit that have received a title of altitude sickness. It originates in pilots, and also in people, who work on high-level regions.

Etiology and pathogeny. Main cause of altitude sickness originating is an acute oxygen deficiency. Oxygen deficiency development is predetermined by reduction of barometric pressure with obligatory fall pO_2 in air or decrease of oxygen contain in air or in man-made gas medium of hermetically sealed rooms. The first situation can arise during high-altitude flights on flight vehicles with cabins of open type or after lesion of air-tightness of cabins of a closed type; the second one is owing to failure of systems that regenerate air in hermetically sealed cabins and rooms.

Adaptive reactions directed on improvements of oxygen transportation to cells, and pathological reactions conditioned by oxygen deficit, are closely

interweaved in pathogenesis of altitude sickness. It can be considered in such sequence. Deficit of oxygen in environment results in decrease of partial pressure in alveolar air and arterial blood. Lowering of partial pressure in arterial blood causes in turn and irritation of chemoceptors of reflexogenic vascular zones (sinocarotid and aortal). Amplification of impulses of chemoceptors is the beginning of many reflex adaptive reactions that determine increase of a minute volume of blood, stimulation of hypophysis-adrenal system and above-vesiculate formations of brain, including cerebral cortex. Development of hyperventilation results in acapnia originating. It plays a certain part in pathogenesis of altitude sickness, and therefore it can be a cause of lesions of blood circulation and breathing regulations. Changes in activity of central nervous system that are showed in view of sensory and motor lesions arise due to considerable deficit of oxygen in arterial blood on the background of adaptation reactions. At those structures that are the most sensitive to oxygen deficiency in blood suffer first of all: photoceptors of eyes' cellular tissue, cortex of cerebral hemispheres, cerebellum.

Clinic. Two basic forms of altitude sickness are marked out: collapse and unconscious.

The collapse form of altitude sickness originates practically at 3 % of able-bodied people in 5 - 30 minutes after altitude-chamber ascent on an altitude of 5000 m. It originates in 25 % cases for persons with functional failure of cardiovascular system regulation, and at 10-15 % cases for a practically able-bodied people after ascent on the altitude of 6000 - 7000 m. At that a general weakness, feeling of fever in all body or only in a head occurs, vision changes, air deficiency is felt, giddiness and loss of consciousness come up. Exterior of ill person, his/her behavior changes: paleness of face dermal cover comes up, sweating increases, features of face are sharpened, and it takes a suffering view. Motion activity is increased at first, and then delayed; a pose becomes constrained, a look is long, fixing on separate subjects. Attitude to surroundings becomes indifferent. Consciousness remains saved for continuous time, but all instructions of doctor are performed slowly and as if reluctantly. If a sufferer will not be supplied with a normal oxygen feed, his/her) condition can sharply worsen - a loss of consciousness will be set in. Frequency of cardiac contractions becomes less often, arterial pressure is reduced, and that testifies a development of collapse form of altitude sickness.

Unconscious form often arises without any precursors. Ill person does not feel unpleasant sensations, loses feeling of adequate attitude to external situation and own condition, the loss of consciousness comes suddenly. In some cases attacks of clonic cramps precede to consciousness loss.

Loss of consciousness at this form of altitude sickness refers to group of homeostatic unconsciousnesses, as its cause is hypoxemia - considerable decrease of blood saturation with oxygen.

At that a cerebral blood circulation in some time after loss of consciousness remains on a rather high level, therefore a renewal of normal supply of organism with oxygen results in recovery of consciousness and disappearance of all symptoms of altitude sickness within 10-20 seconds.

Treatment. It is necessary to transfer a sufferer with altitude sickness to breathing by oxygen or mixture of oxygen with 3-5 % contents of carbon dioxide; it is the only reliable method of this disease treatment. The oxygen therapy for fast and full recovery of health in light cases is sufficient.

Except for the oxygen therapy it is necessary to use a medicinal therapy at high-gravity forms of altitude sickness, if a sufferer is unconscious during continuous time or if a loss of consciousness arises multiply times and is accompanied by attacks of cramps, vomiting. Citramonum, caffeine, camphor, cordiaminum, strophanthin, lobeline or cytitonum are prescribed with this purpose. Drugs with dehydrational properties (mannitol, dextrane, and glucose) are recommended for a preventive measures and elimination of posthypoxic brain hypostasis.

Heat exchange. It is necessary to take into consideration nature of changes and feature of work at solution of problems, connected with capacity for work. Experiencing of light forms of altitude sickness that has not resulted a health condition in nonperishable negative changes later on is not contraindication to work on a profession.

Expressed and nonperishable changes result in disablement. Medical social commission of experts determine a degree of decrease of capacity for work, solve a problem concerning necessity of person transfer to disablement, give recommendations concerning a training for a new profession with allowance for degree of manifestations of occurred changes.

Preventive measures. The most effective way of preventive measures of altitude sickness is usage of oxygen equipment that supports normal entry of oxygen in organism. It is necessary to perform trainings in conditions of an altitude chamber: regular ascents up on the altitude, which increases step by step (from 3 000 up to 5 000 m), and also under high-level conditions for increase of resistance to altitude sickness. Preliminary and periodic medical examinations of aircrews are of great importance. Contraindications to ascent to altitude are any lesions in a central nervous system, hypophysis and endocrine disorders, cardiovascular diseases, organs of touch and alimentary glands.

Decompression sickness

Some technology processes are carried out under conditions of heightened atmospheric pressure. For example, a drifting of horizontal and vertical underground excavations through watered seams or fulfillment of work under water that is possible only under condition of water forcing out from an air working chamber using compressed air. Pneumatic work is

performed in special units named torsion boxes and they are most widespread at building bridges and dams, foundations under various facilities, tunnels, undergrounds, in coal and mining industry and so on. Influence of heightened atmospheric pressure is testing with help of divers and scuba diving.

The caisson disease is a pathological condition that develops owing to formation of gas bubbles in blood and tissues in case of decrease of external respiration (in a man on leaving caisson and emergence).

Etiology and pathogenesis. Caisson sickness is a consequence of transition of gases of blood and tissues from dissolved condition in free one – similar to gas - in case of decrease of environment atmospheric pressure. At that, gas bubbles are formed, they destroy normal blood circulation, stimulate nervous endings, deform and damage tissues of organism. Main part of general pressure of gases in lungs and consequently blood and tissues falls on a portion of nitrogen, a physiologically inert gas that does not take participation in gaseous exchanges. High partial pressure of nitrogen in lungs, its physiology and non-reactivity predetermine its basic role in formation of gas bubbles in case of decompression development. Term of dynamic balance recovery for various tissues of organism is unequal at change of nitrogen partial pressure in external and alveolar air. Blood, lymph and tissues, which perfuse well, are saturated faster and destroy it.

Dynamic balance of gas becomes broken, tissues and liquid of organism become oversaturated with gases first of all by nitrogen at lowering of pressure of environment (when a worker leaves a caisson box, at ascent from depth onto surface). Process of excess nitrogen removal from tissues before arrangement of a new gas balance at sluggish decompression usually flows without formation of gas bubbles. Oversaturation of tissues with gases reaches a critical level in case of fast decompression. Conditions for bubbles formation in tissues and liquids are formed. There are two basic types of bubbles. The first on include bubbles located outside of vessels, formation and return development is determined by process of diffusion - exchange of gases between a bubble and medium that surrounds it. The bubbles located inside tissues, definitely refer to this type. They are capable to enlarge and press on tissues that surround them, causing their deformation, and that invokes sensation of pain in patients. Mechanism of development of sensations of muscular-articular decompression pain at patients has such characteristics.

The second type involves gas bubbles, evolution of which is conditioned not only by processes of diffusion, but also by junction of one bubble with another or, to the contrary, by its splitting into even finer bubbles. They join one another, being formed in venous channel, that gives possibility for acute aeroembolism development in circulatory system.

Pathologic and anatomic picture. The most expressed and specific morphological manifestations in case of fast death from a high-gravity

decompression disease are availability of numerous bubbles in venous system, a right half of heart overflowed and spread by gas bubbles, phenomenon of edema and emphysema of lungs, numerous zones of hemorrhages in various organs and tissues.

Clinic. Three degrees of gravity of a decompression sickness are marked out: mild, mean and high-gravity.

Itch of skin, eruption, non-acute pain in muscles, bones, joints and along nerve trunk is characteristic for the *mild* degree. More often, continuous pain arises in one or several joints of extremities, in particular in knees, shoulders, and also in radiocarpal, elbow joints and ankles. The pain has no concrete localization. Most of all it is felt around of joint, being diffused to all directions from it. The pain, as a rule, strengthens at palpation of joint and bending of extremities. Joints and muscles experiencing the greatest physical loadings are involved in the process most often.

Itch of skin is felt on a body or on proximal segments of extremities. It reminds itch of skin after a bite of an insect.

Some portions of skin have mottled pattern due to skin vascular embolism. Gas accumulation in hypodermic gives start to development of hypodermic emphysema.

The disease of the *mean* degree of gravity is characterized by disease of an internal ear, gastrointestinal tract and organ of sight. First of all syndrome Menyera is formed as a result of gas bubbles origination in labyrinth of internal ear. Acute weakness, gravity and headaches are watched in a clinical picture. These signs integrates with a loss of consciousness, vomiting, buzzing in the ears, and decrease of hearing. Strong paleness of dermal covers, heightened hidrosis appears. Patients complain that all subjects are revolved before eyes; a minor turn of head strengthens agonizing sensations. There is a possibility of consciousness loss. Gastrointestinal lesions are characterized with accumulation of gas in intestines, vessels of mesentery and are accompanied by arise of strong abdominal pain, often defecation. Palpation of abdomen is agonizing; it is strained. Visual acuity is reduced and accompanied by dilatation of pupils and oppression of their reaction on light.

The high-gravity degree of caisson sickness is met today seldom. It is characterized by formation of emboluses in vessels of central nervous system, heart and lungs. Patients complain on general weakness and weakness in legs, sharp coughs, strong pain in thorax, in particular at breathing, asphyxia. Clinical signs of oedema of lungs occur in due course. A significant amount of gas bubbles of different size that produce lesion of cardiovascular activity is accumulated in cavities of right heart and in vessels of lungs in case of originating of multiple aeroembolism. Thus paleness, strong weakness, often and surface breathing is marked in patients: arterial pressure drops. Pulse falls down, dermal covers gain cyanotic tint. Loss of consciousness can be set at expressed phenomena of hypoxia. Myocardial

and lung infarction is probable. The cerebral lesions are conditioned by gas emboluses in brain. Weakness, headache arises after a short-lived latent period. Sensitiveness of one half of body disappears in light cases, and phenomena of paralysis arise in more gravity cases: speech is lost; signs of facial nerve paresis and paraparesis of lower extremities appear. It is accompanied by distress of urination and defecation.

The chronic decompression disease is determined. Two forms of it are marked out: primary and secondary. The primary chronic decompression sickness develops slowly. Deforming osteoarthrosis is the main clinical manifestation of this form.

The secondary chronic form represents a complex of pathological changes owing to experienced acute caisson sickness. Its main clinical symptom is aeropathic myelosis and Meniere's syndrome.

At chronic form of the disease, gas embolas are localized in different organs, mainly in bones. At first the clinical picture flows without symptoms both permanent pain symptom and lesion of function of extremities arise only at complication of the process by deforming osteoarthrosis. Head and proximal ending of diaphysis of thigh are violated at the first turn. After that, head and upper part of diaphysis of shoulder, then distal parts of thigh, proximal endings of shinbone, lower endings shoulder and radial bones are struck.

Diagnosis of caisson sickness is established on a basis of the characteristic complaints and clinical symptomatology that come up after decompression. Occurrence of dermal itch, pain sensations, Meniere's syndrome, paralyses, sudden development of collapse - all this with allowance for the preceding decompression is a direct evidence of caisson sickness.

Treatment. A radical method of a caisson sickness treatment is recompression that influences patient by heightened pressure in a recompression chamber. The method is based on the fact that gas bubbles located in patient’s organism decrease their volume and solve at recompression. The recompression renders assistance for dissolution of the bubbles, that is it eliminates etiological factor of illness. The medical recompression is carried out under a special program. Symptomatic treatment is used depending on patient’s condition: stimulation of cardiovascular system, warming, oxygen, means directed on struggle with pain, with a possible oedema of lungs. Application of a hyperbaric oxygenation gives quite good outcomes.

Verification of the ability to work. The sick-leave is given for a period of treatment for 10 days at mild degree of illness. Patient can be temporarily given a work outside of heightened atmospheric pressure and other unfavorable factors operation with issue of a labor sick-leave in case if further treatment in out-patient conditions is necessary.

Return the sufferer of caisson sickness of mean gravity to the same work is authorized after a period of temporary incapacity for work.

Availability of complications in the form of firm organic changes on the part of organ of sight and gastrointestinal tract leads to a steady disablement with a rather large list of counterindicative kinds of labor activity.

The labor forecast at a high-gravity degree of a caisson illness is always unfavorable. It is necessary to send patients on commissioning for disablement degree definition and rehabilitational measures elaboration.

Preventive measures. The warning of decompression disease is envisioned, first of all, by observance of rules of work in caisson-box. So, the maximal pressure during their realization should not exceed 3.9 atm. A working day in a caisson box is divided on two parts with a rest between them not less than 9-10 hours outside of a caisson box. General number of working hours during a day, including time of locking and unlocking, is ranged from 6 h till 2 h 40 minutes depending on pressure in a caisson box.

Breathing with oxygen, struggle against overcooling of workers is a preventive action against caisson sickness.

An ambulatory or a medicine post with a day-night duty of medical staff is organized for the well-timed and qualified health services on each site of construction where the caisson work is realized. Isolation ward on the occasion of a decompression sickness, medical airlock can be at a medical center.

Persons permitted to decompression and diving jobs should pass preliminary medical examination. Contraindications for admittance for these jobs is hypertensive disease, pulmonary tuberculosis, respiratory tract lesion of not tubercular etiology, peptic ulcer of ventricles and duodenum, illness of nephroses and urinary bladder, sugar Diabetes, and excessive stoutness.

All people working in a caisson box are subject to weekly medical examination with participation of doctor - therapeutist and otolaryngologist.

ILLNESSES ARISING OWING TO INFLUENCE OF UNFAVORABLE FACTORS OF MANUFACTURE'S MICROCLIMATE

Unification of temperature, humidity, air movement speed and infrared heat radiation in a working area is understood as manufacture's microclimate.

It largely depends on meteorological or climatic conditions of the given region, and at definite kinds of jobs (in open air) can be completely conditioned by them. Besides, the microclimate of manufacturing premises depends on nature of a engineering procedure, conditions of an air exchange and other factors.

Overheat

Overheating of organism is a condition that is characterized by disturbance of a heat balance, increasing of quantity of heat in organism. Overheat arises under influence of heat of ambient manufacture medium, and also if there is the factors that preclude from withdrawal of heat in environment. Overheat is watched on works with high temperature of environment (blast furnaces, open-hearth furnaces, rolling shops, coal mines with deep deposit of coal), and also in climatic regions with a hot climate. Increase of heat exchange that arises in case of fulfillment of hard physical work, in particular, in clothing impermeable for water evaporating, at high humidity and immovability of air assist overheating.

Pathogenesis. Mechanism of effect of various kinds of heat (convection, conduction, radiation) on an organism is not identical. Convection and conduction heat being transmitted from molecule to molecule produces heating of tissues surface and blood circulating in them. This blood transfers heat both in more deep tissues, and in organs.

Radiation (infrared) heat can permeate deeply into tissues of man.

Starting device of reactions of physical and chemical heat regulation in organism is a thermal irritation of dermal and vascular thermal receptors with subsequent corresponding reaction to it from a center of thermal control. Change of physiological functions at considerable overheat arises also owing to effect of heated blood on a central nervous system. Blood vessels of skin are dilated, their filling with blood is augmented, and temperature of skin increases. Heat emission through irradiation, convection, conduction decreases, and heat emission through evaporation of moisture from a body surface increases in case of environment temperature rise.

So, for example, it is considered that a portion of heat emission through convection and irradiation is reduced from 73 up to 8 % from summary heat emission, and a portion of heat emission through evaporation of moisture is augmented from 27 up to 92 % at environment temperature rise from 16 up to 30 °C and at work of a middle gravity degree for a dressed person. Emission of heat from the body surface through convection and irradiation is ended at environment temperature of about 33 °C.

Clinic. Four degrees of overheat are marked out. At the *I degree*, a general condition of ill is satisfactory, there are complaints on feeling of heat, listlessness and sleepiness, absence of desire to work and move. At that, body temperature can reach 37.5 °C, arterial pressure (systolic and diastolic), volumes of lung ventilation, consumption of oxygen and excretion of carbonic acid are reduced. A minute volume of heart is increased, pulse becomes more frequent on 15-20 beatings a minute. Hyperemia and humidification of skin can take place.

Overheating of the *II degree* of gravity is characterized by an acute hyperemia of skin, profuse emission of sweat, as well as sensation of fever. Body temperature can reach 38.5 °C.

Systolic arterial pressure is increased by 5-15 mm of mercury column, diastolic one is reduced by 10-20 mm of mercury column; pulse becomes more frequent by 40-60 beatings a minute; minute and systolic volume of heart, lung ventilation, quantity of absorbed oxygen and discharged carbonic acid are augmented.

Overheat of *the III degree* can be observed at effect of temperature of above 60 °C. State of health deteriorates, what can be proved by the sensation by strong fever, palpitation, pulsation and pressure in temples, gravity and pain in head. Motion agitation is marked; skin has sharp hyperemia, and sweat flows off by drops; pulsation of carotid and temporal arteries are watched. Body temperature reaches 40 °C, systolic arterial pressure is reduced by 30-40 mm of mercury column, pulse becomes twice and more times faster in comparison with the initial value, reaching 160 beatings a minute on average.

Overheat of the *IV degree* is characterized by lesion of activity of cardiovascular and central nervous systems that takes place at the so-called thermal shock.

A face has hyperemia from the beginning, and becomes pale-cyanotic later on. Skin is dry, hot or covered with sticky sweat; temperature increases up to 41-42 °C; diuresis is decreased.

Breathing is shallow, fast, and irregular. Pulse is rapid, fine, and thread-like, cardiac sounds are toneless. Consciousness changes from mild degrees up to coma; cramps have tonic and clonic nature; psychomotor excitation; often dreaming and hallucinations develop. Clotting of blood with increasing of residual nitrogen and urea, and decreasing of chlorides takes place in it. Mortality at this stage reaches 20-30 %.

Peculiar form of overheat is a sunstroke. It is mostly caused by direct effect of solar radiation (for example, on builders, workers of open-cast mines and agriculture) and is a consequence of influence of infrared radiation on a central nervous system. Gravity of sunstroke flow is conditioned by degree of lesion of brain membrane and other structures of central nervous system. Infrared radiation permeating to considerable depth (2-3 cm) in tissues can cause diseases of meningitis and encephalitis. It is necessary to mark that such pathology in conditions of works does not develop even at high intensity of infrared radiation.

Clinic. General weakness, sensation of indisposition, headache, loss of consciousness, twinkling before eyes, tightening in a thorax, buzzing in the ears, sometimes nose bleedings, giddiness, vomiting, irregular feces come up at this form of overheat, skin gains red color, in particular on face, and excretion of sweat strengthens. Obviously expressed lesions on the part of a

central nervous system arise in high-gravity cases: aberration, sharp excitation, convulsions, hallucinations and visions. Body temperature of body, as a rule, does not increase.

Lesion of water-electrolytic exchange that is showed in the form of cramps is possible owing to overheat of organism and loss of a large quantity of liquid through sweat. In these cases, evident titanic cramps that are spread to various groups of muscles, in particular sural, thighs, shoulders, forearms, and their sharp morbidity during motion alongside with phenomena referenced for thermal shock, are watched. Patients are adynamic, with sharpened features of face. Skin obtains cyanotic tint, and gets dry and cold on touch. Distresses on the part of cardiovascular system take place: pulse becomes more frequent (up to 110-120 beatings a minute), arterial pressure is low, cardiac sounds are muffled. Diuresis makes 50-100 ml a day; content of chlorides in urine is sharply decreased. Signs of blood clotting can be found: quantities of erythrocytes and hemoglobin are augmented; and viscosity of blood is increased.

Attacks in the form of epilepsy, disorder of psychics can be in high-gravity cases.

Treatment. The treatment of overheat is directed first of all on struggle against of hyperthermia and for normalization of activity of cardiovascular system. For this purpose wet wrappings, chill on head at cramps are recommended. It is recommended to take much liquid, injection of glucose solution of Ringer parenterally together with vitamins. Oxygenotherapy and injections of adrenalin subcutaneously give a positive result at collapse; injections of cardiac glycosides, hyposensitizing means intravenously at acute cardiovascular failure is recommended. Milk and vegetative diet is recommended.

Help at thermal shock foresees transfer of a victim in a cool place, local cooling of head, sponging off a body with cold water. Oxygen inhalation gives quite good result. Intravenous injection of polyglucine, rheopolyglucine, 5 % glucose solution, isotonic solution of chloride natrium (up to 1.0-1.5 l) is recommended in case of arterial pressure decrease and in the presence of other signs of vascular failure. Seduxen/diazepam, Dimedrol, and Droperidol are prescribed at observation of arterial pressure level.

Dehydrational therapy with osmotic diuretics (manitol 1 g/kg of body weight), lumbar puncture is carried out at phenomena of brain hypostasis. Medical hypothermia with a continuous forced ventilation of lungs and correction of disturbance of acid - alkaline and water-electrolytic balances is recommended in high-gravity cases. Reanimation measures are carried out in case of lesion of breathing and blood circulation termination.

Verification of the ability to work. Patients can be temporarily disabled for a term of treatment with light signs of overheat. They can return to their previous work after full convalescence.

When residual functional distresses on the part of a nervous or cardiovascular system take place after experienced overheat, it is necessary to provide the patient temporarily a job, which is not connected with the influence of high temperatures and intensive heat radiation, until his/her full convalescence. Patient is given a sick-leave if necessary.

If the marked stable functional lesions after overheat remain, the further work in conditions of influence of high temperatures and intensive heat radiation is counter-indicative.

Patients in case of full invalidity or absence of possibility of a rational provision of employment are sent on commission for definition of a group of physical inability.

Preventive measures. Improvement of working conditions on works with intensive emission of heat is implements with application of technological, sanitary, hygienic and medical measures.

Application of perfect engineering, localization of heat emission, shielding and thermal insulation of sources of radiant and convection heat, rational ventilation, application of air douche, rational organization of operational and rest mode, observance of drinking and alimentary rations, which are directed on recovery of water-salt balance, render assistance in prevention of overheat unfavorable influence.

Receding and periodic medical examinations that are carried out once per 2 years including therapeutist, neuropathologist, ophthalmologist also have large value. It is necessary to take into consideration medical contraindications for work in conditions of high temperatures and intensive heat radiation presence at realization of the preceding medical examinations.

Cooling

The cooling is a distress of organism functions as a result of low temperature effect. Low temperatures of air are met in rooms where it is connected with an engineering process (refrigeration chambers, textile manufacturing etc.), or with cold season of year for want of heating.

Etiology and pathogeny. Cold as a parasitic factor of manufacture medium influences unfavorably on organism and production activity of a man. Speed and depth of cooling depend parallel with force and duration of chill factor effect as well on condition of organism and conditions, in which one is. Resistance of organism to cooling is decreased at physical tiredness of a person; overcooling comes faster in conditions of high air humidity or high wind.

Cooling is a consequence of disturbance of a heat balance and develops in those cases when heat output in organism exceeds heat production. Phases of compensation and decompensation of heat regulation are distinguished during development of cooling.

Thermoregulator reactions of an organism in a phase of compensation of heat regulation have reflex, adjustment nature and are directed on prevention of decrease of body temperature by the way on the one hand decreasing of heat emission and on the other hand by increasing of heat production. Decreasing of heat emission is reached by discontinuance of hidrosis, spasm of skin and muscles vessels, decreasing of blood circulation in them. Production of heat strengthens at the expense of increase of metabolism. Balance in phase of decompensation of heat regulation between production of heat and a heat emission breaks, heat emission prevails, and consequently a condition of pathological hypothermia develops. Thus, hypoxia takes place as outcome of disorder of breathing and blood circulation. This condition is strengthened by break of microcirculation owing to a decrease of vessels tone, slowing down of blood circulation and deterioration of rheological characteristics of blood.

Pathologoanatomic picture. Morphological changes at cooling are showed with plethora of internals, main and spinal cords and their membranes, cerebral haemorrhages, and haemorrhages into mucosa of ventriculus, small-sized nuclear necrosises in internals, hypostasis of lungs.

Clinic. Four stages in clinical process of cooling are distinguished: compensator, adynamic, soporose and comatose.

In compensator stage suffereds are excited, they complain on chill. Lips are cyanotic, dermal covers are pale; cold, shivering of muscles, shortness of breath, tachycardia, increase of arterial pressure, more frequent urination are marked.

In adynamic stage consciousness is not lost, however sufferer is stopped, sometimes euphoric, the delicacy complains on a headache, loss of consciousness. Availability of adynamia, decrease of muscles tonus, depressing of tendon jerks is marked. Pulse is normal or delayed up to 40 b/minute, arterial pressure does not change essentially, and cardiac sounds are damped down.

In soporous stage general dormancy, flaccidity, sleepiness down to condition of sopor, sometimes euphoria, disorder of memory, dysarthtia is marked. Pupils are dilated or are periodically narrowed down and extended, breathing is slowed down (till 8-10 in 1 minute), surface, sometimes with moans; pulse is infrequent (from 50 up to 30 b/min), arterial pressure is lowered. Incontience of urine and feces is possible.

In comatose stage consciousness in sufferers is missed, arbitrary motions with head and extremity, convulsive tonic contractions of muscles, predominantly flexors, can appear.

Motional excitation arises sometimes. Pupils are narrowed down; their reaction on light is absent. Breathing is surface, infrequent (up to 3-4 in 1 minute). Pulse is determined only on large arteries, infrequent (up to 20

b/minute). Arterial pressure is lowered, cardiac sounds are muted and auscultated hardly.

Complications at cooling are caused by fall-off of an immunological reactivityof organism, because of what a threat of development of inflammatory diseases appears in the course of time: pneumonias, pleuritis, acute respiratory diseases. Besides, functions of other organs and systems can be upset, and that quite often results in asthenisation of psychosises, trophic lesionses.

Treatment. Nature of medical care at overcool depends on depth of hypothermia and degree of disorder of vital functions of organism. The Influence of cold factor on normalization of condition of patients in compensator stage of cooling needs only to be stopped. In adynamic stage temperature of a body also can be normalized independently under according conditions (stay in warm room, warm drink, application of a heater, warm bath).

Persons suffered in soporous and comatose stages of cooling are needed in the first aid. Main efforts should be concentrated on support of breathing and blood circulation, prevention of further cooling and warming of the organism. Sufferers are immediately transferred in a warm room, wet clothing should be removed off them, and they should be put around with heaters. Possibility of falling a tongue back is eliminated, slime from a mouth is pumped out, and oxygen inhalation is performed. If breathing and cardiac activity break has taken place, all complex of reanimation measures is executed. The main method of taking a sufferer from a critical condition is an intensive warming. During the warming a heated 5 % solution of glucoses is introduced dropwise, polyglucin. 40-50 ml 40 % glucose solutions with insulin and ascorbic acid are introduced with purpose of power stuff replenishment. Euphyllin, neuroleptics or ganglioblocators in doses that do not cause a decrease of arterial pressure is possible to use for improvement of cerebral circulation. For correction of acidosis 100-200 ml 5 % solution of hydrocarbonate natrium are poured in; antihistamine drugs with purpose of decreasing of a vascular wall permeability are applied also. Diuretic drugs (furosemid - 40-60 mg, manit - 1 g/kg) for preventive measures and treatment of post hypoxia edema of head brain and stimulation of uropoiesis during warming and after it are applied .

Local lesions in view of obliterating endarteritis and frosted parts of body are possible except for general cooling due to influence of cold also.

The obliterating endarteritis arises at continuous cooling of extremities and is met mainly at fishermen, workers on mining of peat, maintenance staff of refrigerating plant. Clinical signs of this disease are an albication of skin of fingers, decrease of dermal sensitivity, paraesthesies, difficult motion of extremities and weakening of pulsation on peripheral vessels.

Four signs of the obliterating endarteritis are marked. On the I stage (spastic) functional angiospastic disorders are found out. Pain, sensation of a cooling-down and numbness in extremities, weakening of pulse on peripheric vessels appears at patients. Such phenomena can be watched rather continuous time, they periodically disappear and come up again. At the second stage (ischemic) the angiospastic syndrome becomes more constant and expressed owing to development of steady structural changes in walls of vessels. A threat of formation of thromboses in walls of vessels exists. Stage III (necrotic) is characterized by formation of ulcers on extremities, what is conditioned by disorder of nutrition of tissues. On the IV stage (gangrenous) a development of dry or wet gangrene is observed.

Freezing as well as the obliterating endarteritis is characterized by local lesion of tissues of organism owing to low temperature effect.

Clinical signs of freezing occur after some period of time after termination of cold influencing. Three degrees of freezing are distinguished. Sensation of itch, pricking occurs at freezing of the I degree, as well as albication of affected sites of body, which transfer later in reddening, is observed. The indicated changes pass over quickly without any consequences. Decrease of skin sensitivity that is accompanied by its albication and edema of affected sites is characteristic for the II degree. Bladders charged with serous-bloody liquid occur. Gangrene develops at freezing of the III degree.

Freezing develops faster in case of joint action of factor of chill with heightened humidity, hypodynamia, local disorders in a blood supply of tissues, due to tight footwear or clothing.

Treatment is carried out depending on form and degree of disease gravity. Thermal physiotherapeutic procedures, means, designed for elimination of pain, vasodilatation are recommended ill with the I and II stages an obliterating endarteritis. A surgical operation is sometimes necessary at the III and IV stages.

General warming of body, rubbing with camphoric or boric alcohol (3 % solution) of lesion sites of skin with the subsequent imposing of dry bandages for patients with freezing of the I degree.

Surgical operation is recommended for patients with freezing of the III degree. It is necessary to remember that appearance of a secondary infection contamination is possible at these stages, therefore prescription of antibiotics should be included in a complex of medical measures.

Verification of the ability to work. Persons, who have experienced light forms of overcooling, can return to previous work after a conducted treatment and full convalescence. If residual phenomena with functional lesions that do not give capabilities to perform previous work take place after high-gravity forms of freezing experienced before, such patients with

allowance for degrees of functional disorders should be rationally provided a job or sent on commission for definition of group of physical inability.

Preventive measures. The main measures on preventive measures of cooling of an organism are following:

- prevention of manufacturing rooms cooling down using thermal insulation of a floor, walls, windows etc.;
- installation of local heating on fixed working places;
- installation of equipment for heating of rooms in large shops without fixed working places (refrigeration cabinets, preparation plants) and during work on open air in cold climatic zones; temperature of air in rooms can be supported in limits 21-24 °C;
- application of means of individual protection from chpatients;
- implementation of measures on hardening an organism, his adaptation to staying in conditions of a cold climate;
- organization of preliminary and periodic medical examinations of persons, who work in conditions of low temperatures effect; the periodic medical examinations should be carried out once during two years including therapeutist, neuropathologist and surgeon.

It is necessary to take into consideration a list of contraindications for work under conditions of chill factor effect at preliminary examinations of persons, who are assigned to work.

Chapter 8

OCCUPATIONAL DISEASES CONDITIONED BY FUNCTIONAL OVERSTRAIN

The pathology, which is referred to diseases of locomotor apparatus, takes a rather considerable place among occupational diseases. First of all these conditions are connected with functional overstrains and microtraumatism.

They are met among blacksmiths, loaders, operatives of rolling mpatients, mine workers, typists, line-operators, pianists, insulation workers, etc. Thus, the basic unfavorable factors in genesis of these diseases are both a high-gravity manual labor and a local loading and considerable static strain owing to motions of extremities that are multiply repeated, as well as continuous stay in an enforced pose.

Majority of this pathology syndromes are localized in the upper extremities, what is explained with large local loadings, significant amount of small stereotyped motions of hands and fingers of arms (in some trades they reach several tens thousand motions). So, gravers of crystal only during one shift do up to 40 thousand press efforts in various positions of hands with considerable pressure of hands and forearms muscles. Milkmaid fulfpatients about 2500 compressions with fingers when milking only one cow during a day. Typist does above 60 thousand strokes on keys of type machine by fingers per one shift, and operators of accounting machines fulfill from 100 up to 200-250 thousand strokes with an effort at each stroke from 0.5 up to 1 N.

These unfavorable factors are strengthened by enforced working positions, continuous static strain of back and extremities muscles, non-observance of operation and rest mode, irrational working methods (incorrect bearing of hands at musicians), microtraumatism of skin and its receptors, temperature drops, constant humidifying of hands by water, oils, emulsions and so on.

Estimation of gravity and strain of labor process is carried out in accordance with «Labor hygienic classification». The I and II classes (they eliminate threat of development of occupational diseases) are considered as optimum and permissible. Thus, such characteristics are standardized:

- power of dynamic work (W) at loading of muscles of the upper extremities should not exceed 45 W for men and 30.5 W for women, and at load of muscles of the lower extremities and trunk 90 and 63 W accordingly;
- weight of load that is lifted should not exceed 30 kg for men and 10 kg for women;
- quantity of hand and finger motions per one shift should not exceed 40000 small stereotype motions;

- static muscular loading should not exceed 430000 Nf per shift when the load is holding with one hand, and 970000 Nf when by two hands;
- working pose should be free; staying in an inclined position under angle less than 30° should not exceed 25 % of shift duration, and if it is necessary a quantity of trunk inclinations under angle more than 30° should not exceed 100 times.

There is a possibility of development of some occupational pathology in case of exceeding of these standards (the III class of labor conditions).

Classification. The following occupational diseases conditioned by a functional overstress are marked.

1. Occupational dyskinesia (coordinator neurosis).

2. Diseases of peripheral nervous system: neuropathy, cervical shoulder plexopathy, cervical and lumbar-sacral radiculopathy, and lumbalgia.

3. Diseases of apparatus of resistance and motion: а) illnesses of muscles (myalgia, myositis, myofascitis); б) illnesses of fibrous-tissue and synovial formations (shoulder scapula periarthritis, epicondylitis, tendovaginitis, stenosing ligamentitis, contraction of palmar aponeurosis, and bursitis); в) osteochondropathy (deforming arthrosis, spondylarthrosis, and bone aseptic necrosis); г) combined syndromes.

Pathogenesis. Mechanism of development of diseases conditioned by a functional overstrain is very complex and is not yet clarified to the end. If a pathogenesis of occupational dyskinesia is evaluated, it is considered that a continuous work, which demands doing of considerable amount of small motions of high coordination with a very fast speed, can cause an overstrain of nervous processes, lesion of cortex neurodynamics with formation of an isolated “ill nucleus” in certain department of motion analyzer. It results in lesion of complex motion stereotype, which is situated in foundation of strictly differentiated motions.

Besides, such factors, as emotional overload, dissatisfaction with job, often conflict situations at work and in a household provoke the development of occupational dyskinesia.

High-gravity physical work, fulfillment of a great number of uniform motions is accompanied with traumatisation of peripheral receptors of a nervous pipe. Thus, lesions of blood circulation and metabolism, which lead to degenerative changes in peripheral nerves, arise. Such factors as strains, compressions, infringement of nervous pipes in marrow or in muscle-binding channels, in particular in a working time that is carried out with permanently bent extremities also assist to development of these processes. Lesion of blood circulation and metabolism develops also owing to accumulation in muscles of underoxidized products of exchange, changes of inflammatory nature in interstitial tissue and as a result of secondary degenerative lesions of muscle fibers, which develops later. The indicated processes as well as aseptic inflammation, which accompany them, can be developed in tendons,

serous bags, periosteum, and cartilages of joints. It results in development of such pathologic conditions, as periarthritis, tendovaginitis, bursitis, arthrosis and so on.

OCCUPATIONAL DYSKINESIA

Occupational dyskinesia takes a special place among all diseases conditioned by functional overstrain, as it is a functional disease of the central nervous system. For the first time, this disease was described as “writing spasm” in the middle of XIX century. The cause of its originating was connected with an intensive written work, as the disease was fixed at office employees and copyists. Later it turned out, that these phenomena of discoordination arise not only during writing, but also at many other kinds of job, which demand fulfillment of hard coordinated motions with fast speed. Today occupational dyskinesia is described for gravers, knitters, graphic artists, draftsmen, fitters, milkers and milkmaids, musicians playing keyboard and string instruments.

Clinic. The disease arises gradually and develops slowly. Such clinical forms of this pathology are marked out: convulsive, paretic, atactic, shivering, neuralgic, and mixed. Increase of tone of hand small-sized muscles during occupational work is characteristic for the convulsive form.

So, persons, whose occupation involves writing, experience discomfort when writing of separate characters or digits, their handwriting becomes worse, and then cramps appear in one or several fingers during writing.

Sudden weakness in hands comes up at patients with paretic form of dyskinesia in time of working; fingers are relaxed involuntarily owing to what a worker can not hold in hands a pen, a pencil or other tool.

A version of the paretic form is so-called atactic form of dyskinesia, which develops predominantly during work on a keyboard (typists, lino operators and pianists). Characteristic symptom of such disease is that an ill cannot touch the key, which is necessary to him/her.

At the shivering form of dyskinesia a tremor comes up predominantly in that hand, which is more loaded during fulfillment of this or that activity.

For the neuralgic form an occurrence of intensive pain is characteristic in muscles of hand, forearm or all arm during work-time. Talk about the mixed form of occupational dyskinesia takes place in the event of presence of combination of several forms at ill.

Diagnosis. It is necessary to remember that the most typical symptoms of the occupational dyskinesia are selectivity with lesion only one function, which makes a basement of this or that occupational work: writing, typing on a printing machine, playing on a musical instrument and so on. Other motion functions of working hand can also suffer in the course of time.

Differential diagnostics. The differential diagnosis of occupational dyskinesia is carried out with hysterical paresis of hand, organic diseases of central nervous system, in particular with those that are accompanied by extrapyramidal and cerebellar lesions. Paresis of hand at hysteria develops usually quickly and is spread to all its functions.

The organic diseases of central nervous system are accompanied with intermediate symptoms and never are limited only with lesions of occupational functions of arm.

DISEASES OF PERIPHERAL NERVOUS SYSTEM

The given group of diseases includes lesions of separate nerves of spinal cord (ulnar, middle, small shank), polyneuropathy of the upper extremities, cervical - shoulder (plexalgy and plexopathy, lumbalgy, neck also lumbosacral radiculopathy). A special place is taken by the so-called tunnel syndromes (compressive neuropathy), which are developed owing to squeezing of meddle, ulnar, radial and other nerves with tissues, which surround them, and which have changed pathologically.

Neuropathy of an ulnar nerve is met most often owing to traumatism of the nerve during fulfillment of work, which demands supporting of elbow with table (carver, graver, engraver, and glassblower). Patients complain of pain and paresthesia in a region of the fourth - fifth fingers. Strength and endurance of hand muscles are reduced owing to what they cannot hold in hands tool or detail that should be processed continuous time. Hypesthesia is met on the fourth - fifth fingers and on ulnar edge of hand; hypotrophy of the fifth finger's muscles arises. Patients cannot bring the fifth finger together with the fourth one.

Lesion of the middle nerve is characterized by that at patients early occurs paresthesia and pain in the second - third or in all fingers, in hand and forearm. The pain is considerably strengthened at night, during cooling, at change of atmospheric pressure. Hands are cyanotic, wet. Hypostasis of fingers, hyposthesia on palms, in particular on the first - third fingers and on back side of distal and middle phalanxes of the second - fourth fingers can be observed. In the course of time atrophy of muscles of the first finger's rise develops, strength of hand's compression is reduced.

The neuropathy of nerve of small shank develops at workers, which stand on knees or squat down during continuous time (parquet floor layers, roofers, workers of agriculture).

Squeezing of nerve in popliteal fossa or near a small shank bonehead takes place because of features of occupational work. Patients complain on a hindered rotation of foot outside; hypesthesia of external surface of shank and back of shank appears. The so-called "cock step" arises at patients.

Vegetative-sensory polyneuropathy. Complains appear on an incipient stage of the disease: ache in bones and forearms, paresthesia in

them (feeling of numbness, “crawling of insects”). These feelings are strengthened during a change of atmospheric pressure, at cooling. In the course of time pain and paresthesias strengthen, become constants, disturb ill after work, at night, upsetting sleep. Peripheral vegetative - vascular lesions come up: hyperhidrosis, puffiness of distal phalanxes, change of skin coloration and hands temperature. Cyanotic color and invariable or heightened temperature, and paleness or marmoreal skin and hypothermia of hands at predominance of spastic phenomena are marked in case of paretic phenomena predominance in capillary tubes and vessels of small caliber. Sensory lesions at vegetative polyneuropathy decrease or hypesthesia takes place.

Surface sensitivity in process of development of a vegetative - sensory polyneuropathy is reduced according to polyneuritic type. Expressiveness of peripheral vegetative - vascular lesions increases: edemas and hard movable properties of fingers, which are over or considerably decrease after beginning of work come up in the mornings. Trophic lesions are often observed: hyperkeratosis of palms, change of form and frailness of nails, deformation of interphalanx joints. Complain on weakness, fatigability, decrease of force in the upper extremities are joined in case of motive fibres retraction in pathological process. Decrease of hands pressure force, hypotrophy of separate muscle groups of the upper extremities, decrease of tendon jerks are determined objectively.

Lumbosacral radiculopathy is met at persons, whose work is connected which a considerable static-dynamic loading on lumbosacral area of vertebral column owing to frequent fulfillments, in particular to forced tilts and trunk turns (operatives of rolling-mill, and lumberers), systematic lifting and transportation of hard loads (loaders, dockers, and carvers), continuous maintenance of forced pose (drifters, fettlers, miners), influence of general jerk similar vibrations (drivers of large trucks, tractor and combine operators). Overfreezing and temperature differences strengthen an effect of these factors.

Steady pain in transversal - sacral region, in particular during trunk tilts and turns is marked at patients. Hypotrophy of gastrocnemius muscle, decreasing of reflex of Achilles' tendon develops. Weakness and hypotrophy of muscles of the front regions of shank develops gradually, in particular in a long extensor of large finger. Knee reflex is reduced or disappears quite often.

DISEASES OF MUSCLES

These diseases are ones of the most widespread forms of occupational pathology conditioned with the functional overstrain. They include two clinical syndromes: myalgia and myositis. These conditions are met at workers of the most different trades, whose jobs are connected with

considered static-dynamic loading. First of all, builders, workers of brickworks and bread-baking plants, milkmaids, typist, pianists are among them. Muscles of forearms are subjected to the affliction most often, muscle of shoulder less often.

Myalgia starts from ache, sensation of discomfort, gravity, and strain in the according muscles. Speed of work is reduced, mechanical excitement of muscles and their bioelectric activity is increased. Strength and, in particular, their endurance to static loads is reduced.

Myalgia is a functional stage of the disease. Condition normalization is already marked in some days of rest. At the same time a relapse of the disease with tendency to further progress down to development of degenerative – dystrophic also of secondary inflammatory changes in muscles is observed rather often at resumption of work.

Myositis is characterized with strengthening of pain and paresthesia in hands that cause patients to complain not only in working time, but also in periods of rest, especially at night. The pain strengthens sharply during active motions. Speed of motions, productivity of work is reduced. The affected muscles become hardelastic, flabby, and atrophic. Very painful dense knots from 3-4 mm up to 2-3 cm are palpated in thickness of a muscle tissue.

DISEASES OF FIBROUS AND SYNOVIAL FORMATIONS

Such diseases as periarthritis, ligamentitis, epicondylitis, tendovaginitis, bursitis belong to diseases of peripheral ligaments or muscle fastenings.

Humeroscapular periarthritis as an occupational disease develops during fulfillment of work, which demands repetition of motions in a shoulder joint, in particular on a background of a considerable static-dynamic loading (bricklayers, plasterers, painters). The disease starts with pain in joints of shoulder at motion, raise, and rotation of shoulder. With the course of time, the pain strengthens, becomes constant, crunching appears in joints at motion. Limitation of motion in the joint increases gradually, especially at rotation and taking the shoulder aside. Taking an arm behind own back is considerably limited, and often it is impossible. Development of small mobility in the shoulder joint is possible later on. Pain comes up at palpation of a large knob of a shoulder bone, deltoid muscle less often, in particular in a place of its fixation to the shoulder bone.

X-ray research at periarthritis of shoulder joint demonstrates sclerotic change of a knob surface of a shoulder bone and availability of shades of calcareous deposits of different sizes, forms and density.

Multiple edge resorptions, which are surrounded by sclerotic process, and destruction of a large knob of a shoulder bone, saline deposits in a synovial bag are observed on a roentgenogram at this pathology most often.

Epicondylitis. This disease is diagnosed at localization of an aseptic chronic inflammation and degenerative-dystrophic changes in a region of place of a node fixation of tendon of protractor-arch supporter group of muscles of forearms (lateral epicondylitis) or in a region of a medial node of fixation place of tendon of flexors of hand and fingers (medial epicondylitis). The disease develops predominantly at persons occupied with high-gravity physical activity, and in case of fulfillment by them often protractor-bending and rotating motions in an ulnar joint (workers of agriculture, construction, blacksmiths, and operators of accounting machines).

Literal epicondylitis is characterized by appearance of ache in the region of a lateral node of shoulder bone during forced motions of a forearm and strained extension of a hand. In the course of time, the pain strengthens and arises at any motions in an ulnar joint.

Soreness at palpation of the lateral node is a constant symptom. A small swell occurs sometimes in this region. Unbending of forearm up to 160-170° for majority of patients is limited. Decrease of hand pressure strength is marked at all patients.

Tendovaginitis. This disease is characterized by lesion of a synovial membrane of tendinous vaginas of degenerative-dystrophic nature with concomitant an aseptic (serose-haemorrhagic) inflammation. Tendovaginitis as an occupational disease develops mainly on the upper extremities, in particular as a result of fulfillment of a great number of motions with fingers, hands (insulator workers, bricklayers, winders, typist, and tailors). A tendon of flexors and extensors of hand fingers are usually struck, tendon of a long head of shoulder biceps muscle, heel tendon is struck much less often.

Acute and chronic forms of the disease are marked according to their clinical developments. Aseptic inflammatory process at acute tendovaginitis is accompanied by a fibrin deposit on a tendon surface and can be spread on cellular tissues around the tendon. The process, which flows chronically, is characterized by accumulation of exudation in synovial room. The exudation contains a lot of fibrin. At first from it the so-called rice corpuscles are formed, and then fibrinous adnations between parietal and visceral leaves of incrassate and loosened synovial membrane come in.

Synovial vaginas, which are placed on back surface of hand mainly in a region of a short extensor tendon of the large finger and long muscle, which brings the large finger of a hand aside, are struck at acute tendovaginitis more often. Start of disease usually is connected with a considerable physical loading of according muscles, immediately after which an ache, feeling of fever, pricking, and gravity in the forearm, feeling of tiredness, weakness in hand appears. Dense, quite often painful swelling of 8-12 cm length and 3-4 cm width comes up on back-radial surface of the lower third of a forearm in some hours or 1-2 days. Crepitations (crepitating tendovaginitis) is revealed at palpation of swelling during several hours or 2-3 days.

Pathological process in case of chronic flow of the disease is developed most often in region of fingers flexors synovial vagina, which is arranged in a wrist canal.

The corresponding tendons are painful during a palpation, incrassated. Swelling can be determined on a palmar surface of a radial carpal joint and distal part of the forearm. Pain and fluctuation takes place during its palpation. Force of this hand soon decreases.

Chronic tendovaginitis also develops gradually or is a consequent of a relapse of the crepitating tendovaginitis. Inflammatory reaction at this version of clinical flow of tendovaginitis is not expressed sharply, though it is necessary to have in view that increasing phenomena of sclerotic process are resulted in a thickening of a synovial membrane, narrowing synovial room and lesion of tendons motions (so-called a stenosis form of tendovaginitis).

Stenosis tendovaginitis. Under conditions of production, this disease is formed mostly in a region of a radial carpal joint. Inflammatory process is spread to ligamentary structures of one of the hand osteofibrous canals. Or the cicatricial corrugation of bundles, which are formed the canal and its stenosis with infringement of veins, which passes through it, owing to primary development in it degenerative-dystrophic changes, is observed. Stenosis ligamentitis of a ring-type part of a finger fibrosis vagina (stenosis tendovaginitis of fingers flexors) can be an example of such lesion. Continuous traumatism of a palm at the level of calcaneum-phalanx articulations (for fettlers and stamp operators) can result in cicatrical changes of a ring-type part of a fibrous vagina of fingers flexors (usually the first - third) with a subsequent narrowing of a tendon vagina and deformation of tendon bundles. Pain arises in sites of a corresponding calcaneum-phalanx joint in case of compression of tool, processed detail and at motions of a finger. An intermittent contraction (jam of a finger, which is fixed in a position of bending, and it is necessary to apply much effort for its unbending) appears at some period of time. It is accompanied with pain. The jam becomes frequent in the course of time, extraction the finger from a pathological position becomes more and more difficult and demands help of the second hand. Sometimes, it becomes impossible, even if such help is rendered.

Occupational bursitis is a chronic inflammation synovial vaginas, which develops as a result of systematic pressure, overstrain and traumatism at mine workers, roofers, bricklayers, pavers, parquet floor layers, branchers, who are representatives of those trades, where continuous leaning on an elbow or a knee takes place with development of vagina aseptic inflammation and formation of serose-haemorrhagic exudation in it.

An inconvenient position, roughness of ground, imperfection of protective clothing, continuous cooling, abrupt change of ambient temperature, and vibrations assist the development of the disease.

Pathogenesis. Dystrophic changes in hollow of affected synovial bags with formation of numerous free bodies, which have chondroid density, arise under influence of prolonged pressure and rubbing near working surfaces of joints. Sites affected with a sclerotic process, as well as liquid contents of the bags also experience a calcification. Loss of elasticity and thickening of the bags, fibrosis in the hypodermic basis, hyperplasia of walls of the synovial bags is an outcome of these processes.

Clinic. Bursitis acute (serous and purulent) and chronic (serous and proliferative) is distinguished. The acute bursitis is diagnosis less often, than chronic.

At acute serous bursitis patients complain of morbidity in a joint, availability of intumescences of soft consistence and spherical shape. The skin in area of the intumescences has signs of hyperemia, local rise of skin temperature in this area is determined at contact, and limitation of mobility in the joint takes place. Content of the bag has serous nature.

Body temperature at the purulent bursitis increases up to 39-40 °C. Punctata of the bursa is purulent with large contents of leucocytes.

Chronic serous bursitis is met seldom. It is characterized by duration of flow, moderately expressed morbidity, availability of restricted intumescences of ovoid shape. Proliferate form is a widespread form of the chronic bursitis. Flow of the chronic bursitis of such form is lingering with periods of remissions and exacerbations; an infection contamination can be affixed often. A fluctuation is determined locally, walls of the bags are incrassate, and an articular slot is reduced.

The Dupuitren’s syndrome is a fibrosis-cicatricle degeneration of aponevrosis with formation of tendogenous contraction of the third - fifth fingers in a site of the finger – phalange and proximal interphalangeal joints. It arises at fulfillment of work that is accompanied with traumatism of a palm. Clinically as a rule, painful feelings arise in case of fulfilment of hard physical work. Hardening and cicatrisation of a palmar aponeurosis close to foundation of the third - fifth fingers, hardening and traction of their tendons, formation of these fingers contraction is marked.

OSTEOCHONDROPATHIES

The types of osteochondropathies are arthrosis, spondylosis arthrosis, as well as aseptic neurosis of a bone.

Arthrosis. The occupational deforming arthrosis is developed at considerable dynamic and static loading on a joint. Concomitant vibration, and also a plenty of motions, jerks make assistance to this pathology development. Representatives of such trades as bricklayers, loaders, blacksmiths, plasterers, painters, typists, pavers, parquet floor layers and so on are affected more often.

An elbow joint on the upper extremities is struck more often, and a knee-joint on the lower extremities.

Sensation of discomfort, availability of crunching in the joint, pain of uncertain nature disturbs patients. Pain is strengthened gradually, becomes continual. Intumescences, deformation of the joint is observed. Amplitude of motions and mobility in the joint is restricted.

Aseptic necrosis of a bone. It is an occupational disease, which is characterized by development of degenerative–dystrophic changes in supporting bones of a hand and foot, and also in bones of large joints of extremities, of the elbow one first of all. A cause of their originating is an occupational chronic overload and micro traumatism of osteal structures during hard physical work, and influence of intensive local vibrations, in which spectrum a low- and mid-frequency vibration prevails. The pathological process develops more often in a fortnightly bone, which experiences the greatest loading from all bones of a radiocarpal joint.

Aseptic necrosis of the fortnight bone (Keenback's disease). This disease is developed at a considerable systematic static and dynamic load of hand at drift miners, fettlers, workers of building trades, joiners. The process is characterized by development of a subchondral aseptic necrosis, which is accompanied with fractures, deformation and fragmentation of the bone.

The disease is characterized with pain in the radiocarpal joint, which arises during motion. With the course of time the pain is strengthened, becomes permanent. The intumescence of 1-1.5 cm in diameter, which is excruciating, can be seen in a region of a fortnight bone projection on the backside of the wrist at palpation.

The amplitude of motions in this joint is restricted.

Diagnostics of occupational diseases caused by a functional overstrain has some complexities that are conditioned, first of all, by absence of specific clinical manifestations of these conditions. Therefore, it is necessary to analyze working conditions, nature and intensity of work of an ill person, presence of concomitant unfavorable factors of working environment, duration of the occupational service, feature of originating, flow and clinical manifestations of the occupational pathology, presence or absence of other etiological factors for diagnosing the disease in each concrete case.

The occupational diseases caused by functional overstrains are usually arisen gradually, developed gradually during of rather continuous time. A considerable improvement of the condition due to rather continuous interruption in work, and in incipient stages and in several days of rest is also characteristic for occupational diseases. The absence of other etiological factors, first of all, infection diseases and traumas, has essential value. The majority of these conditions are developed at persons, whose duration of an occupational record makes not less than 10-12 years. Localization of the

disease, which depends on a degree of loading for this or that hand, matters also.

Treatment. Main medical practice at the diseases caused by functional overstrains should include medicaments drugs, physical and reflex methods.

Total and rather continuous termination of occupational work is by an obligatory condition for treatment of dyskinesia. Sedative drugs, tranquilizers, method of autogenic training, psychotherapy, electrosleep and balneotherapy are applied in the treatment. The medical measures are rather effective only on incipient states of disease.

Drugs of antispasmodic effect and those influencing on condition of microcirculation: Xantinoli nicotinas 0.15 g three times a day or "Nicoverinum" 1 pill and Nicospanum three times a day after meals are applied when treating the vegetative - sensory polyneuropathy. Vitamins of the B group (bromine thiamine, hydrochloride pyrodoxine, cobalamin cyanide), cocarboxylase, adenosine triphosphate, and biostimulants (aloe, FIBS) are prescribed with the purpose of metabolic process normalization. Ganglionic blockers: benzohexonium, Pachykarpin, spasmolytin (0.05-0.1 g three times a day), intravenous injections of 5 ml of 0.5 % Novocain solutions (10 injections per a course) are applied at presence of more expressed and steady changes.

Method of reflexotherapy and physiotherapy - electrophoresis 5 % of Novocain solution on hands, and ultrasound provides positive results. Massage of a cervical - collar region, balneotherapy-radon and hydrosulphuric baths are recommended.

Treatment of the occupational radiculopathy is carried out with analgesics, non-steroid antiphlogistic drugs (Analginum 0.5 g, Butadion 0.25 g, Indomethacine 0.25 mg), neuroleptic drugs (Haloperidolum 1.5 mg triply per day), myorelaxants (Mydocalm 0.05 g, and Sibazone 0.5 mg during a day), injections of vitamins of the B group, biostimulants. Ultra-violet exposure, diathermy, radon and hydrosulphuric baths, massage, medical gymnastics are widely used.

The treatment of diseases of muscles is the most effective in the incipient stage. Application of ultrasonic sound, short-wave diathermy and Bernar’s currents gives a positive result. Medical gymnastics and massage have large value in treatment of myofascites. Intramuscular introducing of 5 ml of 2 % Novocain solution per day during 10 days, vitamin B_1 (30 mg per day during 15 days intramuscular) is recommended at presence of muscles sharp morbidity. Warm baths with a subsequent massage of arms and shoulder girdle muscles are prescribed also.

Therapeutic measures at a periarthritis of the shoulder joint consist of mobilization, novocainic anesthesia, physiotherapy, including roentgenotherapy and treatment by ultrasound, punctures and washing of a subacromial bag. Rheopyrinum, Analgin with Amidopyrin, electrophoresis

with Novocain are applied in the period of worsening. Continuous immobilization of the shoulder joint is not recommended, as it can result in development of hard mobility of the joint.

Treatment of epicondylitis of a shoulder in an incipient stage starts from immobilization of hand and forearm with a plaster bandage. Hidrocortizon (from 5 up to 25 mg) every other day during 6 - 8 days inject in a region of the node. The novocainic blockade of the region of the node gives positive results. The course of the treatment consists of 3 to 4 blockades with an interval of 5 days with a simultaneous immobilization of an extremity. Dyadynamic therapy and paraffin applications on the region of the elbow joint (in 3-4 weeks after the immobilization) are effective physiotherapeutic procedures.

Treatment of tendovaginitis foresees a dismissal of the ill from work, a prescription of Novocain blockades in the forearm region (8-10 ml of 0.5 % solution) and imposing of a plaster bandage. Warm, UHF therapy, paraffinic applications (4-6 performances) are prescribed from the 3 - 4-th day. The plaster bandage is removed on the 7 - 8-th day, the volume of motions with the hand and fingers is increased.

The treatment at the acute bursitis is conservative: rest, antiphlogistic substances (antibiotics, sulfanilamide drugs, blockade with Hidrocortizon or introducing of 25 mg (1 ml) suspension of Hidrocortizon in emptiness of the bag after washing it with 0.5 % Novocain solution, 2-3 injections in 3-4 days), physioprocedures (UHF, paraffin application, dyadynamic therapy with 10 % potassium iodide solution during 15 minutes, only 10-20 sessions).

Surgical treatment is applied at a relapse or transition of the process in chronic.

Injections of Hidrocortizon, Novocain, Rumalon, Lydas (per 1 ml) under cicatricially changed tissues each day or in a day (10-15 injections), and also phonophoresis of Hidrocortizonum, electrophoresis of Iodine, lithium, and Lydasum are prescribed in the incipient stage of palmar aponeuvrosis contraction; a surgical treatment is recommended later on.

It is necessary to ensure a relative rest for the affected joint at arthrosis of a knee joint. Analgesics, non-steroid antiphlogistic drugs (Analgin, Rheopyrin, and Indomethacin), and vitamins are prescribed. Injections of Rumalon of 2 ml intramuscular a day for 5 or 6 days are prescribed. Electrophoresis with Novocain, Iodine, Chlorine, phonophoresis of Hidrocortizon, UHF therapy, paraffin applications, balneotherapy, and massage are recommended from physiotherapeutic procedures.

Verification of the ability to work. It is possible to conduct the treatment of the ill with presence of a myalgia, vegetative polyneuropathy, serous bursitis ambulantly with a shift of the patient on an easier work for the period of two weeks according to a medical board recommendation. It is necessary to dismiss the patient from work at other forms of the pathology:

for 5-6 days at myalgia with a pain syndrome and lumbalgia, for 2-3 weeks if there is polyneuropathy, myositis, tendovaginitis with crepitation, epicondylitis, for 3-4 weeks at a humeroscapular periarthritis, arthrosis, aseptic necrosis of a bone.

Then, patients are transferred to easy work under conditions, which eliminate traumatic effect, for the term from 10 till 14 days at myalgia, tendovaginitis, and from 4 till 6 weeks at other conditions of this pathology for strengthening of the treatment results. After that a sanatorium treatment is recommended.

Development of dystrophic changes, often relapses, development of a course of pathological process, presence of the steady pain syndrome, steady lesion of the function are a basis for a training for a new profession of ill and its rational job placement that is not connected with effect of the functional overstrain. Patients are sent to medical board for a solution of the problem of the degree of disablement (1-2 years) in case of proficiency lowering due to change of the working place. And the group of physical inability can be established at a steady decrease of functional capacities of ill (aseptic necrosis of wrist bones).

Preventive measures. Complex mechanization of the most hard works and those operations, which demand fulfillment of a huge number of stereotyped motions with hands and fingers, is the most effective mean among preventive measures for warning of unfavorable effect of functional overstrain. Correctly organized operating mode and rest, gymnastics on a working place, massage and warm baths for hands, prophylactic vitaminization and general ultra-violet lighting, which are carried out in autumn and spring, have also important value.

Particular value in preventive measures of these diseases alongside with occupational selection is given to periodic medical examinations, which are carried out once every two years with participation of a doctor-neuropathologist, surgeon, gynecologist, and therapeutist.

Chapter 9

FIRST AID AT THREATENING CONDITIONS OWING TO EFFECT OF THE OCCUPATIONAL FACTORS ACUTE POISONINGS

The acute poisoning is a pathological process, which arises owing to coming of different materials into the organism from the environment in quantities that produce the disorder of the homeostasis. Gravity of the poisoning depends on a dose, concentration, rate and paths of entry of these substances.

In industry, the acute poisonings can be met at persons, which are busy at works, or at those who have direct contacts with organic dyestuffs, pesticides, organic compounds of chlorine, with ammonia, preparations of cuprum etc.

Four periods are distinguished in clinical flow of the acute poisoning:

1 — latent (from the moment of poison entry till appearance of the first signs of a poisoning);

2 — increase of resorptive effect (from the first signs up to a typical clinical picture);

3 — the period of maximum effect of the poison, which is the most threatening condition, is a development of the collaptoid condition;

4 — rehabilitation.

The basic principles of treatment at acute poisonings are:

- Elimination of the poison from an organism;
- Neutralization of the poison (antidotal specific therapy);
- Symptomatic therapy (correction of affected functions).

Elimination of the poison from organism. Medical measures in case of entry of the poison deep into organism are directed on elimination of that part of the poison, which was not absorbed, and on speeding-up of elimination of that part of the poison, which was absorbed. The first is reached with help of a catharsis (artificially caused vomiting, application of purgatives, cleansing enema).

The catharsis is the main method of elimination of not absorbed toxiferous agent. This method is mostly effective within the first 6 hours. Method of the catharsis consists that 400 — 500 ml of liquid is introduced once only in the ventricles with help of a stomach pump, and then it is aspirated. Only 4 or 5 liters of the liquid will be used for the catharsis.

The catharsis is carried out also with an artificially caused vomiting.

The purgatives are prescribed for all patients. Magnesium or natrium sulfates in a dose of 0.5 g/kg dissolved in 200 — 300 ml water are used more often. Oil in a dose of 0.5 g/kg can be used as purgative.

Different methods of detoxification are applied for speeding-up of withdrawal of toxiferous substances that were already absorbed: an artificial diuresis, exchange blood transfusion, hemosorption, haemodialysis, peritoneal dialysis, forced ventilation of lungs.

The artificial diuresis is by an effective method of detoxification, which is applied at poisonings with matters that are moved from an organism by kidneys.

Osmotic diuretics (10 % manit solution, 10 % and 20 % glucose solution) and saluretic substances (Furosemide 40 — 200 mg) are used most often for its realization. Good effect is reached at combining these drugs. The introduction of diuretics is combined with constant replenishment of liquid losses. The constant control for an electrolyte mixture of blood plasma, volume of a circulating blood also is essential. Carrying out of the artificial diuresis in case of insufficiency of functions of kidneys, heart, and vessels is not recommended.

Exchange blood transfusion. This method is applied at poisonings, which are accompanied with toxiferous lesion of blood (formation of methemoglobin, hemolysis, and destruction of cholinesterase). The method is effective at the first 3 to 5 hours from the moment of poisoning; its essence is reduced to replacement of a large volume of patient's blood with a donor blood.

The hemosorption represents a perfusion of the patient's blood through a unit with ion-exchange resins absorbing toxiferous matters. This method is applied at presence of high concentration of poisoning matter in blood, deep coma, and impossibility of haemodialysis realization.

The haemodialysis with use of a unit «Artificial kidney» is applied at poisonings caused by poisons capable to be brought out from an organism during dialysis, and also in those cases when the poisoning becomes complicated due to acute kidney insufficiency. The principle of the haemodialysis is based on elective infiltration of nitrous matters, poisons, and electrolytes through a semipermeable membrane from blood into a dialyzating liquid owing to their concentrations difference.

The haemodialysis is not done in case of hemorrhagic phenomena availability.

The peritoneal dialysis is applied for removal of poisons, which during continuous time are located in a vascular bed and intertissue liquid. A preceding drainage of abdominal cavity, preparation of a special dialyzating liquid, which would not give the organism a capability to lose Na+, K+, P5+, HCO-3, is necessary for realization of the peritoneal dialysis. The peritoneal dialysis should not be done if there is a purulent process in the abdominal cavity.

The artificial ventilation of lungs is applied in case of abrupt respiratory depression and poisoning with matters, which are excreted by

lungs. It is done after a previous intubation of a trachea with the help of special equipment, and its efficiency is controlled on gas composition of the blood.

The neutralization of poison (antidotal therapy) can be carried out with help of physical, chemical and physiological antidotes.

The physical antidotes absorb toxiferous matter on their surface. Activated carbon, porcelain clay, starch, chalk fall into to this group. At last years the enterosorbents are applied. Activated carbon (in a dose of 1 g / kg) can be used at any poisonings, however it is ineffective in case of poisonings with acids, alkalis, alcohols. White chalk, starch are applied in combination with purgatives.

The physiological antidotes effect in accordance with a principle of functional antagonism. They react with those biological structures, to which the effect of the toxicant is directed.

Influencing of chemical antidotes is based on a specific chemical interaction with poison, as a result of what the last becomes deactivated. Thus, the antidote (Unithiolum, Tetacinum-calcium) by linkage, deposition, and replacement of competitive or other reactions transforms the poison into an innocuous matter, which is removed from an organism with urine, feces. See in Appendix 2 about the main means of antidotal therapy .

The symptomatic therapy is carried out pursuant to clinical developments of a poisoning. Thiopentalum-natrium, Fenobarbitalum (2-5 mg/kg), Sibazonum (10-20 mg), natrium oxybutyrate (50 — 100 mg/kgs) are applied at cramps. Adrenomimetics: Isadrinum (0.3-0,5 ml of 0,05 % solution), ephedrine (0.5-1.0 ml of 5 % solution), mesaton (0.3-1.0 ml of 1 % solution) are applied in case of depressing of cardiac activity, decrease of arterial pressure. Glucocorticoids (Hidrocortizonum 50-100 mg, Prednisolonum 15-30 mg) are applied for normalization of hemodynamics indexes.

Correction of water-electrolytic exchange and acid-alkaline condition obtains a special value at poisonings, which are accompanied with vomiting, diarrhea, and in case of poisonings with salts of heavy metals also. Whole blood, plasma is introduced intravenously at decreasing of volume of circulating blood.

Treatment and preventive measures of complications, which are developed at acute poisonings, has an important value. Edema of brain, acute hepatic and kidney insufficiency, hypothermal syndrome is observed more often.

The manifested lesions of water-electrolytic exchange demanding of adequate infusion therapy (5 % glucose solution) are marked at the hyper thermal syndrome.

Analgesics without narcotics (1 ml of 50 % Analginum solution, 1 ml of 4 % Amidopyrinum solution three times per day); neuroleptics

(Droperidolum 0.15-0.3 mg/kg per day) together with antihistaminic drugs (Dimedrolum, Suprastinum); intravenous dropwise introduction of the glucose-Novocainum mixture (500 ml of 5 % glucose solution, 50 ml of 1 % Novocainum solution) are prescribed for normalization of central mechanism of heat regulation.

The oxygen therapy is carried out as a preventive measures of posthypoxia of encephalopathy. Euphilin - 10 ml of 2.4 % solution, complami - 2 ml of 15 % solution, rheopolyglucin? - 400 ml, Trentalum - 5 ml of 2 % solution are injected. Prescription of 8 - 12 g of 20 % noothropole solution per day intravenous, medications that have an antihypoxic effect, natrium oxybutyrate 50 - 100 mg/kg, Sibazonum - 10-20 mg makes good effect.

EDEMA OF LUNGS

The edema of lungs is met most often owing to inhalation of toxic gases (chlorine, sulphur dioxide, ammonia, phosgene, formaldehyde, carbon oxide). Massive transudation of liquid enriched with protein into interstitial tissue and alveoli origins at acute edema of lungs. The causes of this can be: increased (over 30 mm of mercury or 4 kPa) hydrodynamic pressure in pulmonary capillary tubes; decreased (smaller 15 mm of mercury, or 2 kPa) oncotic pressure; increased permeability of alveolar membrane, increased rarefaction in alveoli (more 20 mm of mercury, or 2.7 kPa).

Liquid as a result of the intensive transudation at first transudates into perivascular space (the interstitial edema), and then in alveoli (the alveolar edema). Obstruction of respiratory tracts by foam (each 200-300 ml of liquid makes 2-3 liters of foam), which washes away the surfactant, comes. Its destruction and an intensive discharge of the foam leads to atelectasis of alveoli with deterioration of gases diffusion and to development of hypoxia, hypocapnia, metabolic acinarity.

A general condition of the ill is grave. Excitation and a comatose condition in grave cases are observed. More often patients are in constrained pose, they complain of shortage of air, sensation of fear. Short of breath of inspiration nature and cyanosis are marked. Stuffy, dry cough can be observed. Quantity of secreted sputum is augmented; the sputum has a foamy nature. The auscultative picture depends on many factors. At first it is rigid at the interstitial edema of breathing, dry crepitations come up later (squeezing of bronchioles with hydropic liquid).

Wet or whistling crepitant wheezes, which are spread to an apex and make difficulties for heart auscultation, are audible at the enroot region in case of the alveolar edema. If the edema progresses, a lot of various crepitations is listened; breathing with a screech can be listened at a distance. Foamy phlegm is secreted.

A sound with a “box” inflection is determined percutory? at the edema of lungs. The phenomena of stagnation in lungs, change of the heart configuration is present on roentgenogram.

Treatment. The medical measures by rendering of the first aid should be given for maintenance of permeability of the respiratory tract and struggle with the hypoxia, for a decrease of blood volume and pressure in pulmonary capillary tubes, on amplification of the left ventricle contractility, on preventive measures and treatment of complications.

The ill should be in a sitting pose with dropped legs. It renders assistance in depositing of blood in the lower departments and in decrease of pressure in vessels of the small circle of blood circulation. If the medical means and necessary equipment are absent, venous tourniquets on the lower extremities, which are superimposed on 10-15 minutes, are applied.

Immediate suction of foam at its large quantity is carried out with the help of catheters and suction means. The oxygen therapy for struggle with hypoxia is realized with 100 % oxygen with an antifoaming agent. Solution of alcohol, which reduces a surface tension of foam bubbles and transforms them into liquid prohibiting from edema increase, is applied as the antifoaming agent more often. Oxygen with speed 8-15 l / minute is passed through the Bobrov’s jar.

If these measures are not effective, intubations of the trachea and transfer of the patient on the artificial ventilation with increased resistance on exhalation (about 5-15 cm of waters, or 0.5-1.5 kPa) is recommended.

The catheter is recommended to enter into one of the large veins for maintenance of effective medicament treatment, and also for the control after the central venous pressure. Ganglioblockators (arphonad - 1000 mg of the preparation in 200 ml of 5 % glucose solution intravenous dropwise with a control of arterial pressure) are the most effective means at a hyperdynamic kind of edema of lungs with high arterial pressure. Solution of hydrochloride ephedrine (1 ml of 5 % solution in 10 ml of isotonic solution of natrium chloride) is applied for struggle with a possible arterial hypotension.

Preparations, which dilate both arteries, and veins (natrium nitroprusside 15 - 400 mkg/minutes dropwise, Prazozinum – 1.5-15 mg internally), are applied last years.

The venous means, which dilate vessels, in particular nitroglycerine are effective at hypodynamic version of the lungs edema at low arterial pressure and high central venous pressure.

Introduction of diuretic means (Furosemidum 40 - 120 mg) is one of the urgent means at the lungs edema. Euphilin is recommended in case of presence of bradycardia and bronchus spastic syndrome.

Narcotic analgesics (Morphinum hydrochloride, Promedolum, Fentanylum) are prescribed for decreasing of psychomotor excitation and shortness of breath.

Measures for decreasing of permeability through the alveolar membrane include application of glucocorticoids, preparations, which improve the condition of vascular walls (1-2 ml of 5 % Acidum ascorbinicum solution).

Trisaminum (200 — 250 ml 3.66 % solution) is applied for struggle with a hypoxia metabolic acidosis at edema of lungs. Natrii hydrocarbonas (1-2 mmol/kg) or its combination with Trisaminum in the ratio 2:1 is prescribed in case of toxic edema.

ACUTE FAILURE OF BREATHING

The acute failure of breathing is a condition, at which either the maintenance of normal gas composition of blood is not supported, or the last is reached due to straining of operation of an external respiration unit, what results in decreasing of functional capabilities of the organism.

Bronchus-lungs failure of breathing, which is caused by lesion of lungs parenchyma or respiratory tract permeability, is marked. At that such its forms are distinguished: а) obstructive form caused by blockade of respiratory tracts (aspiration of sputum, blood, vomitive mass), bronchitis, spasm of bronchus; б) restrictive, conditioned by limitation of lungs respiratory surface (pneumothorax, swelling in lungs); в) diffusive, caused by disorder of gases diffusion through the alveolar-capillary membrane (edema of lungs, pneumoscleritis). Besides, the centrifugal failure of breathing caused by lesion of function of the respiratory center as a result of exogenous intoxication with organophosphorus combinations can be met in clinic of occupational diseases.

Early disorder of consciousness is its distinctive feature.

Clinic. The acute failure of breathing that is accompanied by disorder of gas composition of blood is evaluated as decompensated, and as compensated one in case of changes of gas composition. There are three degrees of the decompensated acute failure of breathing: the I degree — a moderate shortness of breath, increase of the minute volume of breathing, tachycardia, arterial pressure is not changed; the II degree — a shortness of breath (frequency of breathing makes 25-35 per 1 minute), the minute volume of breathing is increased on 150 - 250 %, tachycardia (110 - 130 per 1 minute), arterial hypoxemia, which is manifested clinically by cyanosis, signs of light dormancy or euphoria, increase of muscle tone; the III degree - a shortness of breath and signs of hypoxia are sharply expressed, disorder of lungs ventilation and gaseous exchange increases and has a threatening nature. It is showed by the way of clinically sharp cyanosis, dilation of pupils expressed by dormancy; development of convulsions is possible.

The hypoxia coma develops at progressing of acute failure of breathing.

Treatment. One of the main problems to be decided while rendering the first aid consists in maintenance of permeability of respiratory tracts. For liquidation of obstruction of the upper respiratory tracts caused by retraction of the tongue back, it is necessary first of all to unbend the head in the atlas cervical joint and to draw out the lower jaw forward. Revision of the mouth and the mouthpart of the pharynx (deleting of foreign bodies in case of their detection is obligatory) are carried out in case of suspicion on obstruction.

The medical bronchoscopy or intubation of the trachea with the subsequent sanitation is carried out at obstruction of the lower departments of respiratory tracks. If it cannot be executed, laryngotomy and tracheotomy can be used. The intensive therapy is used for improvement of the lung drainage in case of large accumulation of sputum (chronic bronchitis, bronchial asthma).

The mechanical stimulation of cough is carried out at stoppage of sputum in respiratory tracks). A nose catheter is used for this purpose. It is entered in the larynx that will cause cough, and connection of a suction unit easies sputum deleting. Proteolytic ferments, preparations containing Iodum, and antibiotics are introduced for its dilution with the help of ultrasonic inhalers.

Bronchial washing with the help of a bronchoscope is applied at expressed bronchus obturating syndrome.

The spontaneous ventilation of lungs is used for liquidation of some types of obstruction, improvement of perfusive ventilation ratio, intensification of the sputum drainage.

The oxygen therapy is performed with the purpose of liquidation or decreasing of hypoxia. Application of helium-oxygen compositions (40-50 % oxygen and 50-60 % helium) is more effective. Such inhalation during 1.5-2 hours improves ventilation of lungs and the ventilation perfusive ratio.

Artificial ventilation of lungs is carried out for elimination of alveolar hypoventilation in grave cases of the acute failure of breathing.

SPONTANEOUS PNEUMOTHORAX

The spontaneous pneumothorax is an intake of air in a pleural cavity as a result of sudden damage of pleura.

The pneumothorax at occupational pathological patients is referred to so-called symptomatic one; it is met most often at patients with silicosis of the III stage at presence of air bubbling emphysema at them.

Clinic. The clinical picture is marked with quantity of air, which has penetrated, and characteristic of an opening in the pleura. The beginning of this condition is a sudden, acute pain, which has connection with breathing, is felt in a site of the thorax cage and is often accompanied with a shortness of breath and cyanosis. Patients complain of dry irritable cough of reflexive nature (irritation of pleural reflexogenic areas can shock). The affected half of

thorax cage of lungs remains behind in the act of breathing; the vibration of voice is sharply weakened or missed. Tympanitis is determined at percussion; borders of liver are displaced down in case of development of the pneumothorax to the right. The weakened breathing is determined auscultative. The borders of heart are displaced in the counter side in case of concentration of a plenty of air in the pleural emptiness or at the valve pneumothorax; a bend of the large vessels is possible and this can cause death of ill because of a grave pulmonary heart failure and asphyxia.

The signs of the acute pulmonary heart can be seen on the electrocardiogram.

Treatment. The urgent measures are aimed at struggle with pain - Morphinum (1 ml of 1 % solution), Omnoponum (1 ml of 2 % solution), Promedolum (1 ml of 2 % solution) subcutaneously or intravenously.

Strophanthin (0.5 ml of 0.05 % solution), Korglykonum (0.5 - 1 ml of 0.06 % solution) are prescribed at cordial failure presence; Cordiaminum (2 ml), mesaton (1 ml of 1 % solution) intravenously or subcutaneously is prescribed for normalization of activity of the cardiovascular system. Oxygen inhalation is recommended.

Urgent decompression therapy by introducing of a needle with a broad open space or trocar in the region of the gas bubble (a puncture of the pleural space in the II or III intercostal regions on the middle-clavicular line) is necessary at the valve pneumothorax and increase of phenomena of breath shortness, cyanosis, inflation of cervical veins, asphyxia.

To adjust a system of communicating vessels for vacuum drainage is best. The vacuum drainage of pleura should be conducted till full and stable straightening of the squeezed lung (under control of the rontgenologic examination).

THE ASTHMATIC STATUS

The asthmatic status is a condition, which is characterized by three main signs:

1) Fast increasing of bronchial obstruction;

2) Absence of effect from introducing of sympathomimetitics;

3) Increasing of respiratory failure.

Clinic. Three stages of the asthmatic status are distinguished: the I stage — *attack of bronchial asthma* with full absence of effect from sympathomimetitics. Acute degree of respiratory failure, expressed anxiety, and fear are marked at patients in this stage. Dermal covers are pale, cyanotic. There is a “box” sound percussively above lungs, lung’s borders are broaden. Auscultative the breathing here and there is weakened and vesicular, rigid; dry crepitations are listened; the II stage – *the increasing respiratory failure*, appearance of regions of a "dumb" lung; decreasing of quantity of dry crepitations, appearance of regions, where the crepitations are not listened

that is connected with the bronchial obstruction. It is the stage of decompensation. The conditions of the patients are determined as a very grave. Asphyxia of the obstructive type increases, breathing is superficial and very often; pale cyanosis is found out. The thorax is in the condition of a deep inspiration. Crepitations during auscultation are not possible to be heard. Sphygmus is frequent, of weak filling and strain. Arterial pressure is lowered; cardiac sounds are deaf. Signs of overload of the right departments of heart can be found out on the electrocardiogram;

The III stage is hypercapnic coma, at which the CO_2 pressure increases until 80-90 mm of mercury column and the oxygen pressure abruptly falls down to 40-50 mm of mercury column. The patient loses consciousness, his/her breathing becomes deep with a lengthened exhalation, cyanosis increases; the intravenous pressure drops, the sphygmus becomes thread-like. Death comes from asphyxia.

Treatment. Introducing of 2.4 % Euphilinum solution intravenous by drops in a dose of 10-15 ml at its dilution in 200 ml of sodium chloride or 5 % glucose; introduction of glucocorticosteroids intravenous by drops: 100–150 mg Hidrocortizon or 90 – 120 mg Prednisolon in 200 ml of isotonic solution of sodium chloride are applied at the I stage of the asthmatic condition. Introduction of the preparations is repeated every two hours. Glucocorticosteroids— Prednisolon per 10 mg, Dexamethazon in the dose of 2 to 4 mg are prescribed internally in the absence of effect. The drugs should be given in such dose every two hours up to sputum going out and patient’s condition improvement. A large volume of liquid up to 1-2 liter per day is entered under a diuresis control. Humidified oxygen is prescribed.

At the II stage of asthmatic condition the introduction of hormonal drugs intravenous and also in ppatients with the increase of the dose by 1.5 to 2 times, introduction of Euphilinum intravenous should be prolonged. 200 ml of 4 % soda solution is introduced intravenously in drops against acidosis. Introduction of the liquid is prolonged with monitoring of diuresis, oxygenotherapy. Prescription of adrenalin in process of treatment of ill with the asthmatic status is counter-indicative, as it can cause an effect of "ricochet" — acute strengthening of bronchial spasm owing to a functional blockade of p-adreno-energetic receptors. If an effect from the carried out therapy is absent, it is necessary to transfer the ill in a ward for intensive care or in a ward for reanimation for monitor control of breathing function and cardiovascular activity and for subsidiary ventilation of lungs.

Patients with asthmatic status of the III stage - hypoxic coma — should be prescribed pulmonary artificial ventilation under conditions of a reanimation ward or a ward of intensive care. Introduction of hormonal preparations, broncholitics, struggle with respiratory failure, disorders of acid-alkaline balance should be prolonged. Criterions of improvement of patients condition are weakening of asphyxia feeling, beginning sputum

going out; ill becomes more tranquil. Quantity of dry crepitations in lungs decreases, and to the contrary in the stage of the "dumb" lung their appearance testifies about improvement of patient's condition. Scheduled glucocorticoid therapy alongside with other measures - daily dose of 20 to 30 mg of Prednisolon during the nearest days, 12-16 mg of Dexamethazon – should be prescribed after moving the ill off the asthmatic status.

ACUTE HEMOLYTIC CRISIS

The acute hemolytic crisis is predetermined by effect of toxin substances, which are strong hemolytic poisons (arsenious hydrogen).

This condition is characterized by fast development of general weakness, back pain, cold and body temperature rise, cerebral manifestations (headache, insanity, loss of consciousness, meningitis symptoms, lesion of vision), pain in bones and joints.

General paleness of dermal covers, which is combined with icteric coloring of sclera and mucosas owing to hemolysis, occurs. The acute renal failure, down to full anuria and uremia can arise. Protein, cylinders can be found in urine in case of diuresis decreasing. The urine obtains brown coloring. Contents of hemoglobin, erythrocytes in blood are decreased, the haematocrite index is decreased, plasma becomes of icteric or pinkish color. A content of reticulocytes in blood is abruptly increased; level of indirect bilirubin, free hemoglobin, residual nitrogen and urea in plasma is also increased. At worsening of chronic forms of anemia the increased lien is usually palpated.

As a rule, all forms of the acute intravascular hemolysis are accompanied by more or less expressed signs of the syndrome of blood intravascular coagulation, the tromboembolic complication, infarcts in organs and bones with presence of strong pain syndrome can be observed at some of them.

Treatment. Warming of the body (heaters), intravenous introduction of 100 – 200 mg of Prednisolonum and 10 000 Units of Heparinum (with the purpose of microcirculation deblocking and tromboembolism prevention). Medical plasmaphoresis with the purpose of fast moving out of blood the agent, which caused the hemolysis, anti-erythrocyte antibodies and immune complexes, is recommended in stationary conditions in case of poisoning with hemolytic poisons. Donor erythrocytes in the form of clean washed erythrocytes suspension are introduced after 5-6 days of their storing if the transfusion therapy is necessary. The transfusion therapy should be performed carefully, as it can provoke the second wave of the hemolysis.

HEPATIC COMA

Appearance of the hepatic coma is possible in case of poisoning with matters of hepatotropic effect (tetra-chlorous hydrocarbon, tetrachloroethane,

nitrates, toluene). It is referred to so-called endogene hepatic coma (acute dystrophia of liver) or to one from three clinical pathogenic kinds of the hepatic comas caused by poisonings. The morphological substratum at such coma is hard dystrophic mainly acute massive necrotic changes of the hepatic parenchyma. Substances rendering a toxic effect on the brain as well as accumulations of aromatic amino acids and amino acids containing sulfur, amino acids that income from the intestine signifies for its pathogeny.

Four stages of the hepatic coma are marked depending on expressiveness of psychomotor disorders and changes on electroencephalogram.

Stage I. Precoma. Development of the hepatic coma can be sluggish: emotional unbalance, sensation of worrying, apathy, euphoria occur; intellection is slowed down, orientation is degraded, distresses of sleep take place. Often patients become ingenuous. Transient light blackouts of consciousness, which resemble a soporous condition, happen. Thus, changes at this stage are showed mainly in disorders of psychics. Besides, appetite is reduced, various developments heightened haemorrhage are possible; patients grow thin fast.

Transient disorders of consciousness are characteristic for patients at the stage of the precoma with the porto-caval failure. Electroencephalogram is not changed or is slightly affected: b–activity predominates, the low-amplitude curve is smoothed, irregularity of a β-rhythm in amplitude or its exaltation takes place, sometimes Θ and Δ-waves are present.

Stage II. Coma in stage of development. This stage is accompanied by further more deep disorder of consciousness. The ill is disoriented in time and space, attacks of excitation are shifted by depression and drowse. Delirious conditions with cramps and motor excitation, during which the patients try to run out from the ward, become aggressive, and in a number of cases dangerous for those who are close to them, arise periodically. Flapping tremor of fingers, lips, eyelids, ataxy, dysarthtia and various changes of a muscle tone, strengthenings of reflexes appear. The further distresses of handwriting take place.

Fever, dyspeptic disorders take place; icterus is possible. The breathing becomes frequent and deeper.

Iirregularity of a-rhythm on frequency, not rough, but rather steady α- and the Δ-waves are found out on electroencephalogram.

Stage III. Stupor. It is characterized by the expressed psychosis, inclination to continuous sleep, but with periodic awakening. The tremor of fingers is expressive.

Electroencephalogram mirrors rough changes. Θ- and the δ-waves prevail, a-αand β-activity disappear. The hypersynchronous Δ - waves dominate in the terminal stage; the electroencephalogram comes nearer to an isoline.

Stage IV. Coma. Consciousness is lost, reaction to pain irritations disappears, rigidity of extremities muscles and nape is marked, the face is similar to mask, pathological reflexes (Babinskiy's, Gordon's, Zhukovskiy's), and in a few of cases prehensile and yoke reflexes appear. In the terminal period the pupils dilate, their reaction on light disappears; paralysis of sphincters and stop of breathing comes.

Clinic. Hemorrhagic and hydropic-ascites syndromes, hepatic smell from the mouth, jaundice (in case of a massive necrosis of the parenchyma can be absent) comes up. Feeling of a strong pain in the right subcostal region and decreasing of the liver arises at acute massive necrosises of the liver parenchyma.

The so-called hepatic-cordial syndrome is possible at hepatic coma owing to disbolism in the myocardium. It is showed as a result of phenomenon, which has obtained a title «pecker's knocking» (premature appearance of the II tone), the interval Q-T is lengthened also the tooth T is expanded. Tachycardia and hypotonia takes place.

Renal plasma flow and glomerular filtration decreases, concentration of natrium in urine is reduced, its density is increased, and excretion of water decreases.

Leukocitosis, hyperazotemia, high level of gall acids, low level of the feneral protein and albumines and high contents of γ - globulins, abrupt decrease of contents of blood coagulation factors, cholesterol, potassium in serum and erythrocytes found out in blood. Bilirubinic ferment dissociation that is an increasing of general bilirubin and decreasing of aminetransferases activity, which were earlier increased, and specific hepatic ferments, including cholinesterase, is observed.

The hepatic coma with the acute and gradual beginning is marked. The premonitory period at the disease flow lasts from 1 till 3 hours, then comatose condition with delirium, excitation and vomiting, which one lasts some hours, comes soon, then patients fall in a deep coma and die in 1-3 days in the most cases.

Sluggish flow of the disease is a premonitory stage, which takes some days and sometimes weeks. It is accompanied with gastrointestinal distresses, icterus, and hepatic encephalopathy. Precoma is accompanied with acute head pain, psychomotor excitation, delirium; all lasts 1-2 day.

The comatose condition is appeared in view of acute decreasing of liver, paresis and paralyses of extremities with pathological pyramidal signs.

Diagnostic. It is grounded on outcomes of anamnesis collection, clinical picture, and on biochemical and encephalographic changes also. "Clank" tremor and change of electroencephalogram is a significant symptom of the threatening coma. A special diagnostic meaning has such biochemical symptom of the hepatic-cell precoma as decrease of blood coagulation factors

in blood: prothrombin, proconvertin. General contents of these factors are decreased in 3-4 times.

Treatment. Patients with hard, hepatic-cell failure demand a doctor to observe them carefully. It is important not miss appearance of “hepatic” smell, psychoneurological signs of precoma and the beginning of coma development. Each day it is necessary to evaluate the size of the liver, daily urine. Fall of activity of specific hepatic ferments and contents of potassium in serum of blood indicates the condition of the lever failure development. The essential help in early diagnostic of hepatic encephalopathy makes electroencephalogram.

Protein is completely eliminated from diet, introduction of albuminous drugs parenterally is terminated. Meals with food value of 1600 kilocalorie is supported with introducing of 5-20 % glucose solution through a stomach pump or intravenously. 20 g, then 35 and 50 g of protein is permitted in 7-8 days after outlet of the ill from coma.

Drugs in case of comatose condition development are introduced intravenously through a subclavial catheter. Acid, α - arginine is applied with the purpose of neutralization of the ammonia absorbed with blood; drugs, which improve exchange of hepatic cells – Lipocoic Acid, and Essentiale are injected.

Ornicetil is a-ketogluconate of ornithine, which binds ammonia. It is used at cirrhosises with the expressed hepatic-cell failure, chronic portacaval encephalopathies. The dose is from 5 till 25 g a day intravenous or from 2 till 6 g a day intramuscularly.

A large quantity of glucocorticoid hormones is introduced for patients with hepatic-cell failure. The cardiac and vascular preparations apply at the hepatic coma; antibiotics of a broad action spectrum (Kanamycinum, ampicilline) are applied for suppression of intestinal flora habitability and of bacterial complications warning.

High clysters, evacuating through a stomach pump everything that is contained in the stomach and duodenum, are prescribed for struggle with toxic products, which are absorbed from the gastrointestinal tract.

Sodium bromide and chloral hydrate is introduced through clysters in case of presence of psychomotor excitation.

Sequence of medical measures at hepatic coma is as follows:

5 % glucose solution with a dose up to 3 liters a day with cocarboxylase 300 mg, vitamin B12 500 microgram, lipoic acid 180 mg is introduced with rate 20-30 drops a minute through an under-clavicle catheter intravenously by drops.

Prednisolon 150 mg is introduced intravenously in jet and further per 90 mg every 4 hours.

α-argirine 25 mg is introduced intravenously in drops and further in the same dose every 8 hours, or 100-150 ml of 10 % glutamine acid solution and

further in the same dose every 8 hours, or ornicetyl 10-25 g intravenously during one day.

Kanamycin 0.5 g or polymyxin M 500000 U or ampicillin 1 g every 4 hours is introduced through a stomach pump.

The intestine is cleaned through an enema every day. Blood exchange transfusions of 5-6 l are executed every day during 7-10 days. Introducing of oxygen is carried out through a nose catheter with rate 2-4 liter a minute. 200-600 ml of 4 % soda solution is introduced in case of metabolic acidosis development.

Potassium preparations are introduced up to 10 g a day at expressed metabolic alkalosis. Ablution of stomach with an ice-cold isotonic sodium salt solution, introducing of native plasma or fresh citrate blood on 100-200 ml three times a day is carried out at hemorrhagic syndrome.

METALLIC FEVER

A peculiar symptom-complex, so-called metallic fever can arise at workers during a casting, melt, and welding operations, as well as at metals gas cutting.

Metallic fever is considered as a manifestation of not specific effect of metal (not chemical nature of metal, but its physical characteristics matter for development of the fever). Its vapors during getting cool and oxidation in air form a highly dispersive aerosol, which in a case of contact with epithelium of bronchi and alveoli will cause a denaturation of cellular protein. The protein "aseptic" fever (reaction to foreign proteins) is a consequence of denaturation products falling into blood.

Metallic fever arises most often during melting of zinc, as its melting point 419 °C is rather low; however similar reaction will also cause vapors of the other metals: cuprum, iron, mercury, lead, nickel, electric welding aerosols and so on. This disease is mostly met at electric welders and oxygen cutters, in particular during work in an enclosed space that is characteristic for a shipbuilding industry.

Clinic. The metallic fever flows in form of acute attacks, which resemble attacks of malaria.

Premonitory period comes some time after inhalation of vapors. Sweetish taste in the mouth, indisposition, headache, weakness, nausea, vomiting, and drowsiness are felt. Dry paroxysmal cough, pain behind the breastbone, heavy breathing can take place. Hyperemia of conjunctivas, yawning, clinical developments of bronchitis, and lungs emphysemas are marked. The indicated period can last 4-5 hours.

The second period of the disease represents "a genuine fever". It is characterized with an acute rise in temperature up to 39-40 °C, shaking fever; the critical temperature drop occur in some hours (usually 5-8) with excretion of copious sweat.

Neutrophilic leukocitosis, shift of the formula to the left, subsequent lymphocytosis, transitional hyperglycemia are observed in blood. Tracks of protein, leucocytes, cylinders, increased quantity of urobilin, sometimes porphyrin is observed in urine. Allergic manifestations in form of herpetic efflorescence, nettle-rash disease can take place. Usually the attack passes by rather easily.

Feeling of a worn out person, general indisposition can last 2-3 days.

Prevalence of symptoms of nervous system lesion (headache, drowsiness, delirium, condition of a consciousness loss), vegetative distresses (fever, reddening of a face skin), lesion of functions of a gastrointestinal tract and liver is possible in the period of fever. It is necessary in particular to mark a syndrome of lesion of the external respiration function. Pulmonary failure of the reflex nature, bronchial spastic stricture takes place in a prodromal stage. Acute swelling of lungs, blurring of the pulmonary picture is observed on the roentgenogram even at the first hours of the poisoning. A development of unilateral or two-sided cellular pneumonia, which sometimes flows past without increased temperature, is possible in the second phase (period of “true fever”). Fine-cellular two-sided diffuse shades are visible on the roentgenogram.

The fever in case of its multiple repetition can cause changes of the organism reactivity, large sensibility to infectious diseases. Diseases of respiratory organs: chronic bronchitis, emphysema of lungs, attacks of asphyxia become more frequent; activating of tubercular infection and development of functional failure of vascular system are possible.

Occupational nature of the disease is usually determined without particular complexity. It is necessary to allow working conditions and its characteristic, suddenness of development, some features of the clinical picture, recurrence of its originating and a group nature of the disease.

Treatment. Intravenous introduction of 40 % glucose solution with ascorbic acid (in quantity 300 mg), Dimedrol, plenty of robust sweet tea, or coffee is prescribed. Cardiovascular means, oxygen, rest, as well as warm, alkaline inhalations are applied, if necessary. Generally accepted treatment is recommended at development of pneumonia.

Verification of the ability to work. Temporary incapacity for work during 5 to 7 days; it is more long-out depending on a basic disease at development of pneumonia or exacerbation of any chronic disease.

Preventive measures. Melt of non-ferrous metals in electric furnaces; mechanization of work connected with cast of metal, effective ventilation. Application of respirators at presence of high concentrations of metal vapors in air of the working area is necessary. It is necessary to take a warm douche for prevention of origination of feverous processes after working.

Chapter 10

BASIC PRINCIPLES OF MEDICAL LABOR EXPERTISE AT OCCUPATIONAL DISEASES

The exact installation of the diagnosis with allowance for etiological factor is the main task at solution of expert problems in case of occupational diseases. The medical labor expertise of occupational diseases has essential singularities of the legal order, as admission of some disease referring to an occupational disease conducts behind itself serious social - legal consequences and is connected with granting of considerable material privileges. The following features concerns these peculiarities: necessity to allow for the operational list of occupational diseases (Appendix 1), knowledge of privileges and advantages, to which patients suffering from affliction of occupational pathology (full payment for a sick-list of occupational disability irrespective of length of service, granting a sick-list of occupational disability, increased sum of pension in case of steady invalidity, payment of compensation for a loss of capacity for work appointed in percents) have rights.

It is necessary to know the order and specificity of legalization of the medical documentation for a correct solution of complex problems of the medical labor expertise. Regulations of invalidity medical certificates issue, indications and contraindications to use of the certificates of occupational invalidity, consideration of cases of its non-rational usage, regulations on correct filling of the certificates of medical examination in medical commissions of experts refer to it. In each concrete case it is necessary to decide, whether an experience of activity for development of the given disease is sufficient or not to take into consideration a connection of the disease with the profession; if is it possible or not to keep the ill on former activity. The objective data, which concern patient’s condition and enable to compare functional reserves of the organism, in which there were pathological changes, with requirements of the trade, have essential value.

The necessity of change of the working conditions determines some features of expert approach to occupational patients. They more often are transferred to physical inability at considerably smaller developments of pathology, than patients with general diseases.

Such features, as necessity of taking into account versions of some chronic diseases flow, for example, development of clinical manifestations after termination of receipt of etiological factor from outside on the basis of its depositing or functional accumulation; significance of specific functional and other auxiliary exploratory methods, for example, pallesthesiometry at vibration pathology, detection of poisons in biological substances at intoxications; obligation of indication on the etiological factor, which in a

number of cases, for example, at allergic diseases, produce considerable difficulties, also refer to medical labor expertise peculiarities bound with ascertainment of the occupational pathological diagnosis.

At transfer to another work, it is necessary to allow a number of social moments: interests of works on maintenance of qualified personnel; interests of a patient, his/her reluctance to part with in particular qualified, well paid work. Allowing for this, it is recommended to fulfill at first such medical preventive measures as temporary transfer to another work on the so-called additionally paid sick-list, realization of an out-patient treatment, sometimes with a subsequent granting of the next holiday, sanatorium-resort treatment or hospital examination.

The disablement can be temporary or constant. The speech about a temporary disablement goes in the event that changes of health condition have reversible nature and in a small time interval the capacity for work is completely restored. The labor forecast is favorable. It is met at the initial or moderately expressed forms of the occupational disease. Medical consulting commissions usually decide the problem of a temporary disablement.

The temporary violation of capacity for work can be full and partial.

The temporary full disablement arises when an ill is temporarily not suitable to any kind of activity during a short period of time. Such kind of violation of capacity for work can arise:

1. At expressed forms of an occupational disease, if an ill requires treatment in hospital and a directive to a commission of experts.
2. At acute condition of occupational diseases.
3. At moderately expressed forms of the disease up to granting of the occupational medical certificate (with the purpose of treatment efficiency increase and increase of term of release from influence of an unfavorable occupational factor).

The partial temporary violation of capacity for work is determined in a case when an ill can not work on a main trade demands temporary transfer to any other work irrelevant with influencing of unfavorable factors.

About a steady violation of capacity for work is spoken, when changes in a health condition take a steady and sometimes nonreversible nature. The labor forecast is unfavorable. The disease does not give any capability for the ill to perform work on the specialty, or any other.

Evaluation of capacity for work, and also determination of groups and nature of physical inability is performed by medical-social commissions of experts. They have to decide problems on a disability by workers or employees, which have received a mutilation or other damage of health bound with their work, and about a size of the caused loss.

A steady violation of capacity for work as well as temporary one can be full and partial. The I and II groups of physical inability are determined at full disablement, the III group - at partial. However, a steady violation of capacity

for work not always means a physical inability, as it can stipulate different kinds of limitation of capacity for work yet. For example, a metalworker, which worked with a pneumatic tool, can work after diagnosing of vibration illness on the same trade, but without use of a pneumatic tool and under condition of exception of considerable physical stress. Therefore, a concept of «steady violation of capacity for work» is a broader concept than a concept «physical inability". If the steady violation of capacity for work results in the termination of occupational work or necessity of lowering of proficiency, and also to acquisition of other trade, then the speech can go about physical inability.

The physical inability of occupational nature is established in the next cases.

1. When the diseases, which are etiologically connected with influencing whether of that or other production factor and also cannot arise in other conditions. The speech goes about so-called specific occupational diseases (silicosis, vibration illness).

2. At the diseases, which are not occupational but under certain concrete conditions can become such (nonspecific occupational diseases): bronchial asthma at medical workers, who are contacting with antibiotics, tuberculosis at workers of TB prophylactic centers.

3. In those cases when an early existed occupational disease was added with complications or any consequences of the indicated disease have affected.

4. At occupational diseases, which play essential role in development of the condition caused by presence of general disease, irrelevant to the trade.

Concept «occupational certificate», or «labor medical certificate» deserves a special attention. If a worker or an employee is temporarily disabled owing to disease connected with his work but can execute another work without prejudice to production and without violation of normal order of treatment, he/she is temporarily transferred to this work pursuant to a conclusion of medical-consulting commission or, if such commission is not exist, of a doctor after confirmation of his/her solution by the chief medical officer.

This transfer is realized by administration of firm or entity. If the new activity, on which the worker is temporarily transferred, is paid below than his/her permanent work, a pecuniary aid is granted him/her on a period of the transfer to base of the sick-list but no more than for two months. Grounds for application of the occupational certificate are the initial or moderately exhibited forms of diseases, which are capable of return development. Such conditions are indispensable for the greater efficiency of these measures: 1) rational provision of job placement for patients (full exception of influencing of the unfavorable factor that has caused the disease and also other factors, which are capable to worsen its flow); 2) simultaneous realization of rational

and active treatment (it is recommended to join the transfer to other activity with staying in a prophylactic ward); 3) sufficient duration of the transfer to a temporary job: the least term it equals to 1-1.5 months at diseases of “local” nature the initial form. At diseases, which are accompanied with more expressed general violations (chronic intoxications and that similar), it is recommended not only to grant the occupational certificate on maximum two months term, but also to prolong term of stay the ill outside of any contact with the harmful factor by means of his/her transfer to harmless job taking into consideration the certificate of medical-consulting commission, granting him/her the next holiday after ending of term of the occupational certificate.

The order of determination by medical-social commissions of experts of a degree of loss of occupational capacity for work in percentage to workers, to which the damage of health was caused in connection with fulfillment of labor responsibilities, is determined by the order of Ministry of Health of Ukraine No. 212 from 22.11.95.

Paying indemnification for a damaged person in accordance with this document includes:

- Compensation of the lost earnings or its respective part by a damaged person depending on a degree of loss of occupational capacity for work;
- Payment of a lump sum to a damaged person (members of the family and dependents of the died) in established cases;
- Compensation of costs on the medical and social help, nourishing diet, prosthesis, additional care and so forth.

In cases, if an acute exhibited restriction of habitability has come owing to a working mutilation or an occupational disease and that has resulted in acute exhibited social de-adaptation and need for a constant extraneous care or help, 100 percents of loss of occupational capacity for work are established.

In cases, if the sharply exhibited limitation of habitability, which has resulted to the exhibited social deadaptation owing to a working mutilation or an occupational disease, has come, but the indicated lesions do not call for need for the constant extraneous care or help for a sufferer and the sufferer can perform the job in adapted conditions, the loss of occupational capacity for work makes from 60 up to 70 percents.

In cases, if the considerable decrease of capabilities of social adaptation comes owing to a working mutilation or an occupational disease, the percent of a disablement given below is established with allowance for of social factor and availability of the work places:

а) 60-50 percents at disablement on the basic trade and possibilities of job placement for the sufferer on work place of lower proficiency;

б) 40-30 percents, if sufferer can work on his/her profession, but under condition of change of working conditions and wage cut.

If grounds for assignment of group of physical inability owing to a working mutilation or other damage of health is absent, but the damaged person due to a health condition demands minor restriction of work to be performed, that results in a minor decrease of earnings, Expert Commission establishes up to 25 percents of loss of occupational capacity for work.

Solution of Expert Commission about a causal connection of the occupational diseases with a concrete trade (position), which rendered assistance to development of the disease, and its approach, is taken on ground of conclusion of a specialized prophylactic institution.

The proprietor of the enterprise provides medical, social and occupational rehabilitation of sufferer pursuant to conclusions of Expert Commission. An appeal against the decision of Expert Commission about determination of a degree of loss of occupational capacity for work is realized in percents according to Section V items 34, 35 «Regulations for medical-social expertise», approved by Cabinet of Ministers of Ukraine as of February 22, 1992 No. 83.

Chapter 11

RESEARCH METHODS IN OCCUPATIONAL PATHOLOGY CLINICAL INSTRUMENTAL METHODS

Outcomes of the instrumental research methods, which are rather widely applied today, essentially supplement data of clinical researches, that enables to obtain more detail representation concerning a functional condition of a number of systems and organs, which take participation in formation of pathological process, provide indemnification and valuable capacity for work at those or other concrete conditions. The differentiated approach to problems of therapy with allowance for changes, which take place in a number of cases, is possible. The dynamic observation enables to control efficiency of realizing treatment.

The rheography is a bloodless method of research of a general blood circulation and blood circulation in organs. The method is based on registration of changes of electrical conductivity of a organ or vessels taking place owing to volumetric oscillations of blood in them. The electrodes are fastened in a zone of research (shank, foot, forearm, hand) for a record of the rheogram of vessels of extremities. Pheoencephalograms are recorded in a zone of the frontal bone and the mammiform outgrowth, and also in the lower department of a hair part of a occiput; the indicated taps image condition of blood filling of the basin of the internal carotid artery and vertebral vascular system accordingly. The researches are carried out in strictly symmetrical zones, thus a contact of electrodes and interelectrode layings with skin of the patient and correct balancing of the device are controlled.

The recording of the rheograms is realized with the help of such rheograph as РГ-2-02, 4РГ-1Г. The electrocardiographs and polygraphs are used as recorders.

The rheogram represents a curve, which resembles a sphygmogram and consists of a not long ascending part (anacrotism) and a descending part, which is more stretched in time (catacrotism). The following parameters are determined: amplitude of the rheogram in Ohms; a rheograph index is a ratio of amplitude of a systolic wave (in mm) to a calibration pulse (in mm). These two parameters characterize pulse of blood filling of the investigated region; the level of incision characterizes a peripheral resistance; the time of maximum systolic filling of vessels characterizes tone and elasticity of arteries; the maximum speed of a blood filling characterizes condition of the retractive function of the myocardium, rate of blood filling of large arteries.

Type of this method is a tetrapolar thoracal rheography pursuant to a technique of Kubichek or Tishchenko.

The rheography method is rather widely used for determination of parameters of the central and peripheral hemodynamics at dust diseases of

lungs, effect of physical (vibration, noise) and chemistry factors: tetrapolar thoracic rheography, rheovasography, rheoencephalography, rheohepatography and so on.

The oscillography is a method of research of arterial vessels, which enables to receive information on elasticity of the vascular wall, levels of maximum, minimum and mean arterial pressure.

The pressure in collar, which serves at the same time as a receiver of pulse oscillations, is raised up to the level, which is a little higher of the expected systolic pressure. Registration of the arterial oscillogram is realized in a mode of a smoothly varying decompression.

The arterial oscillogram reflexes pulse oscillations of tissues volume, which depends on non-constant ratio between pressure and backpressure in collar. Such oscillogram is an objective document, which registers a level of systolic and mean arterial pressure. The discontinuous increase of amplitude of oscillations during decompression (Mx) and the same their acute decrease when the pressure in collar becomes equal diastolic (Mп), serves an indicator of these parameters. A pressure level in the collar, at which oscillations reach the greatest amplitude, is accepted to consider as a level of the mean arterial pressure (My).

Values of arterial pressure obtained by this method exceed similar values obtained after Korotkov's method a little. An average value of arterial pressure for able-bodied people equals approximately 90 mm of mercury.

This method is applied at vibrational disease, obliterating endarteritis under effect of low temperatures and so on.

Thermometry. The method allows evaluating a skin blood supply. The thermoelectric method is the most precise.

The thermoelectric device consists of a galvanometer and several thermoelectric couples. Indications from any thermoelectric couple can be read by the galvanometer that enables to log temperature on miscellaneous sites of a body practically simultaneously. Temperature on hands skin is equal from +25 °C up to +31 °C, on feet from +25 °C up to +27 °C, on forehead from +32 °C up to +33 °C.

The method is applied during examination of patients with vibrational disease, obliterating endarteritis.

Cold assay. The patient's hands are put in cold water for 3 - 5 minutes. The assay is considered positive in case of albication of phalanxes, several fingers or hands. Expressiveness of these violations reflects the vessels lesion depth. Duration of the phalanxes albication shows a period of blood flow recovery that can be also estimated due to period of time of temperature recovery (table).

Electrothermy of hands fingers skin	
At able-bodied person	**At vibration disease**
Initial t°C	
+28°C and >	+24°C and <
After cold assay	
From +24°C до +21°C	+16°C and <
Time of t° recovery after a cold assay	
After 20 - 30 minutes	After 40 minutes and more

It has a diagnostic value at examination of patients with vibrational disease, obliterating endarteritis.

Capillaroscopy. The method enables to evaluate a degree of fine vessel changes. The capillaroscopy consists of a microscope with a lamp, which enables to carry out researches at incident light. Capillary of a transient fold of a nail-bed of fingers or toes should be investigated. Significant amount (up to 15 - 20 in a field of vision) of capillary loops are seen on a light-pink or pink background in the norm.

Blood flow in them is uninterrupted, rapid.

A picture of capillary tubes at vibrational illness can be different. Considerable narrowing or disappearance of arterial pipe, quantity of visible capillary tubes decreases, their torsion is marked characterizes a spastic stricture. The blood flow becomes pulsating. Atonic condition is showed in form of sharp distension of arteries and veins. Permeability of capillary tubes is upset, as a result of which small-sized haemorrhages occur. Blood flow becomes slower. Joining of signs, which characterize spastic and atonic conditions (Fig. 10), is marked at spastic-atonic condition.

Norm **Spasm** **Atony**

Fig. 10. Capillaroscopy of a nail-bed.

Microcirculation of bubble conjunctiva vessels. Research of microhemodynamics in a vascular bed of sclera conjunctiva allows evaluating microcirculation. With this purpose a biomicroscopy of conjunctival blood

flow is carried out with fulfilment of microphotography and subsequent qualitative and quantitative evaluation of sclera conjunctiva vessels condition. The quality assessment includes determination of blood flow nature, ratio of quantity of operating microvessels, presence of spastic-atonic and rheological changes. The quantitative assessment foresees calculation of conjunctival index on ground of the obtained microphotographs.

Algesiometry. The method based on determination of depth of a needle deepening before coming of pain sensation enables to find a threshold of pain sensitivity with help of algesiometer installed upright and a calibrated scale, which is revolving. This value on a hand rear surface makes no more than 0.5 mm in the norm. At vibration diseases,

Increasing of this factor is watched at vibration illness, diseases of the peripheral nervous system, which are connected with a local muscle overstrain, at neuropathies of toxic genesis.

Palestesiometry. Palestesiometer (Fig. 11) is applied for evaluation of vibration sensitivity.

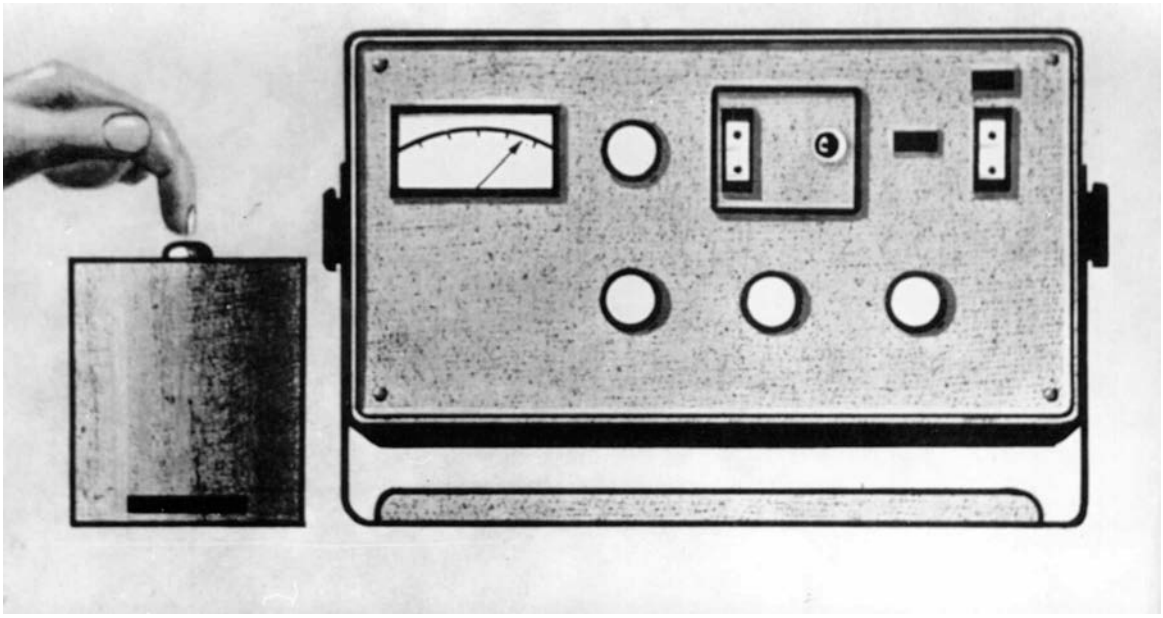


Fig 11. Measuring device of vibrational sensitivity (ИВЧ-02)

The unit consists of a measuring device and electromagnetic vibrator, which transform electrical oscillations in mechanical. It is necessary to touch skin on any site of a body with a contact of the vibrator. The parameter is tuned in a given frequency. An ill is asked to inform, in what moment he/she will begin to experience vibration. A threshold to the given frequency of vibration is determined by change of oscillation frequency. The threshold of vibration sensitivity on fingertips for able-bodied people equals 40 - 80 arbitrary units, on feet 70 - 100.

Research of vibration sensitivity for want of palestesiometer can be fulfilled with a tuning fork C-128. The tuning fork is put on bone ledges of separate sites of a body, and determines duration of vibration sensation.

Dynamometry. Muscles force of upper extremities is investigated with a spring dynamometer. Average indices for men according to a norm make 392 - 490 N (40 - 50 kgf), for the women 294 - 392 N (30 - 40 kgf) with advantage of a right hand force on some N (kgf). Decrease of force is marked at development of changes in tissues of a locomotor apparatus of upper extremities, that is characteristic for vibration disease, diseases of a locomotor apparatus of occupational genesis.

Thermography. The remote thermography is a rather informative and accessible method of testing of infrared radiation from a person body surface with the purpose of diagnostic of different diseases and pathological conditions. Warmer sites of the body will be imaged more brightly, and cold ones are registered by dark tones. A conclusion about temperature of investigated object is done depending on intensity of glow.

In clinic of occupational diseases this method is used for vibration illness, obliterating endarteritis, diseases of a locomotor apparatus (bursitis, osteoarthrosis) and so on.

The following is characteristic for a normal thermal picture of the upper extremities: uniform background, symmetrical glow, minor temperature drop in the proximal - distal direction, absolute temperature of hands is not lower 28°C, the hand is lighter in the field of the first finger, interphalanxe interspaces and along a course of large veins.

Considerable decreasing of glow intensity of distal departments of extremities down to full «thermal ablation» of one or several fingers can be observes at vibration disease; thermal asymmetry, decreasing of absolute temperature of extremities (Fig. 12) can take place.

Spirography. The method enables to determine a number of indexes, which characterizes a function of external respiration.

A number of processes, which provide gaseous exchange between gases of capillary tubes of pulmonary artery and external air, are understood as breathing clinically. Clear correlation between three components: ventilation, diffusion and perfusion is necessary for this process realization. Respiratory failure means an organism condition, at which supporting of normal gas composition of arterial blood is not provided, or last is realized by abnormal operation of an apparatus of external respiration, that conducts to decreasing of functional capabilities of the organism. Ventilation of alveoli supports an indispensable composition of alveolar gas that is the partial pressure of oxygen and carbon dioxide. Adequacy of lungs ventilation is conditioned by interaction of such factors: central regulation of breathing, function of respiratory muscular system, permeation of respiratory tracts, and capacity of a pulmonary tissue to be expanded.

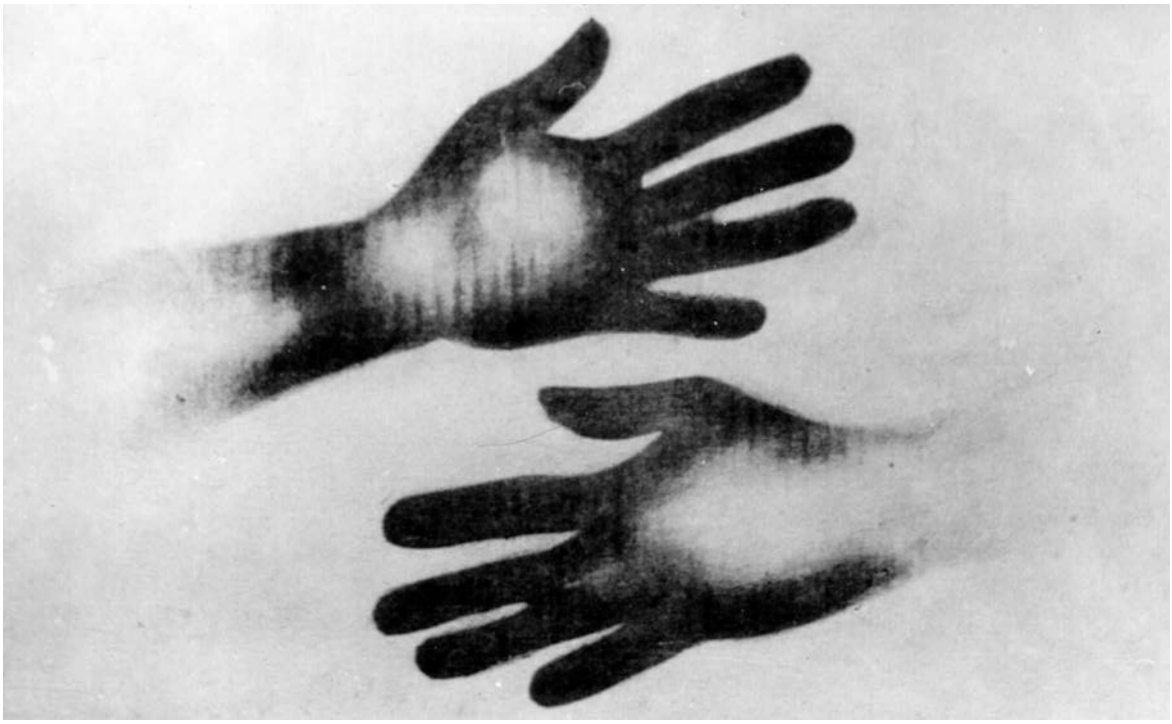


Fig. 12. Thermogram of hands with vibrational illness

In usual conditions about 50 ml O2 comes in 1 liter of blood, which flows through pulmonary capillary tubes, from alveolar air and 45 ml CO_2 comes in from the blood in alveoli.

Diffusion, gaseous exchange between alveolar air and blood depends on a difference of gas partial pressure on both surfaces of the membrane, membrane depth and diffusion surface. Diffuse capacity of lungs (DL) indicates the quantity of gas in milliliters, which passes through the pulmonary membrane per 1 minute at the difference of gas partial pressure on both surfaces of the membrane equal 1 mm mercury. DL for an able-bodied person equals to 20 ml/minute^{-1}*mm mercury. Fibrosis of lungs (berylliosis, sarcoidosis, silicosis), violation of blood circulation in the small circle (spasm of pulmonary arteries) and others conduct to decreasing of DL.

Perfusion means quantity of blood, which passes per a unit of time through capillary system of pulmonary artery (about 5 liters per one minute in rest), as a result of which venous blood transforms in arterial.

Spirogram is registered with a help of devises of closed type: СГ-1, Metatest-1, Metatest-2 and others.

Determination of lung space has important meaning for evaluation of lung volumes (Fig. 13).

The lung vital capacity (LVC) is the greatest volume of gas, which can be removed from lungs at a maximum exhalation after a maximum inhalation. LVC enables to draw a conclusion how fully lung ventilation is realized. For more precise evaluation it is necessary to compare the obtained LVC value

with necessary value, which depends on sex, height, and weight of body. Deviation of actual LVC from necessary value is allowed in limits of ± 15 %. LVC lowering is a consequence of decreasing of pulmonary tissue quantity, which functions (pneumonia, atelectasis, or edema of lungs).

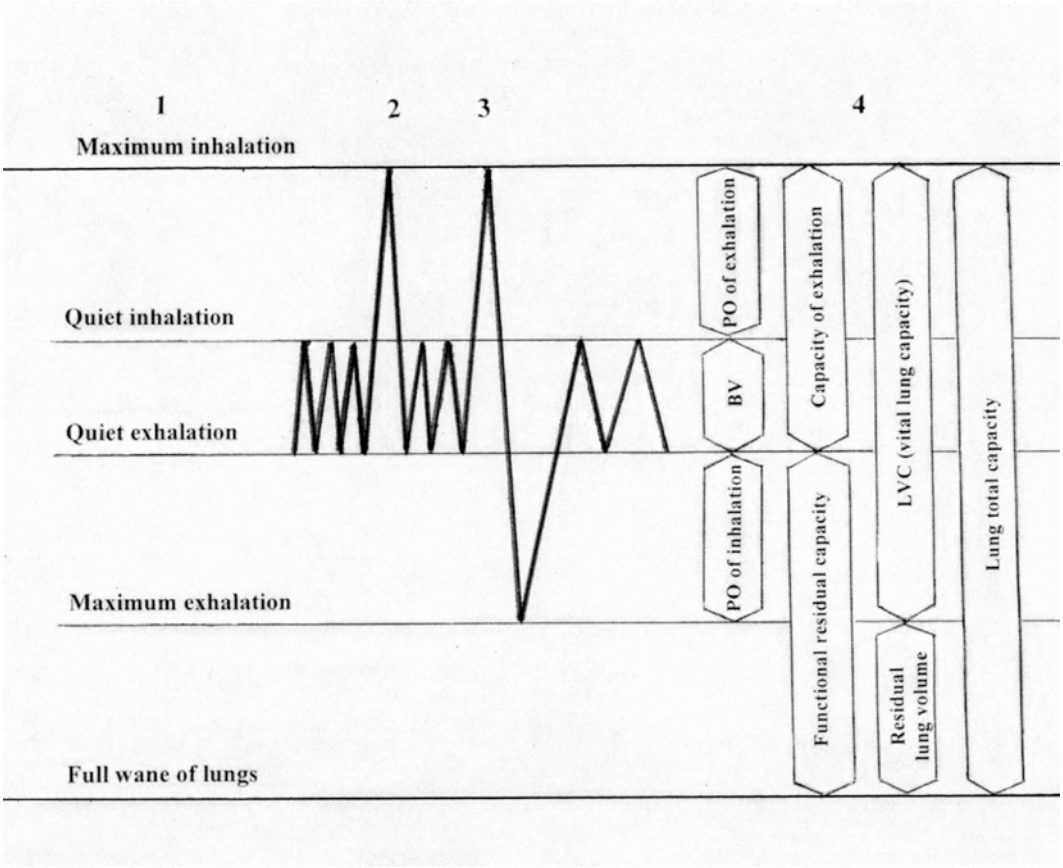


Fig.13. Volumes and capacities of lungs: 1 - respiratory excursions levels; 2 - exhalation capacity spirogram; 3 – LVC spirogram; 4 - placement of lungs volumes and capacities between levels of respiratory excursions

The respiratory volume (BV) is a quantity of air, which is inhaled at every cycle of breathing and makes 10 - 20 % from LVC.

The lung forced vital capacity (LFVC) is registered usually with help of a graphic recording.

It is determined at the forced maximum fast exhalation. Volume of the forced exhalation per 1 second (VFE1), ratio VFE1 to LVC, which is expressed in percents and is called Tifno's index, is a significant index of external respiration function.

LVC consists of PO inhalation and PO exhalation. The PO inhalation equals 1500 - 2000 ml and is lowering in process of pulmonary tissue elasticity loss. The PO exhalation equals 1000 - 1500 ml and is lowering at increase of lungs filling with blood, fibrosis changes of pulmonary tissue.

The respiration minute volume (RMV) is a quantity of air, which is ventilated in lungs during 1 minute for necessary quantity of oxygen delivery to organism and carbonic acid removal. RMV is determined by multiplication of respiration rate on the respiration volume. Increasing of RMV is observed not only at lungs diseases, but also at cardiovascular system pathology. The given parameter can vary within a wide range (3 - 10 liters). Respiration equivalent (RE) is applied for more precise evaluation of RMV that is for determination of conformities between its normal meanings and the given height, weight, sex, age. For this number 7.07 divides the standard characteristic of metabolism, which is determined under the Harris's – Benedict's table. Standard MVB (in ml) is divided by standard O2 absorption per 1 minute and then by 10. The resulted part is RE. The indicated value determines quantity of air liters, which is necessary to ventilate for 100 ml oxygen adoption. RE varies in limits from 1.8 up to 3.2.

If necessary, RMV increasing is achieved by acceleration of breathing, increasing its depth or joining both of them.

The lung maximum ventilation (LMV) is the maximum quantity of air, which can be ventilated during 1 minute (50 - 80 liters per minute). More precise evaluation can be given after determining standard lungs maximum ventilation (SLMV). For this purpose the SLVC value is multiplied by coefficient 22 (until 45 years) and 17 (after 45 years). The difference between LMV and RMV is called a respiration reserve (RR) and indicates how much the examined person can increase ventilation.

Pneumotachometry. At pneumotachometry (PTM) the condition of bronchial permeability is evaluated by airflow power of a single exhalation or inhalation. The examined person makes a deep exhalation through the mouthpiece of a pneumotachometer for exhalation power determination.

The examined person makes an abrupt deep inhalation after a maximum exhalation for inhalation power determination. As normal values of PTM at exhalation are varied within a wide range, its LVC value in liters multiplied by factor 1.2 is proposed to consider as the standard value for every examined person. The PTM value at inhalation is usually less than at exhalation.

Violation of bronchial permeability is quite often marked at pathological processes, which flow past with manifested signs of bronchial spasm. The listed tests allow to reveal the bronchial spasm in the absence of clinical manifestations and to evaluate a degree of its expressiveness.

Two groups of respiratory failure are distinguished with allowance for pathogenic mechanisms: with a primary lesion of extra-lungs and pulmonary mechanisms.

The first group is a respiratory failure with a primary lesion of extra-lungs mechanisms includes the following forms: 1) central form: violations of the central regulation (traumatic, metabolic, neuro-infectious, and other brain

lesions; 2) nervimuscular: violation of nervimuscular impulse transfer (poliomyelitis, hypokalemia); 3) parietal disease (Bekhterev's disease).

The following forms of respiratory failure are referred to the second group: 1) obstructive: violations of bronchial permeability (obstruction of respiratory tract); 2) restrictive: limitation of lungs expanding (fibrosis, atelectasis, pheumothorax); 3) diffusive (idiopathic fibrosis, and berilliosis); 4) mixed.

Three forms of respiratory failure are determined depending on gravity of condition: latent, compensated and decompensated.

Functional capabilities of the respiration system are reduced at the latent respiratory failure. The decompensation condition during physical job appears at compensated respiratory failure. The decompensated respiratory failure is characterized by an abnormal composition of arterial blood, and it is divided into three degrees depending on gravity. Impossibility to execute job, which exceeds the daily one, is marked at the I stage of gravity; restricted capacity to carry out hourly loads is marked at the II degree of gravity; manifestations of respiratory failure take place at the III degree of the failure even in a state of rest.

SOME METHODS OF LABORATORY DIAGNOSTIC OF OCCUPATIONAL POISONINGS

The detection of carboxyhemoglobin in blood is a specific method of laboratory diagnostic on intoxication by carbon monoxide, especially of acute forms. It is based on a difference of absorbency of carboxyhemoglobin and oxyhemoglobin solutions. 0.6 ml of blood at detection of carboxyhemoglobin in accordance with the method of F. A. Ivanova are solved in 10 ml of 0.4 % ammonia solutions, placed in a cuvette, and absorbency of solutions at wave-lengths of 578 and 564 microns is determined with spectrophotometry. The outcomes are calculated by the formula:

$$\text{COHв} = \frac{1{,}7 - D578 / D564}{1{,}7 - 0{,}75} \cdot 100\%,$$

where D578, D564 are absorbencies at wave-lengths of 578 and 564 microns.

Standard contents of carboxyhemoglobin varies from 0 up to 8 %.

The detection of methemoglobin MtHb in blood has large significance at poisonings by derivatives of nitro- and aminobenzol and its homologues. The method is based on a difference of light absorptions of cyanmethemoglobin and methemoglobin solutions.

The order of detection (on Eveline and Melles) is following. 0.2 ml of blood are added in 20 ml of a normal saline solution and centrifuged. The erythrocytes are hemolyzed 30 - 60 minutes with adding 4 ml of 0.1 M of phosphate buffer with pH = 6.8. The obtained hemolysate again is centrifuged, divided into two portions and pour in cuvettes. In one of the

cuvettes a drop of 5 % red salt solution is added. Then two test-tubes are taken: No. 1 with the primary hemolysate, No. 2 with the hemolysate with red blood salt, in which the haemoglobin passes in MtHb. For every cuvette a photometry is made doubly at wave-length 630 microns - before and after addition in them 5 % Cyanidums – and 4 extinctions (E) are received.

The calculation is made by the formula:

$$\text{MtHb (в \%)} = \frac{E1 - E2}{E3 - E4} \cdot 100,$$

where E1, E2 - extinction of the assay No. 1;
E3, E4 - extinction of the assay No. 2.

Blood of practically able-bodied people contains up to 3 % of methemoglobin.

Basophilic grain of erythrocytes. The detection of erythrocytes with a basophilic grain is performed as follows.

A thin blood smear is put on a microscope object-plate. It is fixed by pure alcohol and is painted over by 1 % aqueous solution of methylene-blue (exposition 1 hour). Attention is paid on erythrocytes painted in blue color with green tint, its grain can be seen in a form of blue spots-grains arranged in the middle of erythrocytes, more often on periphery. It is necessary to calculate quantity of erythrocytes with a basophilic grain on the investigated preparation. The calculation is conducted in 1 million of normal erythrocytes. Quantity of erythrocytes at able-bodied people can reach 400 - 500, and 800 - 1000 on 1 million erythrocytes (0.4 - 0.5 and 0.8 - 1.0 % accordingly) at research in a dark field. Considerable increase of quantity of erythrocytes with a basophilic grain appears at a chronic leaden intoxication.

Reticulocytes. The coloring and calculation of reticulocytes is run in such order. 1 % alcohol solution azure II stands a week then is filtrated. A smear from paint working solution is done on an object-plate, and then is dried. The thin blood smear is put over this smear, placed into a humid chamber for 5 minutes, and then is dried. Young unripe erythrocytes colored in greenish color are visible at examination of the smear. A tender net colored with dark gray or blue color can be seen in the middle of an erythrocyte. Calculation is carried out by 1000 erythrocytes and expressed in %%.

Quantity of reticulocytes makes from 4 to 12 % at able-bodied people. Quantity of reticulocytes at a radial sickness is augmented.

Toxic grain of neutrophils. The toxic grain of neutrophils is detected at poisonings with benzol and at other occupational diseases: grain, which looks like small-sized spots, occurs in the middle of the cell. The blood smear with the purpose of preparing a drug is done on a microscope object-plate, fixed by pure alcohol and filled up with makeup paint: 7 drops of paint No. 1 (1 g of fuchsine + 100 g of carbolic acid + 15 g of alcohol) are added to 2 ml of water and are carefully mixed, 5 drops of paint No. 2 (1 % methylene blue

solution) are added and again are carefully mixed. After an exposition (1 hour) the preparation is washed away by water, dried and is examined through an immersion objective.

Thrombocytes. Coloring and calculation of thrombocytes is realized in such a way.

A drop of blood, which freely flows off from a finger, is placed on an object-plate and admixed with a small drop of 14 % magnesium sulfate, then a smear from the mixture is done. It is dried up, fixed by Leischman's dye for 2 minutes, then is filled up by Gymza's paint (20 drops of the paint on 10 ml of water) during 1 hour. Blood plates on preparations are placed between erythrocytes in form of dark - violet plates, sometimes in form of packets. Calculated quantity of the plates is re-counted for a quantity of erythrocytes in 1 liter of patient's blood. Quantity of thrombocytes in 1 liter of blood for able-bodied people equals $180 \circ 10^9$ - $320 \circ 10^9$. Thrombocytopenia is developed at radial sickness, at intoxication with benzol, and at other diseases.

The Heinz's corpuscles, which are a specific symptom at intoxication by methemoglobulin-generators, represent inclusions in erythrocytes. They as a rule are found out at marked acute poisonings. Such working solution is prepared for their detection: 1 g methyl-violet on 0.6 % NaCl aqueous solution (1 g of methyl-violet + 100 g of 0.6 % NaCl solution). In a week the solution is filtrated. A drop of solution is solved with a drop of blood with the help of a covering glass and is placed in a humidity chamber on 1.5 - 2 hours. The Heinz's corpuscles in form of small round dark violet inclusions are found out at microscopy with an immersion microscope on periphery. The Heinz's corpuscles in a peripheral blood are not found at practically able-bodied people.

Detection of porphyrins in urine. Porphyronuria is one of the "cardinal" symptoms of a lead intoxication. The method of qualitative detection of porphyrins is based on its extraction with the help of ether from acid medium. Porphyrins discover capacity to be imbued in black color at the next irradiation of solution by flow of ultraviolet rays. 0.5 ml of acetic acid, 1 - 2 drops of 3 % hydrogen dioxide and 1.5 ml of ether are added in 10 ml of new-collected urine for detection of this. The test-tube is shaken. Foam, which is formed at that, is irradiated with a flow of ultraviolet rays. A bluish-green color of the upper layer indicates absence of porphyrins. Coloring of an ethereal ring in light pink or red color is evaluated as a positive reaction, it testifies increasing of porphyrins excretion. The quantitative determination of porphyrins (in form of coproporphyrins) in urine is realized with the help of spectral photometry. Quantity of porphyrin at practically able-bodied people does not exceed 120 n-gram-molecules per 1 g of creatinine in urine diurnal quantity.

Detection of lead in urine. The method is based on concentration of lead in a result of its deposition by calcium carbonate with the subsequent mineralization of a deposit and on detection of lead in the form of sulphide with the help of nephelo-calorimery.

For this purpose 500 ml of urine diurnal quantity are placed in a cylinder, added with 4 ml of $CaCl_2$ normal solution, and 0.5 н of sodium carbonate (Na2CO3) solution are added at constant concitation in some movements before appearance of light cloudiness. Examined assay is reserved for settling-out of created deposit of calcium carbonate and lead carbonate entrapped with it until the following day. Liquid above the deposit is pumped out; the deposit is centrifuged, solved in 2 - 3 ml of aquafortis, transferred in a Kjeldal's flask and evaporated on a sand bath. 1 - 2 ml of hydrogen nitrate and some drops of perhydrol are added to the deposit, and the assay is repeatedly mineralized at heating. This operation is repeated until obtaining a snow-white residue of calcium nitrate. This cooled dry residue is solved in 5 ml of hydrochloric acid diluted in the ratio 1 : 1, carried in a glass with the help of 5 ml 40 % solution of sodium citric acid, alkalinized by 30 % solution of caustic soda with the help of litmus up to the expressed alkali reaction. After that 1.25 ml of Sodium hyposulphitum solution and 0.2 ml of glycerins - sulfide reagent are introduced to the assay. The assay is carried in the colorimetric test-tube. A check solution with the same chemical reagents is prepared in another colorimetric test-tube. In 5 minutes the check assay is titrated with a working solution of lead (10 mkg/ml). Degrees of cloudiness and intensity of coloring of both test tubes are compared 3 - 5 minutes later. To calculate the obtained result of titration on concentration of lead in 1 liter of urine, it is necessary the quantity of lead working solution, which was spent for titration, in milliliters to divide on a volume of the urine examined assay, to multiply by 1000 (for reduction up to the volume equal 1 liter) and by 10 (for transfer of lead working solution quantity in milliliters into its content in micrograms) and to divide by 1000 (for transfer of micrograms in milligrams). The results of detection show the quantity of lead on 1 liter of urine (at the norm till 0.05 mg/liter, or 0.19 micro moll/liter) in milligrams.

The detection of mercury in urine is carried out in 500 ml of urine (from a diurnal volume) and consists of three stages: precipitation of mercury on a copper wire at presence of concentrated acids; sublimation of iodine, which vapors reacting with mercury create iodine mercury (amalgam); colorimetric detection of mercury.

For this purpose 25 ml of concentrated sulfuric acid and 15 ml of hydrochloric acid are added to 500 ml of diurnal volume urine, and 1.5 m of chemically pure copper wire of 0.1 - 0.2 mm diameter in form of spiral is lowered there and is set aside for a day. In a day the urine is drained, the wire is washed by water some times, and then it is squeezed between sheets of filter paper, collected in a heap and carried in the test-tube, in which some

crystals of iodine are put. The test-tube is heated on fire with continuous even turning. The wire is moved away after cooling, 4 ml of Lugol's solution and 3 ml of the mixture, which consists of 7 % copper sulfate and saturated solution of sodium sulfite, is filled in into the test-tube and is intensively shaken. The milk-white residue with pink tint, which is compared with a standard series of test tubes, where the known quantity of mercury is put, is formed.

Until 0.01 mg/liter (50 normal gram-molecule/liter) of mercury can be brought out with urine at practically able-bodied people, which have no contacts with mercury at work.

Detection of manganese in urine. The method is based on a precipitation of manganese from urine together with phosphates at abrupt alkalizing of medium, oxidation of colorless divalent manganese in the colored salt of manganese acid and in subsequent application of colorimetry for the solution.

50 ml of concentrated ammonia are added in 500 ml of urine and is set aside for a day. The urine above a residue is carefully pumped out by pomp. The residue with a remained small amount of urine is shaken, brought in a Hagedorn's test-tube and centrifuged for 15 minutes. The urine above the residue is pumped out, 8 - 10 ml of water are added to the residue, this mixture is shaken and centrifuged, then the residue is washed triply. 5 mg of hot sulfuric acid (1 : 5) are added to the washed residue and set aside for 30 minutes, then the liquid is filtered out through a Shot's filter, the residue is washed by 10 ml of sulfuric acid (1 : 5), and the filter is brought in a porcelain casserole and evaporated on an oven. The formed residue is incinerated in a muffle furnace till white color obtaining, then it is solved in 2 - 3 ml of sulfuric acid (1 : 5), poured in the measuring centrifugal tube. The residue is washed away from the cup into the test-tube with several milliliters of sulfuric acid in such a way that quantity of liquid was equal 5 ml.

A scale, which contains a series of manganese solutions, is prepared. For this purpose increasing quantities of the working manganese solution (0.1; 0.2; 0.3 ml and so on) are poured in the centrifugal test-tubes. Then sulfuric acid is added in the prepared series of solutions up to the volume of 5 ml. 3 drops of silver nitrate and 0.3 g of ammonium persulphate are added in every test-tube with standard and investigated solutions. The test-tubes are heated on a water bath till appearance of pink coloring at the standard solution. Presence of manganese is found out by appearance of pink coloring at the investigated solution. The conforming standard is selected to this color.

Quantity of manganese discharged with urine at practically able-bodied people does not exceed 0.01 mg/l.

Qualitative detection of some toxic substances in urine. It is necessary to carry out a sublimation of urine in a special installation for detection of Trichlorfon, chlorine-organic compounds, carbon disulfide and many toxic substances. Presence of toxic substances is established as a result

of a qualitative reaction, which is realized at addition of the conforming chemical agents in the sublimation.

Detection of Trichlorophon. Pink color of the solution, which indicates presence of poison, comes up in an alkaline medium after addition of the conforming chemical agents (o-tolidine, acetone) in the sublimation. The pink tint of the solution comes up the faster the more Trichlorophon is in the investigated urine.

Detection of chlorine ion. The detection of chlorine ion is performed at acute intoxications with chlorine-organic compounds. For this purpose the test-tube with sublimation is heated on a spirit lamp during several minutes at the presence of alkali. After that the free chlorine is easily found out by addition of salts, with which it creates a residue. Nature and degree of manifestation of the residue correspond to quantity of chlorine.

Detection of carbon disulfide. The detection of carbon disulfide has practical value only during the first hours after a poisoning, as it is fast blasted in an organism. For detection is used a qualitative test, which is received at addition of Fe^{37}, salts of cuprum, aniline, triethylphosphine to sublimation of urine.

Appendix 2

PERMANENT ASSETS OF ANTIDOTAL THERAPY

Aloxime is a representative of the group of cholinesterase revivifiers.

It is applied at poisoning with organic phosphorus compounds in combination with preparations of cholinolytic effect (atropinum).

It is administered intramuscularly depending on heaviness of intoxication single or some times. A single dose makes 0.075 g, and diurnal 0.2 - 0.8 g.

Amyliitritis is an antidote at poisoning with salts of prussic acid.

It is administered with inhalation through a nose with help of gauze or handkerchief, which are moistened with 2-3 drops of this drug. The daily dose makes 30 drops.

The drug should not be prescribed if there is hematencephalons, heightened intracranial pressure, and acute myocardial infarction.

Calcium tetacinum falls into complexones generative compounds. It is applied at intoxications by salts of heavy metals.

It is administered intravenously (in drops) in isotonic solution of chloride natrium or in 5 % glucose solution per 20 ml of 10 % solution 1- 2 times daily during 3 - 4 days with the following rest during 3 - 4 days. Full course of the treatment makes 1 month.

Contraindications: disease of kidneys and liver with their function lesion.

Cuprenilum falls into a group of compounds that forms complexones. It is applied at intoxication by heavy metals and their compounds. They are prescribed inside in capsules or in tablets of 0.15 and 0.25 g. On average a daily dose makes 1 g.

Contraindications: hypersensibility to Penicillin and diseases of kidneys with lesion of excretory function.

Methylene-blue is an antidote at poisoning with carbonic oxide, hydrogen sulphide, nitrates, aniline. 50 - 100 ml of 1 % aqueous solution or 1 % solutions in 25 % glucose solution are administered intravenously at poisoning with carbonic oxide and hydrogen sulphite.

0.1 - 1.15 ml of 1 % solutions on each kilogram of a body weight are administered intravenously at poisoning with aniline, nitrates.

Desferal is a preparation, which bring out iron from an organism.

It is administered intramuscularly or intravenously (dropwise) per 500 - 1000 mg every day.

It is not recommended to prescribe it for pregnant women.

By-effects at continuous application are following: dermatitis, collapse, and cataract development.

Dietixim is a cholinesterase revivifier. It is applied at poisonings by organophosphorus compounds, which are accompanied with muscarine- and nicotine-like symptomes.

It is administered intramuscularly in form of 10 % aqueous solution. It is necessary to apply dietixim together with cholinolytic preparations, vitamins (B1, B6, Acidum ascorbinicum), tranquilizers, glutamine acid, drugs, which normalize condition of central and vegetative nervous systems.

Dipyroxime is a representative of the group of cholinesterase revivifiers. It is applied at intoxication by organophosphorus pesticides.

It is administered subcutaneously together with prescription of cholinolytic preparations (atropinum, scopolaminum).

The single dose makes 1-3 ml of 15 % solution.

Glucose falls into a group of preparations, which stimulate metabolic processes. It is applied at a lot of intoxications: by cyanhydric acid, carbonic oxide, aniline, hydrogen arsenide.

It is administered intravenously slowly in form of isotonic solution (50 ml of 4.5 - 5 %) and hypertensive solution (20 ml of 10-20-40 %).

Isonitrosin is an antidote at poisonings with organophosphorus compounds.

It is administered intramuscularly 3 ml of 40 % solution, and every 30-40 min if necessary.

Natrium thiosulfuricum is an antidote at intoxications with compounds of arsenic, mercury, lead, cyanhydric acid, salts of Iodine and Brome.

5-50 ml of 30 % solution, 2-3 g inside in form of 10 % solution is prescribed intravenously.

Pentacinum falls into a group of complexones generative compounds. It is prescribed at intoxications by lead, zinc, and plutonium.

It is administered intravenously (slowly) 5 ml of 5 % solution. 30 ml of 5 % solution in 1 or 2 days at acute intoxications. The course of treatment makes 10 or 20 injections.

Contraindications: fever, lesion of kidneys function, hypertension, ischemic disease.

Succimerum is an antidote at acute and chronic intoxications caused by organic and inorganic compounds of arsenic (except for hydrogen arsenide), lead, and mercury.

Preparations of 0,5 g (1 tablet) triply per day or intramuscularly 0.3 g doubly per day from the 1st until the 4th day in 12 hours during 5 or 7 days at light intoxication by compounds of Hydrargyrum are prescribed inside. The preparation is administer intramuscularly at acute intoxication of the mean and high-gravity degree of gravity: 4 injections during the first day, 3 in the second day, 2 - 1 injection during the following five days. In total it is required up to 5.1 g of the preparation for the treatment course.

Succimerum can be administered in form of aerosol during seven days per 2 inhalations daily (the preparation is diluted in 5 % solution of hydrocarbonate, 5 mg of 10 % solution) in case of acute intoxication with vapours of metallical Hydrargyrum. Only 7 g of Succimerum is required for the treatment course.

Unithiolum plays a role of an antidote at intoxications by matters, which are capable to react with sulfhydryl groups. It is applied at acute and chronic intoxications by compounds of arsenic, Hydrargyrum, bismuth.

It is administered intravenously or subcutaneously 5-10 ml of 5 % solution 3 - 4 times a day during 6 - 7 days.

APPENDICES

Appendix 1

OCCUPATIONAL DISEASES LIST*

No.	Diseases names	Dangerous and harmful substances and production factors, which influencing can result in originating of occupational diseases	Provisional list of enterprises and works, which are performed at them
1	2	3	4
1.	Diseases, which arise under influencing of chemical factors.		
1.1	Acute, chronic intoxications and their consequences, for which flow a insulated or bound lesion of organs and systems are characteristic: - toxic lesion of respiratory organs: rhinopharyngolaryngitis, erosion, perforation of nose membrane, tracheitis, bronchitis, pneumosclerosis and other. - toxic anemia - toxic hepatitis	Chemical substances: primary raw, intermediate, waste and final products. Nitric acid, ammonia, nitrogen oxides, isocyanites, organic-silicon compounds, selenium, sulfur also its compounds, formic aldehyde, phthalic anhydride, chlorine and its compounds, benzole and its derivatives, amino-, nitro- and chlorincompounds of hydrocarbon of aromatic series, hexamethylene diamine, pesticides (organic chlorine), lead, its inorganic compounds and so on. benzol and its derivatives, amino- and nitrocompound of hydrocarbon of aromatic series, halogenous-like of	All kinds of activities, bound with processes of obtaining, processing, application (including laboratory activities) of poisoning matters in a number of industries, construction, agriculture, on transport and in non-manufacturing business

* Approved by the order of Ministry of Health of Ukraine, Ministry of Social Protection of Ukraine, and Ministry of Labor of Ukraine from 02.02.1995 No. 23/36/9.

1	2	3	4
		hydrocarbons of fatty series, hydrazin and its compounds, pesticides (chlorine-organic, phosphorus and its compounds, fluorine and its compounds	
	- toxic nephropathy	beta-naphthol, cadmium, carbon tetrachloride, and others	
	- toxic lesions of nervous system (polyneuropathy, neurosolike conditions, encephalopathy)	benzol and its derivatives, benzol homologues, amino-, nitro-, and chlorine-compounds of hydrocarbons of aromatic series, hexamethylene diamine, styrol, haloid-derivatives of hydrocarbonic fatty series, hydrazin and its compounds, manganese, pesticides, mercury and its compounds, carbon bisulfide, tetraethyl lead and others.	
	- toxic lesions of eyes (cataract)	trinitrotoluene	
	- conjunctivitis, kerato- conjunctivitis	nitric acid, ammonia, nitric oxide, isocyanates, sulfur and its compounds, formaldehyde, chlorine, fluorine and it compounds	
	- toxic lesions of bones (osteoporosis, osteosclerosis, necrosis of jaws)	yellow phosphorus and it compounds, fluorine and it compounds	
1.2	Skin diseases: epidermis, contact dermatitis, photodermatitis, онихії, параніхії, toxic melanoderma, oil folliculitis	Products of petroleum, coal and slate refining (benzine, petrol, white-spirit, lubricating fluid and oils, cresol, Lysol, tar, black oil, asphalt, pitch and its	Enterprises of chemical, petroleum refining, engineering, metallurgy, woodworking, tanning, food industries, construction, furniture production,

1	2	3	4
		distillates), chlorinated naphthalene, acids, alcalis, organic solvents, hydrosulphite, chloride of lime, salts of heavy metals, compounds of arsenic, stibium, formalin, glues and so on.	cleaning of oil-tankers, shaft construction and others.
1.3	Metallic fever, fluoroplastic (Teflon) fever	Aerosols of non-ferrous metals condensation (zinc, cupper, nickel, stibium and others), aerosols of fluoroplastic secondary polymerization	Enterprises of nonferrous metallurgy, plastics (fluoroplastic) processing of material from nonferrous metals.
1.4	Allergic diseases	See item 6	
1.5	Neoplasm	See item 7	
2.	Diseases caused by influencing of industrial aerosols		
2.1	Pneumoconiosises: silicosis, silicatosis, metalconiosises, carboconiosises, pneumocaniosises from combined dust, pneumocaniosises from dust of plastic	Long inspiration of dust that contents silicon dioxide in free or compound state, dust containing carbon (coal, cox, soot, graphite, diamond); dust of metals and their oxides including aerosol for welding, dust of organic and artificial mineral fiber, plastic, combined kinds of dust	Work in ore mines, coal mines, opened carriers, concentrating mpatients of metal and coal mining industry; mining, processing of rock products and materials, asbestos and other silicates, crushed stone and so on; production of asbestos cement and other materials containing asbestos (pipes, roofing slate, panels, planks, friction and asbestos-reinforced laminates production and others), production of white ware, glass goods, production and use of coke, soot, graphite; metallurgy and foundry works, machinery construction; metal-working; welding; grinding of loose materials; production, processing of plastic and other kinds of works with dust release
2.2	Bisynos	Long inhalation of various kinds of fibrous plant dust (flax, cotton, hemp and others)	Processing (including primary) of flax, cotton, hemp and others fibrous plant

1	2	3	4
			cultures (enterprises of primary processing, textile manufacture)
2.3	Chronic bronchitis (dust, toxic-dust in compound with undesired weather conditions)	Long inhalation of all kinds of dust said above and also organic dust of phytogenous and animal origin (flour, grain, hair, wool, tobacco, paper, sugar and others). Simultaneous action of dust and chemical factors (irritating materials, components of self-propelled mining machines and others), unfavorable microclimate conditions	Works listed in the 4 table column in item "pneumocaniosises" and enterprises as follow: bumping and leather, flour-and-cereals, sugar industry, primary processing of cotton, flax and other fiber crops, as well as other kinds of works connected with dust release.
2.4	Chronical rhinopharyngolaryngitis: - allergic diseases - neoplasmas	Long inhalation of dust indicated in items 2.1., 2.2. See item 6 See item 7	Works listed in items 2.2., 2.3.
3.	Diseases caused by effect of physical factors		
3.1	Diseases connected with effect of ionizing radiation: a) radiation sickness (acute or chronical) b) local radiation lesions (acute or chronical)	Non-permanent short-term general influence of external ionizing radiation or penetration of considerable quantity of radioactive substances or their compounds into organism Systematic effect of ionizing radiation in doses exceeding the level allowed for occupational irradiation External local effect of ionizing radiation, radioactive substances	All kinds of work with radioactive substances and sources of ionizing radiations

1	2	3	4
3.2	Diseases connected with influence of non-ionizing radiation: vegetative-vascular dystonia; asthenic, astheno-vegetative, and гіпоталамічний syndromes	Systematic effect of electromagnetic radiations of radio frequency range; of coherent and non-coherent mono- and polychromatic radiations	All kinds of work with sources of electromagnetic radiations of radio frequency range; all kinds of work with radiations of optical generators, masers
3.3	Tissue local lesions with laser emission (burns of skin, lesions of eye's cornea, retina)	Local influence of laser emissions	All kinds of work with radiations of optical generators, masers
3.4	Vibration disease	Long systematic effect of work local vibration, that is transferred on arms of workers, and vibration of working places	Work with hand appliances and working places located close to the mechanisms that generate vibration
3.5	Sensor-neural deafness	Systematic effect of production noise	All kinds of working activity connected with effect of intensive production noise in industry, construction, agriculture, and on transport
3.6	Vegetative sensor (angioneurosis) or sensor-motor polyneuropathy of hands	Contact transfer of ultrasound on hands	Work with ultrasound defectoscopes and medical apparatus that generates ultrasound
3.7	Electro-ophthalmia	Intensive ultra-violet radiation	Work in condition of intensive ultra-violet radiation (gas-arc welding and others)
3.8	Cataract	Systematic effect of radiation energy (infrared, ultraviolet, ultrahigh radiofrequency, X-ray, gamma radiation; neutron, proton radiation)	Press-forging, electric welding and thermal works, production of ????, works connected with infrared radiation in metallurgy, with effects of ionizing and non-ionizing radiations
3.9	Decompression (caisson) disease and its consequences	Increased atmospheric pressure, violations of decompression processes	Work in caissons, pressure chambers, diving and other jobs in conditions of heightened atmospheric pressure
3.10	Overheating: - acute (heat stroke, cramp condition); - chronical (vegetative vessel dysfunction	High temperature and intensive thermal radiation in working area	Work in deep mines, foundry, marten, sheet-rolling, tube-rolling workshops; repair of industrial furnaces, cleaning of fire-chambers, boilers, glass melting and other works in condition of increased

1	2	3	4
	with permanent or paroxysmal flow, with lesion of thermoregulation, electrolytic exchange and decreasing of erythrocytes thermal resistance)		temperature, including open areas in the hot season
3.11	Obliterate endarteritis, vegetative sensor polyneuralgia (angiopathy), polyradiculitis-neuropathy	Low temperature in working area	Work at fish and meet-packing factories, fishing-boats; refrigerators; geological works; timber cutting; peatery, in damp, water-logged grounds, minings; works in conditions of water-bearing excavations, underground oil production and other kinds of work in conditions of low temperature of production environment
3.12	Onichia-dystrophies, mechanical epidermoses (arising of calluses and so on)	Processing of inner side of hide, temperature and weather factors	Leather and fur production, agricultural work (in field), work on vessels and shore stations on fish processing
4.	Diseases connected with physical overloads of separate organs and systems		
4.1	Coordinate neuroses including urinary spasm	Works, which demand high coordination of motions and are carried out in a fast imposed rate	Work on key apparatus and musical instruments, stenography, hand-written, drawing, engraving, copying and other works
4.2	Diseases of peripheral nervous system and support-motion apparatus; mono- and polyneuropathy including compression and vegetative-sensor neuropathy, cervical and cross-sacral radiculopathy, chronic myofibroses (myofascites, tendovaginitis, stenos legamentosis, styloidosis, a ginger that is squeezed, and others), epicondilloses of shoulder, плечолопаткові periarthrosis, bursitis, deforming osteoarthrosis including	Works connected with local muscular overstrain, of the same kind motions that are carried out in a fast rate; pressure on nervous columns, muscles, ligaments, tendons, their traumatism, systematic holding of load on hands, its lifting and displacement by hands or with use of force; works connected with systematic tilt of body, movement in forced working pose (on knees, навпочіпки, lying, inclined ahead and so on)	Works on key computer-perforation calculators, telephone set, typewriter, hand milking; polishing, drilling, forging, painting works; work of drivers of large-tonnage and self-propelled vehicles including agricultural machines; on musical instruments, circus and other works. All kinds of works in communicating and breakage faces including water-logged grounds with unfavorable weather conditions

1	2	3	4
	spondilloarthrosis, aseptic osteonecroses		
4.3	Prolapsus of the uterus and vagina walls	Enduring (10 years and more) systematic (not less 50% changeover time) lifting and displacements of loads along with forced working pose and vibration effect (or without it) at women up to 40 years old at absence of signs of traumatism of muscles of pelvic bottom bones in labor period	Work connected with lifting and displacements of loads by hands with application of force
4.4	Marked varicose veins on legs complicated with inflammatory (trombophlebitis) or trophic lesions	Lingering stay at forced working pose standing	Works connected with enduring static load, standing, systematic carrying of considerable loads. Work in mine excavations: road heading, drilling, holding and others, especially at steeply pitching seams
4.5	Diseases caused by overstrain of vocal chords: chronic laryngitis, vasomotor monochordit, nodes of vocal chords (“nodes of singers”), contact sores of vocal chords, phone-asthenia	Systematic stress of vocal chords during a long period of tim	Work of teacher, radio and television announcer, vocal- colloquial kinds of actor works; work at telephone exchanges and so on.
4.6	Progressive short-sightedness	Increased strain of sight at distinguishing of fine things from a short distance	Cartography, assembling of ferrite parts for electronic apparatus and mechanisms, cutting and checking of quality of precious and semiprecious stones, assembling of watches, work of corrector, with scopes
4.7	Emphysema of lungs	Systematic difficulties at exhalation during work	Work connected with playing wind instruments, non-mechanized glass blowing
4.8	Neuroses	Long-continued direct service of mentally diseased	Work of medical personnel at mental houses including teachers and maintenance staff at special schools for mentally defective children
5.	Diseases caused by effect of biological		

1	2	3	4
	factors		
5.1	Infection and parasitical diseases homogeneous with the infection, with which workers contact during work: tuberculosis, brucellosis, glanders, anthrax, vernal encephalitis, parrot-fever, nodules of milkmaids, toxoplasmosis, viral hepatitis, skin mycoses including mycosises of feet at miners and workers of mines, Rosenbach erysipeloid, itch, syphilis and so on.	Contact with infectious patients, infected materials or carriers of diseases, with ill animals, products of animal or vegetative origination (skin, wool, bristle, horsehair, meat, leather and fur raw, utility refuse, grain, cotton and others); contact with gnawers, surface contaminated with respective microorganisms including in subterranean conditions	Work in infection, veterinary contra tuberculosis establishments, labor workshops for tuberculosis patients, live farmings, aid posts, meat-packing factories, confectionaries, canneries; at enterprises on processing of leather and fur raw; hunting, fishing-boats, enterprises of fish industry; various kinds of works in condition of forestlands, in mines and iron mines and so on.
5.2	Dysbacteriosis, candidamycosis of skin and mucous membranes, visceral kandidosis	Antibiotics, funguses-producers, albuminous-vitaminous concentrates (AVC), nutrient yeast, mixed fodder	Work in various branches of microbiological industry; application of substances indicated in item 3 in medicine industry and practice, pharmaceutical and other establishments
6.	Allergic dieases (conjunctivitis, rhinitis, rhinopharingitis, rhinopharyngolaryngitis, bronchial asthma, asthmatic bronchitis, exogenous alveolitis, dermatitis, eczema, toxicodermia, Quincke's edema, hives, anaphylactic shock, toxico-allergic hepatitis, central and peripheral nervous system disorders and others).	Allergens are chemical substances: synthetic polymers, tars, varnishes, paints, detergents; metals and their compouns (chrome, nickel, cobalt, manganese, beryllium, platinum, zirconium, silver, gold and others), formaldehyde, epichlorinehydrin, turpentine and ethereal oils, chloramines, phthalic malein anhydride, hexamethylenediamine, dinitrochlorinebenzol, cyanides, замаслювачи, captax, thiuram, ніазон D, chlorinated naphthalines, furans, amines, acrylonitrile, ethylenenimines, pesticides and so on; medical products (vitamins, antibiotics, novocain, neuroleptics, Brom preparation, of furacillin series,	Works connected with effect of allergens in various branches of industry (enterprises of chemistry, chemical-pharmaceutical, construction, woodworking, textile, bristle and brushing industry, metallurgy engineering industry; fur enterprises, medical and pharmaceutical establishments, laboratories of industrial enterprises, institutions and so on; варії, production of mixed fodders, albuminous vitamin concentrates and others; in agriculture (battery farms, farms, work with pesticides and other); on transport, establishments for homehold services (dry-cleaner's, hairdressing saloons, wash-houses and

1	2	3	4
		sulfanylamides preparations and others), matters of biological nature (serums, vaccines, hormone-containing and enzymatic preparations, microbe, fungi cultures, albuminous vitamin concentrates, mixed fodders, excretes and poisons for gnats, snakes, helminthes and others); dust of vegetable and animal origin (grain, floury, wooden, down, feather, hair, wool, silk, cotton, flax, tobacco), pollen and others.	others).
7.	Neoplasmas		
7.1	Skin swellings (hyperkeratoses, epitheliomes, papillomas, cancer, leucokeratoses)	Products of distillation, coal, petroleum, slates (tar, pitch), anthracene, phenanthrene, aminoanthracenic oil, derivatives of nitric compounds, tar, paraffin and others. Influence of ionizing radiation (X-ray, gamma radiation and others).	All works in various branches of industry connected with influencing of matters listed in column 3. Work with radioactive substances, other kinds of ionizing radiations.
7.2	Tumors in mouth cavity, respiration organs	Compounds of nickel, chrome, arsenic, tar; asbestos, asphalt, dust of radioactive ores and dust from carbon hydrates absorbed on it	Works connected with production and application of nickel, arsenic, and chromium compounds; geological exploration, mining and processing of radioactive ore; asbestos and materials containing asbestos; works connected with production of artificial granite and articles from it, asphalt work.
7.3	Swellings in liver	Vinyl chloride, enduring contact with radioactive substances trope to tissue of liver (polonium, thorium, plutonium)	Work with vinylchloride; work in radiochemical manufacturing and others.
7.4	Stomach cancer	Hexavalent chromium compounds	Production of chromium compounds
7.5	Leucosis	Benzol, effect of various kinds of ionizing	Work with benzol and sources of ionizing

1	2	3	4
		radiation	radiation
7.6	Urinary bladder tumors (papilloma, cancer)	Amines of benzol and naphthalene series (benzidine, dianizidine, naphtylamine and others)	Work with substances indicated in column 3 in various branches of industry
7.7	Cells tumors	Enduring contact with osteotropic radioactive substances (radium, strontium, plutonium)	Work on enterprises of radiochemistry, in radiology and radiochemistry laboratories