PNEUMOCONIOSIS CLASSIFICATION, ETIOLOGY, POSITIVE DIAGNOSIS, TREATMENT AND PROPHYLAXIS.

Definition
Pneumoconioses – occupational diseases of the lungs, caused by prolonged inhalation of dust and characterized by the development of diffuse interstitial fibrosis.
The term pneumoconiosis comes from the Greek language: "pneumon" - lung; "conios" - powder, and "oza" - suffix used with the meaning of chronic aseptic, sclerogenic inflammation.
Pathogenesis
 For the development of pneumoconiosis, 3 factors are necessary:
Exposure to a specific substance
Particles of a suitable size for retention in the lungs (1-5 mcm)
Exposure of sufficient duration (typically ~10 years)
Etiology The etiological agents are represented by dusts, inhaled by workers during their professional activity, such as:
silicon dioxide (SiO2), some natural silicates,
coal,
some metals (beryllium, aluminum, carbide mixtures of hard metals: tungsten, titanium, tantalum), etc.
Powders can be classified into:
 Inhalable powders (6-25 mcm) – enter the body, but are stopped in the upper respiratory tract.
 Respirable powders (1-5 mcm) – penetrate through the upper respiratory tract, avoiding clearance and mucus mechanisms, can be retained in the lungs.
 Particles of <1 mcm are exhaled.
The cause of the pathology - the inflammatory reaction against the foreign antigen and not the direct action of the dust. The lung injury caused by the inflammatory reaction subsequently leads to pathological regeneration - fibrosis.
The progression of the fibrotic process contributes to the perfusion disorder, lymphostasis and further expansion of the connective tissue. The association of the inflammatory and atrophic process in the bronchi invokes the development of pulmonary emphysema, pulmonary cord and respiratory failure.
Favorable factors
Facilitating factors belonging to the body and the subject's vicious habits are:
bronchopulmonary diseases (including history of tuberculosis);
smoking;
regular alcohol abuse;
age (young people are more vulnerable);
intense physical exertion (increases the respiratory rate and, consequently, the amount of inhaled dust.
Favorable factors belonging to the workplace are:
unfavorable microclimate (low temperatures); increased humidity;
air currents;
lack of means of protection;
exposed professions (machine building industry, metallurgy, mining, etc.)
The classification of pneumoconiosis consists of two groups:

The first group includes the etiology of pneumoconiosis depending on the type of dust.
The second - roentgenological and clinical characteristics of the disease
According to the etiological principle, 6 groups of pneumoconioses are distinguished:
1. Silicosis as a result of inhaling SiO_2 dust
2. Silicatosis - as a result of the action of silicates (asbestos, talc)
3. Metalloconiosis from the action of metallic dust (beryllium, iron, aluminum, etc.)
4. Carboconiosis from carbon dust (coal, graphite, ash)
5. Pneumoconiosis as a result of mixed dust (anthracosilicosis, siderosilicosis, pneumoconiosis of welders, etc.)
6. Pneumoconiosis caused by organic dust (cotton, flax, cereals)
The clinical-roentgenological group and instrumental characteristic of pneumoconioses includes:
Roentgenological interpretation of opacities.
The clinical-functional manifestations of the disease.
Evolution.
Complications.
Roentgenologically determined opacities are subdivided into three forms:
 Nodules (small regular)
 Interstitial (small irregular)
 Nodulation (large regular/irregular) that corresponds to the morphological forms of pneumoconiosis
Morphopathology:
From a pathological point of view, pneumoconioses can be divided into:
 collagen forms;
 non-collagenous forms and
 mixed forms.
Collagenous forms of pneumoconis are caused by fibrogenic dusts, for example silicosis caused by silicon dioxide.
The collagenous forms of pneumoconiosis present the following histopathological lesions:
 A permanent alteration or destruction of the structure of the alveoli; a collagenous reaction of the stroma of different degrees and forms;
 Lung injuries are permanent, irreversible.
The most important collagen pneumoconioses are:
 silicosis;
 asbestosis;
 pneumoconiosis caused by mixed dusts (silico-anthracosis).
The collagenous forms of pneumoconiosis are produced by a non-fibrogenic dust and have the following characteristics:
 pulmonary alveoli are not morphologically reached;
 Fibrosis is minimal and consists mainly of proliferation of reticulin fibers;
 the reaction to dust is potentially reversible.
The most important forms of non-collagenous pneumoconiosis are:
 "pure" anthracosis (simple, uncomplicated form), caused by coal dust without free silicon dioxide content;
 barytosis;
 tinosis;
 siderosis;
 kaolinosis.
Mixed pneumoconioses:
 appear in workers who extract, process or use several minerals or metals, which generate fibrogenic and non-fibrogenic powders.

 Thus, in a coal mine there can be simultaneous exposure to both coal particles and SiO2 particles.
 Mixed pneumoconioses bear the names of both pneumoconioses, the first name being the most serious pneumoconiosis (the collagen one): silico-anthracosis, silico-siderosis, etc.
The clinical picture of pneumoconioses has a series of common features: long chronic evolution with a tendency towards progression, with the alteration of work capacity and the development of irreversible sclerotic changes in the lungs.
Formulating the diagnosis includes:
1. the name of the disease;
2. stage;
3. expression of coded radiological changes according to the international classification;
4. evolution;
5. functional diagnosis: type and degree of ventilatory dysfunction (or absence of respiratory dysfunction);
6. complications, in order of importance;
7. associated diseases.
Example of formulating the diagnosis:
Pulmonary silicosis, stage II-III, with calcifications of the lymph nodes. Chronic, purulent, obstructive bronchitis, in exacerbation. Diffuse pneumosclerosis. Pulmonary emphysema. (Radiological: 3q, ax, em, es, cp). Slow evolution. IR gr. III. Chronically decompensated pulmonary cord. IC II
Silicosis
- one of the most frequent and serious pneumoconioses, which appeared as a consequence of prolonged inhalation of silicon dioxide.
Silicosis is a chronic disease and the severity and rate of evolution can be different and are directly dependent both on the inhaled dust and on the duration of the action of the etiological factor and the individual characteristics of the body.
Professional groups exposed to the risk of developing silicosis:
Mining industry: workers in gold mining mines
Machine construction industry: smelters (casting-former, sandblaster, polisher)
Glass industry: raw material preparation operations, sandblasting
Tile and porcelain industry
Construction of tunnels, mechanical processing of quartz rocks (sandstone, granite)
Distribution in the scope of application:
Particles of silicon dioxide in the air are formed during explosions, softening, drilling, fragmentation and crushing of rocks. A particular danger is presented by granite and sandstone dust as well as polished sand.
The role of the dust left in the lungs on the body is increased by:
Widening the level of action (concentration of dust in the atmosphere of the workplace)
High level of dust actions in the antecedents.
Pathological antecedents of the lungs (especially tuberculosis).
Assessment of the state of the environment:
The best method of evaluating the action of silicon dioxide crystals at the workplace is the selection of samples from the area where one breathes directly with the selective calculation of the size of the dust particles.
Mechanism of action:
SiO2 dust particles with a diameter between 5-15 microns, which enter the pulmonary passages, are eliminated due to the mechanical cleaning action of the microciliary apparatus, but particles of 0.5-5 microns that reach the terminal regions of the lungs and alveoli can be fixed there.
The dust particles left in the lungs are captured by macrophages and transported to the lung parenchyma. The progressive atrophy of the pulmonary epithelium rapidly reduces the natural

elimination of dust from the respiratory apparatus and thus predisposes to its retention in the alveoli. In the interstitial tissue, the primary process of sclerosis is initiated with a constant tendency towards progression.
Particles with a diameter of 1-2 microns have been proven to be the most aggressive, capable of penetrating the most distant segments of the bronchial tree, the pulmonary parenchyma being trapped there.
An important role is played by the mechanical but also the toxic-chemical injury of the lung tissue.
The particularities of the etiological factor
Among the most important inorganic crystalline forms of free silicon dioxide are quartz, tridymite and cristobalite.
Histological lesion – silicotic nodule.
These nodules occur due to the death of macrophages containing silicon particles, which release them along with intracellular enzymes causing more damage and fibrosis.
There are 3 forms of silicosis
 Acute – with exposure to quartz. Signs appear in a few weeks - months after exposure. It is associated with fever, weight loss.
 Accelerated – after 5-10 years of exposure to dust with a high silicon content
 Chronic – after 10-20 or more years of exposure to dust with <30% silicon content. The most common form.
The clinical picture of silicosis:
Patients have few complaints. The vast majority of patients have symptoms typical of any chronic lung disease: dyspnea, chronic cough, pain in the chest region.
Cough and dyspnea are often related not so much to the severity of fibrosis evolution as to the simultaneous presence of silicosis and bronchitis. Bronchitis, characterized by coughing with mucous or muco-purulent expectorations, often with dust particles (petroleum, graphite). The pain in the chest in silicosis is insignificant and depends on the changes in the pleura.
Objective data.
The general condition is satisfactory.
The ribcage is usually of the usual shape, but in emphysema the anteroposterior diameter changes
Depending on the severity of the fibrosis, supraclavicular and infraclavicular spaces may be uneven.
As the pneumofibrosis progresses, the gradual thinning of the distal phalanges of the fingers and toes can be detected in association with the change in the shape of the nails in the form of an "hourglass".
1. In the initial stages - the percussive sound of the lungs has a box tone, especially in the lateral-inferior regions.
2. In the case of more pronounced fibrosis and the formation of large fibrosis nodules, the percussion sound can be subdued, especially in the scapular and interscapular regions. Percussive data are "in mosaic", which is related to alternating regions of fibrosis with emphysema.
3. On auscultation in stages I, especially II and III of the disease, rough breathing can be detected, which above the massive areas of fibrosis may have a bronchial tone, above the emphysematous regions the breathing is diminished. Approximately 1/3 of patients hear diffuse dry rales, usually they are not permanent
Laboratory data:
Progressive forms of silicosis are characterized by an increase in the general protein in the blood, the concentration of haptoglobin, fibrinogen can be increased.
An increase in C-reactive protein can often be detected. These biochemical changes in the blood are non-specific as they can also be found in a category of other inflammatory and destructive systemic diseases.
Silicosis is usually associated with the onset of respiratory failure, the degree of development of which often does not correlate with the severity of pneumofibrosis.

Patients with silicosis can be found in stages II, III without essential changes in the respiratory function, while in other people the respiratory disorders are manifested from the early stages of the disease.
X-ray
Nodular lesions are characteristic.
At first, the opacities are small round, in the parahilar region, they can be in all lung areas. Then there is the accentuation and deformation of the pulmonary pattern, the appearance of a reticular pattern with rare micronodular elements, the thickening of the interlobular pleura; these changes are usually symmetrical.
 With the advancement of the process, the deformation of the bronchial drawing increases, multiple shadows of irregular shapes appear among which round silicotic nodules with regular contours are located ("snowy" lung image). Fibrosis progresses, signs of emphysema appear.
 According to the clinical-roenghenological particularities, 3 forms of silicosis are distinguished: nodular, interstitial and tumoral. Silicosis tends to progress even after stopping work in dusty conditions containing SiO2.
Wheezing with prolonged exhalation is a little characteristic of silicosis, it can only be heard in patients with advanced stages of the disease in the case of deformation of the airways following their compression by lymph nodes or fibrosis fields.
Uncomplicated silicosis usually proceeds without fever and without essential changes in the peripheral blood.
The evolution of silicosis can be different depending on the working conditions, the form of the fibrosis, the degree of severity of the bronchitis and complications.
The decisive factor in the evolution of silicosis is:
 aggressiveness of the dust
 the individual predisposition, which depends on the condition of the upper respiratory tract, the diseases suffered, especially the pulmonary ones, the age of the patients and the genetic characteristics.
 The disease will have a negligible evolution in people who started working at a very young age and at middle age (over 40 years old).
According to evolution, silicosis is divided into slowly progressive, rapidly progressive and late silicosis.
 In the case of slowly progressive silicosis, the transition from one stage to another (more often from I to II) sometimes takes decades, but sometimes signs of progression of the fibrosis process are not even detected.
 Rapidly progressive silicosis. Its development is related to the aggressive action of the dust as it seems with a special predisposition. The evolution of the disease is serious with pulmonary insufficiency. The life span usually does not exceed a few years.
 In the case of the non-permanent action of high concentrations of quartz in the composition of the dust, late silicosis develops. It is a special form of disease that develops after a long period of time (10-20 years and more) after the cessation of contact with dust. Internship at these patients usually do not exceed 4-5 years. The triggering moments in its development can be severe pneumonia, tuberculosis, rheumatoid arthritis.
Complications of silicosis:
Chronic obstructive bronchitis
Development of pneumonia
Spontaneous pneumothorax
Bronchial asthma
Bronchiectasis disease
Pulmonary cord
Silicotuberculosis

Silicoarthritis
The complication or more precisely the association of silicosis with lung cancer is, according to the results of several investigations, quite rare.
prognosis
 The disease usually progresses slowly.
 The immediate causes of death are often intercurrent infections of the respiratory organs and right ventricular failure. Even after the cessation of the action of the dust, the disease continues to progress, sometimes leading to respiratory failure and heart failure, the development of which occurs twice as fast as the early symptoms of silicosis.
Differential diagnosis
The diagnosis is established according to the professional route of the patient, excluding
 tuberculosis,
 lung cancer,
 sarcoidosis,
 rheumatoid arthritis and other diseases with the help of usual clinical and laboratory investigations.
Treatment
At the appearance of the first signs of silicosis or active tuberculosis, the patient must be removed from work related to the action of dust.
The treatment aims to normalize the exchange of substances, primarily the protein metabolism, with the help of a rational diet, the saturation of the body with vitamins from group C, B and PP.
Preparations with expectorant action, oxygen therapy, respiratory gymnastics are indicated; in case of difficult breathing – bronchodilator preparations (theophylline, euphylline, atropine in aerosol, ephedrine); in decompensation of the pulmonary heart – diuretics and cardiac glycosides.
In the initial stages, sanatorium treatment is indicated (Southern coast of Crimea, Kislovodsk, Kazakstan, etc.).
Expertise of work capacity and rehabilitation. Silicosis and silicotuberculosis are occupational diseases that lead to a decrease in work capacity. In silicosis, the following are contraindicated:
1-continuation of the work activity in conditions of dust and irritating substances;
2-hard work activity;
3-working in adverse weather conditions.
Criteria for assessing the degree of disability are:
1- the evolutionary stage of silicosis;
2-form;
3rd degree of IR;
4- the degree of RV insufficiency.
In the first stage of the patient's silicosis, the III degree of disability is established. The patient is transferred to another workplace, where there is no contact with occupational hazards.
In stage II with a satisfactory condition of the patient and the absence of progression of fibrosis, IR degree I-II, a light work activity is allowed under favorable conditions: lack of powders, toxic substances, favorable weather conditions.
In stage II-III with progression of fibrosis and IR gr.II-III, IC, work capacity is lost. The patient is assigned the II degree of disability.
Patients who develop decompensated HF or other serious complications are assigned the first degree of disability, caused by an occupational disease.
The possibility of complete medical rehabilitation of patients affected by silicosis is very limited.
Patients with stage II, and III - if the general condition is satisfactory, IR gr.I-II, a light work activity is allowed, without contact with dust and toxic substances.
Silicatoses are pneumoconiosis caused by the inhalation of silicic acid compounds with metal oxides (Mg, Ca, Al). They can be natural metals: asbestos, talc, kaolin, as well as synthetic

compounds (cement, etc.). Silicates are widely used in industry as construction materials, as thermal and electrical insulators, anticorrosives resistant to acids and bases.
More often fibrosis develops
interstitial.
Asbestosis is a collagen pneumoconiosis caused by asbestos fibers. The time of professional exposure until the appearance of the first radiological signs can be from 5 to 10 years. Jobs with higher risk are:
1-asbestos fiber industry;
2-in construction;
3-manufacturing protective equipment (for thermal insulation);
4-plastics industry;
5-manufacture of brake pads and sealing materials.
The fibrosis is linear and not nodular, with thickening of the interalveolar and interlobular septa. Interstitial pulmonary fibrosis becomes diffuse over time, predominantly in the lower fields. Emphysema is local, bilateral. Fibrous lesions are associated with pleural thickening. In the late stages – extensive fibrosis with destruction of the architecture.
Radiological examination. In the initial forms, the lung pattern appears accentuated by bilateral diffuse fibrosis. The pleura is thickened, the hilums are indurated and deformed. Signs of pulmonary hypertension and chronic pulmonary heart disease are associated. In the advanced stages, pronounced fibrosis occurs. Micro- and macronodular shadows are characteristic. Examination of sputum can highlight asbestos bodies.
Functional respiratory examination:
The functional changes can precede the radiological ones and are characterized by:
restrictive dysfunction (lung volumes are reduced, except for the residual volume which tends to increase);
decrease in alveolar-capillary gas diffusion capacity; · hypoxemia.
 Final exam
It can highlight asbestos bodies of yellowish color, with extremities dilated into "dumbbells".
 Pleural fluid examination (pleural effusion case)
It highlights an exudate with mixed cytology (serofibrinous pleurisy, rarely serohemorrhagic). Asbestos fibers are usually not detected in the pleural fluid.
 Pulmonary biopsy
The diagnosis can be confirmed by transbronchial lung biopsy, by puncture or thoracotomy (as a rule, it is not mandatory in daily practice).
Complications:
 chronic pulmonary heart;
 bronchiectasis;
 serofibrinous pleurisy;
 lung cancer;
 laryngeal cancer;
 malignant mesothelioma.
 The incidence of cancer up to 15-20% of cases.
Talcosis is a non-collagenous pneumoconiosis. Talc is a magnesium silicate, which is used in the textile, paper, ceramic, perfumery, etc. industries. Talcosis occurs after prolonged exposure to talcum powder (15-20 years). Morphologically, a process of diffuse sclerosis develops located in the alveolar, peribronchial and perivascular septa. The tendency towards the progression of the process is less expressed. The talcosis caused by the powder evolves more seriously.
Metalloconiosis. Metal dusts can lead to diffuse pulmonary fibrosis. Metalloconiosis with the accumulation of Fe, Pb, Ba powders in the lungs shows a more favorable evolution and the moderate development of fibrosis.

Aluminosis develops when aluminum dust is inhaled. Morphologically, interstitial, perivascular and peribronchial sclerosis develops, fibrosis of the alveolar septa. In some metalloconioses, the toxic and allergic action predominates with the development of secondary fibrosis (Be, Co) and progressively severe evolution.
Siderosis is produced by inhaling iron oxide. It is asymptomatic. Radiologically, it shows small opacities with a weakly pronounced fibrous reaction. Siderosis has a favorable prognosis and does not progress. Possible complete recovery - regression of the process with self-purification of the lungs.
Beryliosis – at work, workers may be exposed to soluble and insoluble beryllium compounds. Soluble compounds can cause acute intoxication (pneumonia, diffuse bronchiolitis, contact dermatitis, conjunctivitis, rhinitis), insoluble ones can produce chronic berylliosis (intestinal fibrosis, granulomatosis).
Carboconiosis. Coal dust deposition around the respiratory bronchioles produces coal worker's pneumoconiosis. Graphite, cox have moderate fibrogenic action. Interstitial fibrosis develops more often.
Anthracosis occurs in workers who work in the thick layers of coal. Anthracosis appears after a long professional contact (over 10-15 years). It is a non-collagenous pneumoconiosis,
clinically asymptomatic. The most exposed workers are: miners, those who work on transporting and grinding coal, in coke ovens. Long-term contact with coal dust leads to reticular changes and the formation of collagen fibers with the formation of nodules. Silicoanthracosis occurs in miners in rock mines. Evolution is a collagen pneumoconiosis. Clinically it is manifested by dyspnea, cough with mucous sputum. Dyspnea becomes progressive. Finally the pulmonary heart develops.
Pneumoconioses caused by inhalation of organic dusts.
They can be considered facultative P., given the fact that pneumofibrosis often does not develop. Bronchitis with an allergic component develops more often, being characteristic of byssinosis, which occurs by inhaling organic powders and fibers (cotton). By inhaling powders: flour, cereals, sugar cane, plastic mass, diffuse fibrotic disorders develop in the lungs with an allergic and inflammatory nature

QUESTIONS TO VERIFY KNOWLEDGE:

1. What is pneumoconiosis?
 Pneumoconioses - occupational diseases of the lungs, caused by prolonged inhalation of dust and characterized by the development of diffuse interstitial fibrosis.
2. What are the risk groups in pneumoconiosis?
 Workers in the mining industry
 Oil
 Asbestos
 Machine constructions
 Other industries.
3. What place does pneumoconiosis occupy among occupational diseases?
 According to the level of spread and the severity of the clinical manifestations, pneumoconiosis ranks first among occupational diseases, due to the intense development of the mining industry, machine construction, etc., related to the production of dust.
4. List the classification of pneumoconiosis:
 The first group includes the etiology of pneumoconioses depending on the type of dust.
 Second – roentgenological and clinical characteristics of the disease.
5. Classification of pneumoconioses according to the etiological principle (6 groups)?
 Silicosis as a result of inhalation of SiO2 dust
 Silicosis as a result of the action of silicates (asbestos, talc)
 Metalloconiosis from the action of metallic dust (beryllium, iron, aluminum, etc.)
 Carboconiosis from carbon dust (coal graphite, ash)
 Pneumoconiosis as a result of mixed dust (anthracosilicosis, siderosilicosis, pneumoconiosis of welders, etc.)
 Pneumoconiosis arising under the influence of organic dust (cotton, flax, cereals, etc.)
6. What does the clinical - roentgenological and instrumental group of pneumoconioses include?
 Roentgenological interpretation of opacities
 Clinical-functional manifestations of the disease
 Evolution
 Complications.
7. What are the 3 forms of roentgenologically determined opacities in pneumoconiosis?
 Nodal (small regular)
 Interstitial (small irregular)
 Nodulation (large regular/irregular) which corresponds to the morphological forms of pneumoconiosis.
8. What common features does the clinical picture of pneumoconiosis have:
 Long chronic evolution with a tendency towards progression, often with alteration of work capacity.
 The development of irreversible sclerotic changes in the lungs.
9. What is Silicosis?
 One of the most common and serious pneumoconioses
 Consequence of prolonged inhalation of silicon dioxide SiO2.
 Silicosis is a chronic disease
 Severity and rate of evolution can be different
 They are directly dependent on inhaled dust (dust concentration, amount of free silicon dioxide, dispersion of particles, etc.)

 They are directly dependent on the duration of the action of the etiological factor
 They are directly dependent on the individual characteristics of the body.
10. What are the most important inorganic crystalline forms of free silicon dioxide:
 Quartz
 Tridymite
 Cristobalite.
11. The particularities of the etiological factor in silicosis?
 The elimination of silicon dioxide, especially that combined with other dusts, can take place in the first days after inhalation through the bronchi and trachea.
12. The role of the dust left in the lungs on the body is increased by:
 Broadening the level of action (concentration of dusts in the workplace atmosphere)
 High level of dust action in the antecedents
 Pathological antecedents of the lungs (especially tuberculosis).
13. The method of evaluating the action of silicon dioxide crystals at the workplace is?
 The best method of evaluating the action of silicon dioxide crystals at the workplace is the selection of samples from the area where one breathes directly with the selective calculation of the size of the dust particles.
14. What does the biological estimation of pneumoconiosis mean?
 Biological methods for evaluating the impact on the body do not exist
 X-ray - to assess the action of silicon dioxide dust
 Radiological interpretation should be done in accordance with the international classification of pneumoconioses according to radiological signs (1980), published by the International Labor Organization.
 The cause of radiological opacities in the lungs is the reaction of the lung tissue and not the direct action of the dust.
 At first the opacities are small round and evenly distributed on all lung areas. Then they become irregular, merge with each other and are mainly located in the upper segments of the lungs.
15. Explain the dispersion of silicon dioxide particles in the sphere of application:
 Airborne silica particles are formed during rock blasting, softening, drilling, fragmentation and comminution.
 Granite and sandstone dust as well as polished sand are particularly dangerous.
16. List the professional groups exposed to the risk of developing silicosis:
 Mining industry: workers in gold mining mines
 Machine building industry: smelters (casting-former, sandblaster, polisher)
 Glass industry: raw material preparation operations, sandblasting
 Tile and porcelain industry
 Construction of tunnels, mechanical processing of quaternary rocks (sandstone, granite).
17. What is the mechanism of action of silicon dioxide on the human body?
 Dust particles with a diameter between 5-15 microns, which enter the pulmonary passages, are eliminated due to the mechanical cleaning action of the microciliary apparatus, but particles of 0.5-05 microns that reach the terminal regions of the lungs and alveoli can be fixed there .
 Dust particles remaining in the lungs are captured by macrophages and transported to the lung parenchyma. The progressive atrophy of the lung epithelium quickly reduces the natural elimination of dust from the respiratory apparatus and thus predisposes to its retention in the alveoli. In the interstitial tissue, the primary process of sclerosis is initiated with a constant tendency towards progression.
 The most aggressive or proven to be particles with a diameter of 1-2 microns, capable of penetrating the most distant segments of the bronchial tree, reaching the lung parenchyma and remaining there. An important role is played by the mechanical but also the toxico-chemical injury of the lung tissue, but the activity of the dust directly depends on the crystal structure and the ability

to bind to plasma proteins due to the presence of silanol groups (SiOH) on their surface. This causes the death of most phagocytes with the release of lipoproteic substances (antigens) and the production of antibodies, which through a precipitation reaction form the basis of the silicate nodule.

 The progression of the fibrotic process leads to perfusion deregulation, lymphostasis and further expansion of the connective tissue. All this plus the inflammatory and atrophic process in the bronchi invokes the development of pulmonary emphysema, pulmonary cord and respiratory failure.

 Immune reactions play a special role in the body's response to the formation of silicosis. Hypergammaglobulinemia, lymphoid infiltration around the silicous nodule, excess of lymphocytes in the alveolar lavages shows the participation of the latter. Immune complexes composed of lung antigens and gamma globulins are determined in the lungs, plasma cell values are also increased.

18. The clinical picture of silicosis represents?

 Accusations typical of any chronic lung disease: dyspnea, cough, pain in the chest region.

 Cough and dyspnea are often related not so much to the severity of fibrosis evolution as to the simultaneous presence of silicosis and bronchitis. Bronchitis, characterized by coughing with mucous or muco-purulent sputum, often in places with dust particles (oil, graphite)

 Chest pain in silicosis is insignificant and is dependent on pleural changes.

19. Describe the objective data in silicosis:

 The general condition is satisfactory

 The ribcage is usually of the usual shape, but in emphysema the anteroposterior diameter changes Depending on the severity of the fibrosis, the supraclavicular and infraclavicular spaces may be uneven.

 As the process (pneumofibrosis) progresses, some patients can detect the gradual thinning of the distal phalanges of the fingers and toes in association with the change in the shape of the nails in the form of an "hourglass".

 In the initial stages of the disease, the percussive sound of the lungs usually has a box tone, especially in the lateral-inferior regions. In the case of a more pronounced fibrosis and especially in the case of the formation of large fibrosis nodules, the percussion noise can be subdued, especially in the scapular and interscapular regions. In connection with this, striking data "in mosaic" related to the alternation of regions of fibrosis with emphysema are often mentioned.

 On auscultation in stages I, especially II and III of the disease, rough breathing can be detected, which above massive areas of fibrosis may have a bronchial tone, above emphysematous regions breathing is diminished. Approximately 1/3 of patients hear diffuse dry rales, usually they are not permanent

Wheezing with prolonged exhalation is a little characteristic of silicosis, it can only be heard in patients with advanced stages of the disease in the case of deformation of the airways due to their compression by lymph nodes or fields of fibrosis

 Uncomplicated silicosis usually proceeds without fever and without essential changes in the peripheral blood. Progressive forms of silicosis are characterized by an increase in the general protein in the blood, and in particular the macrodisperse fractions of the globulin, the concentration of haptoglobin, fibrinogen can be increased

 An increase in C-reactive protein can often be detected in the blood of these patients. These biochemical changes in the blood are non-specific as they can also be found in a category of other inflammatory and destructive systemic diseases

 Silicosis is usually associated with the onset of respiratory failure, the degree of development of which often does not correlate with the severity of pneumofibrosis. Patients with silicosis can be found in stages II, III without essential changes in the respiratory function, while in other people respiratory disorders are manifested from the early stages of the disease

20. What does the evolution of silicosis depend on?

 Working conditions
 Form of fibrosis
 Degree of severity of bronchitis
 Complications.

21. What is the decisive factor in the evolution of silicosis?
 Aggressiveness of dust (concentration and dispersion of dust particles)
 The individual predisposition that depends on the state of the upper respiratory tract, the diseases suffered, especially the pulmonary ones, the age of the patients and possibly the genetic peculiarities.

22. Are the people at risk of unfavorable evolution in silicosis?
 people who started working at a very young age and at an average age (over 40).

23. Unfavorable evolution of silicosis (more often nodular) occurs in which type of workers?
 workers in stone mines, drillers, those who participate in the manufacture of abrasive materials, etc.

24. According to evolution, silicosis is divided into:
 Slowly progressive
 Rapidly progressive
 Late onset silicosis

25. What is specific for slowly progressive silicosis?
 The transition from one stage to another (more often from I to II) sometimes takes decades, but sometimes signs of progression of the fibrosis process are not even detected

26. What is specific for rapidly progressive silicosis?
 Its development is linked both to the particularly aggressive action of the dust and, it seems, to a special predisposition
 The evolution of the disease is serious with marked pulmonary insufficiency
 The life span usually does not exceed a few years
 In the case of non-permanent action of the action of high concentrations of quartz in the composition of the dust, late silicosis develops
 This is a special form of disease that develops after a long period of time (10-20 years and more) after the cessation of contact with dust
 The work experience of these patients usually does not exceed 4-5 years. The triggering moments in its development can be serious pneumonia.

27. What is the diagnosis of silicosis?
 Radiological investigation
 In the initial stages, the emphasis and deformation of the lung pattern, the appearance of a reticular pattern with rare micronodular elements, the thickening of the interlobular pleura can be observed on the tadiological cliché; these changes are usually symmetrical, sometimes more pronounced in the right lung with a predominant localization in the middle and lower regions.
 With the advancement of the process, the deformation of the bronchial drawing increases, multiple shadows of irregular shapes appear among which round silicotic nodules with regular contours are located ("snowy" lung image).
 When the process moves to stage III, the shadows merge into tumor conglomerates with the formation of cavities in some cases, more often in association with tuberculosis; signs of emphysema are evident.

28. Are roentgenologically determined opacities subdivided into three forms?
 Nodular
 Interstitial
 Tumorous.

29. What is specific about silicosis in terms of progression?
 Tendency to progress even after cessation of work in dusty conditions, containing SiO_2.

30. What are the complications of silicosis?
 Chronic obstructive bronchitis
 Development of pneumonia
 Spontaneous pneumothorax
 Bronchial asthma
 Bronchiectasis disease
 Pulmonary cord
 Silicotuberculosis
 Silicoarthritis
31. What is the prognosis of silicosis?
 The disease progresses slowly.
 The definitive diagnosis is quite difficult to establish even in the evolutionary periods of the disease after the cessation of contact with dust.
 The immediate causes of death are often intercurrent infections of the respiratory organs and right ventricular failure.
 Even after the cessation of the action of the dust, the disease continues to progress, sometimes leading to respiratory failure and pulmonary heart failure, the development of which occurs twice as fast as the onset of the early symptoms of silicosis.
32. State the main diagnostic methods of silicosis:
 For people exposed to industrial dust, it is not difficult to establish the diagnosis of silicosis in cases of the appearance of nodular opacities on the radiological clichés, their fusion in the upper regions of the lungs.
 In special cases, the diagnosis is established according to the professional route of the patient, excluding tuberculosis, lung cancer, sarcoidosis, rheumatoid arthritis and other diseases with the help of usual clinical and laboratory investigations.
33. Treatment of silicosis includes?
 At the appearance of the first signs of silicosis or active tuberculosis, the patient must be removed from work related to the action of dust.
 At the onset of the disease in appropriate working conditions, it is not necessary to limit the sick person in work and other activities, but he must be permanently under medical supervision.
 The treatment aims to normalize the exchange of substances, primarily the protein metabolism, with the help of a rational diet, the body's saturation with vitamins from the C, B and PP groups.
 Preparations with expectorant action
 Oxygen therapy
 Respiratory gymnastics
 In case of difficult breathing – bronchodilator preparations (theophylline, eofylline, atropine in aerosol, ephedrine).
 In decompensation of the pulmonary heart – diuretics and cardiac glycosides.
 In the initial stages, sanatorium treatment is indicated (Southern coast of Crimea, Kislovodsk, Kazakstan, etc.).
34. What are the preventive measures in silicosis?
 Introducing the means for dust suppression (irrigation, wet drilling), and the remaining dust to be removed with the help of effective ventilation.
 Regular monitoring of dust concentration at the workplace and SiO_2 content.
 In the event of failure of the usual technical means intended for dust removal and in extreme situations, workers must wear masks, respirators or use other means of protection.
35. Name the stages of medical investigation in silicosis?
 Primary examination. It includes anamnesis, clinical investigations, with special attention on the respiratory system. It is necessary to carry out the radiological investigation to rule out tuberculosis.
 The frequency of periodic medical check-ups depends on atmospheric pollution. If the hygienic

norms of the atmosphere are satisfactory, the medical control must be repeated in 3 years. The purpose of the medical check-up is to prevent the occurrence of the disease during the professional activity.

36. What is contraindicated in silicosis?

 Continuation of the work activity in conditions of dust and irritating substances
 Hard work activity
 Carrying out the work in unfavorable weather conditions.

37. What are the criteria for assessing the degree of disability in silicosis?

 Evolutionary stage of silicosis
 Form
 IR degree
 The degree of RV insufficiency.

38. What are the criteria according to which activity at the workplace is allowed in silicosis?

 In the first stage of the patient's silicosis, the III degree of disability is established. The patient is transferred to another workplace, where he has no contact with professional noxes.
 In stage II with a satisfactory condition of the patient and the absence of fibrosis progression, IR degree I-II, a light work activity is allowed under favorable conditions: lack of powders, toxic substances, favorable weather conditions.
 In stage II-III with progression of fibrosis and IR gr.II-III, IC, work capacity is lost. The patient is assigned the II degree of disability. Patients who develop decompensated HF or other serious complications are assigned the first degree of disability, caused by an occupational disease. The possibility of complete medical rehabilitation of patients affected by silicosis is very limited. Patients with stage I silicosis without complications are allowed light and medium work activity without contact with dust and toxic substances.
 Patients with stage II and III - if the general condition is satisfactory, IR gr.I-II, a light work activity is allowed, without contact with dust and toxic substances.

39. What is silicosis?

 Pneumoconiosis caused by inhalation of compounds of silicic acid with metal oxides (Mg, Ca, Al).
 They can be natural metals: asbestos, talc, kaolin, as well as synthetic compounds (cement, etc.).
 Silicates are widely used in industry as construction materials, as thermal and electrical insulators, anticorrosives resistant to acids and bases.
 Interstitial fibrosis develops more often.

40. What is asbestosis?

 Collagen pneumoconiosis caused by asbestos fibers.
 The time of professional exposure until the appearance of the first radiological signs can be from 5 to 10 years.

41. Jobs with high risk of asbestosis?

 Asbestos fiber industry;
 In constructions;
 Manufacturing of protective equipment (for thermal insulation);
 Industry of plastic materials;
 Manufacture of brake pads and sealing materials.

42. Do you characterize fibrosis in asbestosis?

 The fibrosis is linear and not nodular, with thickening of the interalveolar and interlobular septa.
 Interstitial pulmonary fibrosis becomes diffuse over time, predominantly in the lower fields.
 Emphysema is local, bilateral.
 Fibrous lesions are associated with pleural thickening.
 At the onset of the disease, signs of laryngitis, sub-atrophic nasopharyngitis appear.

 A complex of symptoms characteristic of chronic bronchitis, pulmonary emphysema, pneumosclerosis appears.
43. Describe the radiological examination in asbestosis:
 In the initial forms, the lung pattern appears accentuated by bilateral diffuse fibrosis.
 The pleura is thickened, the hilums are indurated and deformed.
 Signs of pulmonary hypertension and chronic pulmonary heart disease are associated.
 Pronounced fibrosis appears in the advanced stages.
 Micro- and macronodular shadows are characteristic.
 Examination of sputum can highlight asbestos bodies.
44. What are the complications of asbestosis?
 Chronic pulmonary cord;
 Bronchiectasis;
 Serofibrinous pleurisy;
 Lung cancer;
 Cancer of the larynx;
 Malignant mesothelioma.
45. What is the incidence of cancer in asbestosis?
 up to 15-20% of cases.
46. What is talcose?
 Talcosis is a non-collagenous pneumoconiosis.
 Talc is a magnesium silicate, which is used in the textile, paper, ceramic, perfume industry, etc.
 Talcosis occurs after prolonged exposure to talcum powder (15-20 years).
 Morphologically, a process of diffuse sclerosis develops located in the alveolar, peribronchial and perivascular septa.
 The tendency towards process progression is less expressed.
 Powder-induced talcosis is more severe.
47. What is metalloconiosis?
 Metal dusts can lead to diffuse pulmonary fibrosis.
 Metalloconiosis with the accumulation of Fe, Pb, Ba powders in the lungs shows a more favorable evolution and the moderate development of fibrosis.
 Aluminosis develops when aluminum dust is inhaled.
 Morphologically, interstitial, perivascular and peribronchial sclerosis develops, fibrosis of the alveolar septa.
 In some metalloconioses, the toxic and allergic action predominates with the development of secondary fibrosis (Be, Co) and progressively severe evolution.
 Siderosis is produced by inhaling iron oxide.
 It is asymptomatic.
 Radiologically, it shows small opacities with a weakly pronounced fibrous reaction.
 Siderosis has a favorable prognosis and does not progress.
 Possible complete recovery - regression of the process with self-purification of the lungs.
48. What is carboconiosis?
 Coal dust deposition around the respiratory bronchioles produces coal worker's pneumoconiosis.
 Graphite, cox have moderate fibrogenic action.
 Interstitial fibrosis develops more often.
49. Name the characteristic of anthracosis?
 It is a non-collagen pneumoconiosis, clinically asymptomatic.
 Anthracosis occurs in workers who work in thick layers of coal without tailings (the tailings contain SiO2) coal of a higher type: anthracite, coal, graphite.
 Anthracosis appears after a long professional contact (over 10-15 years).
 The most exposed workers are: miners, those who work on transporting and grinding coal, in

coke ovens.
 Long-term contact with coal dust leads to reticular changes and the formation of collagen fibers with the formation of nodules.
 Silicoanthracosis occurs in miners in tailings mines.
 Evolution is a collagen pneumoconiosis.
 It is clinically manifested by dyspnea, cough with mucous sputum.
 Dyspnea becomes progressive.
 Finally, the pulmonary heart develops.
50. What can be pneumoconioses caused by inhaling organic dusts?
 They can be considered facultative P., given the fact that pneumofibrosis often does not develop.
 Bronchitis with an allergic component develops more often, being characteristic of byssinosis, which occurs by inhaling organic powders and fibers (cotton).
 By inhaling powders: flour, cereals, sugar cane, plastic mass, diffuse fibrotic disorders develop in the lungs with an allergic and inflammatory nature.
TESTS TO VERIFY KNOWLEDGE:
1. CS. Silicosis is a chronic lung disease, caused by long-term inhalation of:
a. Silicates
b. *Silicon dioxide (SiO2);
c. Asbestos
d. Synthetic dust
e. Organic dust.
2. C.S. The dust of: contributes to the formation of silicosis.
a. Lead
b. phosphorus
c. arsenic
d. boron
e. * free silicon oxide
3. C.S. The dust of: contributes to the formation of silicosis.
a. Lead
b. Phosphorus
c. *Asbestos
d. Arsenic
e. Free silicon oxide
4. C.S. Dust from: contributes to the formation of metalloconiosis.
a. Phosphorus
b. Asbestos
c. Arsenic
d. *Beryllium
e. Lead
5. C.S. The dust of: contributes to the formation of carboconiosis.
a. Beryllium
b. Iron
c. Lead
d. Asbestos
e. *Ash
6. C.M. Pneumoconioses resulting from long-term inhalation of mixed dust are:
a. *Anthracosilicosis
b. Silicatosis
c. Carboconiosis
d. Metalloconiosis

e. Silicosis
7. C.S. Entering the body, but are stopped in the upper respiratory tract, powder in pneumoconiosis with dimensions:
a. Inhalable powders ≥20 mcm
b. Inhalable powders 2-10 mcm
c. *Inhalable powders 6-25 mcm
d. Inhalable powders 5-30 mcm
e. Inhalable powders ≤5 mcm
8. C.S. Which powders in pneumoconiosis penetrate through the upper respiratory tract and can be retained in the lungs?
a. Powders with dimensions 6-25 mcm
b. Powders with dimensions of 5-10 mcm
c. Powders with dimensions of 16mcm
d. *Powders with dimensions 1-5mcm
e. Powders with dimensions of 15 mcm
9. C.S. Dusts that are experienced through the respiratory tract in pneumoconiosis, what sizes are they?
a. With dimensions 1-5mcm
b. With dimensions of 5mcm
c. *With dimensions <1mcm
d. With dimensions 2 mcm
e. With dimensions >1mcm
10. CS. Asbestosis is a severe collagen pneumoconiosis, the clinical picture being characterized by:
a. Fever (39-40°C)
b. *Progressive dyspnea;
c. Angina pectoris;
d. Digestive signs;
e. Increase in body mass.
11. C.S. Early clinical symptoms of silicosis are as follows except:
a. *Hemoptysis
b. Coughing
c. Dyspnea
d. Chest pains
e. Respiratory failure
12. C.S. In chronic SiO_2 poisoning, with the presence of pneumofibrosis, it is characteristic:
a. *Hourglass-shaped nails
b. Discolored nails
c. Leukonychia
d. Digital Hippocratism
e. Thickening of the distal phalanges of the fingers and toes
13. C.S. Name the most common complications of silicosis except:
a. TB
b. Chronic bronchitis
c. Pulmonary emphysema
d. Spontaneous pneumothorax
e. *Cardiomyopathies
14. C.S. Name the activity in the workplace where there may be a risk of anthracosis:
a. Coal mining – silicosis
b. Loading coal into wagons
c. Firemen shoveling coal

d. The manufacture of carbon electrodes and carbon black
e. *Footwear manufacturing
15. C.S. Informative instrumental investigations performed for the diagnosis of pneumoconiosis are the following, except:
a. * Pleural puncture
b. Tomography
c. Bronchography
d. Spirography
e. X-ray
16. C.S. Specify the pathogenesis of anthracosis, except:
a. Coal powders do not have fibrogenic properties, they act by agglomeration
b. The harmful effect is accentuated by the absorption of toxic gases at the level of coal particles
c. Reticulin proliferation
d. Collagen proliferation
e. *The anticholinergic effect
17. C.S. Name the basic charges in anthracnose except:
a. It is often asymptomatic
b. Lack of effort
c. Dry cough or expectoration
d. *Cardialgia
e. The objective examination reveals: bronchial rales, pulmonary stasis
18. CS. The differential diagnosis of silicosis is carried out, except:
a. Other pneumoconioses
b. Miliary TB
c. Sarcoidosis
d. * Broncho-pulmonary cancer
e. Lung abscess
19. CS. The radiological examination of anthracosis is characterized by, with the exception of:
a. Small round or irregular opacities (less dense and fewer than in silicosis)
b. Large blinds (placards) of type A, B or C
c. Pleural symphysis or thickening of the interlobular pleura
d. Areas of emphysema
e. *No pathological changes
20. CS. The jobs at risk of asbestosis are the following except:
a. Exploitation of asbestos, processing, sorting, obtaining mixtures, packaging, transport
b. Asbestos-cement factories: tiles, corrugated sheets, pipes, floors, asphalt-asbestos
c. Ship construction - thermal insulation, sound
d. Ferodos for brakes and clutches (mixed with phenolic resins)
e. *Oil industry
21. SC. The clinical manifestations of asbestosis are the following except:
a. Difficulty in deep inspiration
b. Difficult yawning due to pulmonary stiffness
c. Cough, often productive (sometimes with asbestosis corpuscles)
d. Hemoptypic expectoration – association with broncho-pulmonary cancer
e. *Diffuse cardiac pain
22. CS. The objective examination of asbestosis is characterized, with the exception of:
a. Digital Hippocratism
b. *Cardialgia
c. Pleural rub

d. Bronchial rales
e. Crepitant rales at the lung bases - asbestosis alveolitis
23. CS. The positive diagnosis of silicosis is established, except:
a. Professional route
b. The clinical aspect
c. Paraclinical examinations
d. Complementary laboratory examinations
e. *Lung biopsies
24. CS. The differential diagnosis of asbestosis will be carried out, except:
a. Basal localized postinfectious fibrosis
b. Tuberculosis
c. Pulmonary granulomas
d. Sarcoidosis
e. *Myocarditis
25. CS. Name the complications of asbestosis except:
a. Chronic bronchitis
b. Pulmonary emphysema
c. Respiratory failure
d. Bronchial carcinoma
e. *Duodenal ulcer
26. CS. Name the degree of dispersion of powders in the installation of pneumoconiosis:
a. 10 - 15μm;
b. 7 - 5μm;
c. 5 - 10μm;
d. *5 – 0.5μm;
e. 0.5 – 0.1μm.
27. CM. Name the pathologies included in pneumoconiosis:
a. *Metaloconiosis
b. *Carboconiosis
c. Dust bronchitis
d. *Pneumoconiosis due to inhalation of mixed dust
e. *Pneumoconiosis due to inhalation of dust containing SiO2
28. C.M. Favorable factors in pneumoconiosis that belong to the body and the vicious habits of the subject are:
a. * Bronchopulmonary diseases
b. *Smoking
c. *Regular alcohol abuse
d. *Intense physical effort
e. Obesity
29. C.M. Favorable factors in pneumoconiosis that belong to the workplace are:
a. *Unfavorable microclimate
b. *Air currents
c. *Professions exposed
d. Work schedule with exposure interruptions
e. *Lack of means of protection
30. C.M. Mark the types of pneumoconiosis (by evolution):
a. *Rapidly progressive
b. *Slowly progressive
c. *Late
d. *Regressive

e. Stationary
31. C.M. Name the syndromes that can form in pneumoconiosis:
a. Pericarditis
b. *Respiratory failure
c. *Pulmonary emphysema
d. *Pneumosclerosis
e. *Bronchitis
32. C.M Specify the main diagnostic methods of pneumoconiosis:
a. Pleural puncture
b. *Tomography
c. *Bronchography
d. *Spirography
e. *X-ray
33. C.M. Roentgenologically determined opacities are subdivided into three forms:
a. Nodal (irregular marriages)
b. *Interstitial (small irregular)
c. *Nodulation (large regular/irregular)
d. *Nodal (small regular)
e. Nodulation (small regular/irregular)
34. C.M. Collagen pneumoconioses, the most important are:
a. *Silicosis
b. *Asbestosis
c. *Silico-anthractosis
d. Metalloconiosis
e. Carboconiosis
35. C.M. The most important non-collagenous pneumoconioses are:
a. *Pure anthracosis
b. *Baritosis
c. *Tanniose
d. Asbestosis
e. *Siderosis
36. C.M. The most important mixed pneumoconioses are:
a. Baritosis
b. Stannosis
c. *Silico-anthractosis
d. Asbestosis
e. *Silico-siderosis
37. S.M. The clinical picture of pneumoconiosis is:
a. Rapid evolution
b. *Long chronic evolution
c. Without altering work capacity
d. *With alteration of working capacity
e. *Sclerotic changes
38. C.M. What characterizes silicosis:
a. *The most frequent and serious pneumoconioses
b. *Appears as a consequence of prolonged inhalation of silicon dioxide
c. Appears as a consequence of prolonged inhalation of silicates
d. *Severity and pace of development may be different
e. Lesions of the bronchial tree in the form of endobronchitis.
39. C.M. Professional groups exposed to the risk of developing silicosis are:

a. *Workers in gold extraction mines
b. *Glass industry
c. *The tile and porcelain industry
d. *Construction of tunnels
e. Metallurgical workers
40. C.M. Airborne silicon dioxide particles are formed during:
a. *Explosions
b. *Ramolirii
c. Metal melting
d. *Crushing of rocks
e. *Rock fragments
41. C.M Methods of evaluating the action of silicon dioxide crystals at the workplace are:
a. *Selection of samples from the area where one breathes directly
b. *Selective calculation of the size of dust particles.
c. Humidity measurement
d. Sample selection at any point in the room
e. Airspeed measurement
42. C.M. The role of SiO2 dust left in the lungs on the body is increased by:
a. Decrease in action level
b. *Broadening the level of action
c. *High level of dust activity in the antecedents
d. Low level of dust action in the antecedents
e. *Pathological antecedents of the lungs
43. C.M. The mechanism of action of SiO2 particles is:
a. *5-15 microns enter the urinary tract and are eliminated from the body
b. 5-15 microns enter the urinary tract and are not eliminated from the body
c. *Of 0.5-5 microns that reach the ternal regions of the lungs and alveoli can attach there
d. 0.5-5 microns are eliminated from the body
e. 6-10 microns that reach the ternal regions of the lungs and alveoli can attach there
44. C.M. The mechanism of action of SiO2 particles is:
a. *Dust particles left in the lungs are captured by macrophages
b. *They are transported in the pulmonary parenchyma
c. *In the interstitial tissue the primary process of sclerosis is initiated
d. *Progressive atrophy of the pulmonary epithelium rapidly reduces the natural elimination of dust
e. SiO2 particles are not transported in the lung parenchyma
45. C.M. The most aggressive characteristic of SiO2 particles is:
a. *Diameter of 1-2 microns
b. Diameter of 2-3 microns
c. Diameter 12 microns
d. * Penetration into the most distant segments of the bronchial tree
e. *Penetration into the pulmonary parenchyma and remaining there.
46. C.M. The most important inorganic crystalline forms of free silicon dioxide are:
a. *Quartz
b. *The Tridimite
c. *Cristobalite
d. Plaster
e. Graphite
47. C.M. The characteristic of the histological lesion in SiO2 poisoning is:
a. *Silicotic nodules are observed
b. *Nodules appear due to the death of macrophages

c. *Intracellular enzymes cause damage and fibrosis
d. In SiO2 intoxication, fibrosis is not detected
e. Silicotic nodules occur in asbestosis
48. C.M. List 3 forms of silicosis:
a. *Acute
b. Subacute
c. Serious
d. *Accelerated
e. *Chronicle
49. C.M. What is characteristic of the acute form of silicosis?
a. *Appears in a few weeks - months of exposure
b. * Fever
c. Cough with sputum
d. *Weight loss
e. Dyspnea
50. C.M. What is characteristic of the chronic form of silicosis?
a. *Appears after 10-20 or more years
b. *Exposure to dust with <30% silicon content
c. *The most common form
d. Appears in a few weeks - months of exposure
e. Exposure to dust with >30% silicon content