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Asbestos

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Asbestos

2

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Compiled by L.A. Timofiyevskaya, Cand. of Sci. (Medicine)

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ASBESTOS

The name "asbestos" pertains to a group of minerals having a fibrous structure. Two groups of asbestos are distinguished: a) chrysotile asbestos; b) amphibolic asbestos (anthophyllite, amosite, tremolite, actinolite, crocidolite, regiclite, rhoducite, magnesia arphvedsonite).

The fibres of individual types of asbestos are rather diverse in lenght that ranges from some nanometers up to 100 mcm.

Molecular formula: a) 3 $MgO{\cdot}2SiO_2{\cdot}2H_2O$ – chrisotile asbestos [1,3]

a) $H_4Mg_3Si_2O_4$ – chrisotile asbestos [2]

b) $(OH)_2Mg_7Si_{22}$ – anthophyllite [1]

$Na_2Fe_3\,MgFe_2\,(Si_4O_{11})_2\,(OH)_2$ – crocidolite

$H_4Mg_5Fe_{18}Fe_2\,Al_2Si_{25}O_{84}$ – amosite [1,3]

$Ca_2Mg_5(Si_4O_{11})(OH)_2$ – tremolite [1]

$(OH)_2Mg_4SiO_{22}$ – anthophyllite [2]

$(MgFe)_7(OH)_2(Si_4O_{11})_2$ – anthophylite [3]

The content of benzo(a)pyrene in the asbestos of the Bazhenovo mines is 9.96 +1.10 mcg/kg, being 3.1 mcg/kg, in the ore before, and 3.8 +0.47 mcg/kg after drying. Commercial asbestos contains 36.97+ +0.47 mcg of benzo(a)pyrene per kg. In the commercial asbestos from the Djetygary mines the content of benzo(a)pyrene is about twice as less [7].

PRODUCTION PROCESS

The main sources of asbestos in the USSR are the Bazhenovo, Aktoburak and Djetygary ore beds.

The system of working the asbestos ore beds is a layer-by-layer one. The bulk of the ore is extracted mechanically without preliminary concentration in the mine. The process of the asbestos ore concentration consists of repeated crushing of the ore, its drying and grading, aspiration of the opened asbestos fibres, their sizing according to the lengt and packing [5,8].

USE

The outstanding properties of asbestos, i.e. its ability to split int thin elastic and robust fibres, low heat- and electric conductivity, hig adsorption and chemical stability, make it a unique material essenti for several industries.

The principal groups of asbestos articles and materials are:

1. Asbestos textiles (incombustible fabrics and clothes, cords, etc.);
2. Asbestos-cement articles (pipers, electroinsulating boards, construction items, etc.);
3. Asbestos-bakelite articles (braking and friction pieces, as well as materials for acid-resistant apparatus);
4. Asbestos-rubber articles (breaking rings and shoes, etc.);
5. Thermoinsulating materials;
6. Asbestos-bitumen materials (bitumen-impregnated asbestos paper; hydroinsulating materials, tiles, road pavements, etc.) [1].

PATHWAYS INTO ENVIRONMENT

The principal sources of asbestos dust which it gets to the air are the processes of the ore extraction and concentration as well as further processing of asbestos [5].

The content of the asbestos dust around asbestos – producing and processing factories was found to range:

at a distance of 0.3 km . . . . . from 0.4 to 6.8 mg/m^3
at a distance of 0.5 km . . . . . from 1.0 to 5.0 mg/m^3
at a distance of 1.0 km . . . . . from 0.6 to 5.7 mg/m^3
at a distance of 1.5 to 2 km. . . . . from 0.7 to 2.7 mg/m^3
at a distance of 3.0 km . . . . . from 0.5 to 2.6 mg/m^3 [9].

The air may be also polluted because of wind erosion. For example, an analysis of the air of an inhabited locality situated in the vicinity of asbestos deposits revealed the presense of asbestos in two ais samples out of six [10]. The air pollution is also possible because of the wear of asbestos slate roofs, braking shoes, etc. [11].

CONCENTRATION

In the mines where asbestos ore is extracted the concentration of dust consisting of serpentine with a small content of asbestos and other silicates averaged at the working place of drillers and hewers 40.6 and 114.4 mg/m^3, respectively. The dust content in the air sucked away from the drilling machine shop averaged 137 mg/m^3. The air of the operator's cabin and the engine compartment of an excavator was found to contain 5 to 7 mg/m^3 of dust. In the air of the cab of a dump truck the dust content varied from 6.0 to 20.0 mg/m^3 (being 14.0 mg/m^3 on the average). High concentrations of the dust reaching 42.0 mg/m^3 were also revealed in the cabs of bulldozers levelling asbestos dust.

The greatest (from 89 to 97.3%) part of the particles of all sorts of asbestos had a size of up to 1 mcm; 0.2 to 1.0% of the particles had a size of 6 to 10, and 0 to 0.6%, of over 10 mcm.

The asbestos ores are concentrated in three main workshops: crushing and sizing, concentrating and packing. In 1947–1961 the dust content in the air of the workshops exceeded, on the average, 100 mg/m^3 (100.1+ +53.7 6 mg/m^3).

As a result of developing and implementing a number of sanitary and technical measures the dust content in the air of the workshops was sharply reduced. For example, in 1972–1973 the average dust content

in the air crushing and sizing workshops varied from 3.1 to 6.9 mg/m^3, in the concentrating workshop from 3.6 to 9.7 mg/m^3 and in the packing workshops from 2.1 to 7.4 mg/m^3 .

The percentage of particles below 1 mcm and above 10 mcm in size averaged in the crushing and sizing workshops 58.5 and 7.4%, respectively.

The incidence of asbestosis among the workers of asbestos – producing and processing plants was much lower than in 1947. For example, with workers whose work record ranged from 6 to 10 years the asbestosis incidence dropped from 100 to 1.2; with those having a 10- to 15-year work record, from 106 down to 7.3, and with persons having a work record of over 15 years down to 11.1. However, the risk of asbestosis, though sharply diminished, has not yet eliminated completely. In 1973 the number of freshly-diagnosed cases was 0.05% [5].

At factories engaged in concentrating chrysotile asbestos, where the latter's content in the dust contaminating the air averages 16%, individual cases of asbestosis can be encountered in persons exposed to dust concentrations of 5 to 10 mg/m^3 for 15 to 20 years of the work (43).

The dust is also the principal occupational health hazard in the production of anthophyllite asbestos. At all the stages of the asbestos ore concentration the air pollution with the dust was rather considerable. The dust concentrations were found to be the highest at the places of ore dumping (412 mg/m^3) and fibre packing (246 mg/m^3). In addition to the anthophyllite fibres the dust contained a great number of granular particles of serpentine and talc-carbonate minerals. The chemical composition of the dust was: bound silicon dioxide, 38.14%; iron oxides, 6.5%; magnesium oxide, 33.7%. The content of particles below 1 mcm in size ranged at various working places from 23.7 to 27.8%; that of particles of 1 to 5 mcm, from 21.4 to 47.3%; the concentration of particles 5 to 10 mcm in size varied from 12.3 to 23.7%, and particles over 10 mcm, from 12.7 to 31.6%.

The workers of such factories develop asbestosis rather quickly. For example, 2 patients (one of whom had asbestosis of the 2nd stage) had a work record of less than 5 years; 15 patients (one with the 2nd stage asbestosis) of 6 to 10 years, and 7 patients of 11 to 15 years [6].

The content of asbestos fibres in the dust polluting the air of the workshops of an asbestos – textile factory was found to be about 30%. The average dust content, as determined over 20 years, was 1.0 mg/m^3 in the sorting, 1.1 mg/m^3 in the carding, 1.6 mg/m^3 in the twisting, 1.6 mg/m^3 in the preparatory, 1.1 mg/m^3 in the weaving, and 2.3 mg/m^3 in the spinning shops. However, since 1958 and up to now only one case of asbestosis has been registered among the workers of that factory: this was a spinner who had worked for 24 years.

In 1975 88 persons with a working record of over 15 years were examined: asbestosis was revealed in none of them. At clinico-roentgenological examinations carried out in the same year asbestosis was suspected in 8 persons 4 of whom had been working in the spinning workshop for 15 to 25 years [43].

Examinations of the dust concentration in the air of another asbestos-textile factory showed that at the mixing machine this concentration averaged 28.4 mg/m^3; at the place of charging the bin of the carding

machine, 15.6 mg/m^3; at the place of the mixture transfer from the bin to the trucks, 22.4 mg/m^3; at the working place of the spinners, 7.4 to 14.9 mg/m^3 (in different sections); at the carding machine, 6.5 mg/m^3, and in the weaving shop, 1.6 to 2.1 mg/m^3.

The incidence of asbestosis among the workers of asbestos-textile factories in 1962 was about 3 times higher than that among the workers of asbestos-concentrating plants who lived in the same city [44].

The main sources of dust in the air of the preparatory workshops of factories producing asbestos-cement articles are the manual packing operations (48 to 60.3 mg/m^3) and the loading of asbestos articles on conveyors (25.8 mg/m^3). The asbestos-cement dust is also formed during mechanical processing of articles, e.g. roofing slate trimming (its content at those places ranges from 2.5 to 39.6 mg/m^3). The concentration of the dust is also high at the places of piling the slates (978 mg/m^3). Much dust is given off also in the process of grinding tube ends: its concentration in that case reaches 45.6 mg/m^3.

On examining the health of workers engaged in cutting and trimming sheet articles and grinding tube ends no cases of bronchitis were revealed even in persons working for over 15 years [17].

Of adversary factors characteristic for the production of asbozurite, vulcanite, sovelite and asbestos cardboard the most significant from the hygienic standpoint is dust of mixed composition, such as, dust of asbozurite (85% of diatomite and 15% of asbestos), vulcanite (60% of diatomite, 20% of lime and 20% of asbestos) and sovelite (85% of dolomite and 15% of asbestos) which appears mainly at the end stages of the production process. The concentration of those dusts vary within 20 to 120 mg/m^3. Most particles are below 2 mcm in size.

On examining 204 veteran workers at those factories changes in the lungs giving one grounds to suspect asbestosis were revealed in 8 of them. An analysis of the disease incidence for a period of 5 years showed that it was higher among the workers of the main production shops than among the factory personnel as a whole [45].

On a clinico-roentgenological examination of 50 workers most of whom had worked for 10 and more years as grinders no cases of asbestosis were diagnosed. On examining grinders of another factory 3 cases suspicious of asbestosis were revealed in persons working for over 11 years. Dynamic examinations of those persons showed no progress of the disease. Asbestosis of the 1st stage was diagnosed in 3 persons working for 15 to 20 years. In 6 persons who were working with press powder for a period of 7 to 21 years an intensification of the lung picture of a finely-reticular type was revealed, however, without noticeable clinical symptoms. At another factory asbestosis was suspected in 8 persons: these were mainly workers dealing with press powder. Two of them who were moulders had been exposed to phaolite for 13 to 16 years [19].

In production of asbestos-rubber articles the content of the asbestos dust at the stage of preparing the mass was found to vary from 62 to 409 mg/m^3, depending on particular operation. Clinical examinations of 103 workers engaged in mechanical processing of asbestos-rubber and friction articles and working for over 3 years revealed no cases of pneumoconiosis, though 10 of them had been working for over 11 years.

Of 39 workers of the mechanical processing shop two cases suspicious of asbestosis were revealed: one of them had a work length of 7, and the other of 10 years (19).

On examining the health state of children aged from one to 14 years and living in residential areas with an increased content of dust in the air a greater (1.7 to 4.5 times) incidence of upper respiratory catarrhs and bronchitis (2.1 to 3 times) was revealed. Fluorographic examinations of 25,769 persons showed that 488 of them (157 males and 331 females) had pleura calcification. In 275 of those persons the disease history was investigated, and detailed examinations of the lungs were carried out. The examinations revealed a relationship between the pleura calcification incidence and the presence of asbestos deposits in the region being examined [10].

MAMMALIAN TOXICITY ARRAY

In experiments on albino rats to which dust of South Rhodesian asbestos was introduced intratracheally a dose of 55 mg per animal appeared to be lethal. The animals died as a result of developing pneumonia [16]. Long-time inhalation of the asbestos dust led to development of asbestosis.

The complex of the asbestosis symptoms is not specific. The disease takes a chronic course. The patients complain of dyspnea, asthmoid dry cough with scanty sputum discharge, persistent dull pains in the chest, less frequently, of weakness and emaciation, cardiac pains, night perspiration, insomnia. On auscultation dry rales and hard respiration, especially in the lower divisions of the lungs, are heard [27].

Characteristic of the initial stage of asbestosis is that there is correlation between the clinico-functional manifestations of the disease and the roentgenological findings. The clinico-functional changes may be rather distinct, while the roentgenological findings still give no grounds for diagnosing the first stage of asbestosis.

The leading role in the diagnosis of asbestosis should be attributed to combined clinical and functional examinations aimed at revealing insufficiency of circulation and external respiration [47].

Initial signs of asbestosis are noted on the part of the upper respiratory ducts (nose, throat, larynx) in 96.5% of patients with asbestosis and 90.4% of workers having no clinical manifestations of the disease. Bronchoscopic examinations show that it is deforming chronic bronchitis which is an early sign of asbestosis not yet accompanied with roentgenological signs [48].

The roentgenological examinations are of great importance in diagnosing asbestosis. The most regular signs of the disease are delicate reticulation of the lung picture in the lower and median divisions of the lung areas, condensation and deformity of vascular and bronchial shadows caused by peribronchial and perivascular fibrosis in the same divisions, and infiltration of the interlobar and less frequently perimural pleura [47, 33, 39, 27]. The more marked the fibrosis, the less mobile the diaphragm and the greater the heart shadow with its outline being smoothed down on the left.

In accordance with the roentgenological picture asbestosis is divided

into three stages. Characteristic of the first stage are intensification of the lung picture and a moderate deformity of a linear or finely-reticular character. In most patients the lung roots are moderately infiltrated; in one – third of the patients the infiltration involves also the interlobar pleura.

In patients with the second-stage asbestosis the deformity of the bronchovascular picture is more marked. The walls of the small meshes get thicker. The large bronchovascular trunes are hardly discernible. In one – third of the cases small, not numerous nodules are seen. The lung roots are infiltrated, dilated and deformed.

In the third stage of the asbestosis numerous nodules often furzing into large spots are seen in addition to the grossly deformed lung picture having a finely-meshy honeycomb appearance. The heart outlines are indistinct [18, 40, 39, 33].

Functional examinations reveal changes of external respiration in a part of the patients: the vital and residual capacity of the lungs is sharply diminished, mainly at the expense of the additional and reserve capacity.

The clinical changes in the lungs of patients with asbestosis are characterized by chronic bronchitis (endobronchitis) and pulmonary emphysema [33].

Of a certain importance in the diagnosis of asbestosis are asbestos bodies which are light yellow or golden-brown formations 15 to 150 mcm long and 1 to 5 mcm thick, with club-shaped ends. Their number, shape and size vary depending on the asbestos nature, the duration and gravity of the disease [40].

On thorough examination of the sputum the asbestos bodies were detected only in individual patients with pronounced asbestosis: in a much greater number of cases it was asbestos fibres that were found in the sputum. The asbestos fibres were also detected in persons having no fibrotic changes in the lungs: among them there were even persons who had ceased working with asbestos long before [39].

Investigations into correlations between the roentgenological changes, clinical manifestations and the pathomorphological picture of asbestosis carried ont in patients operated for asbesto-tuberculosis showed that the first-stage asbestosis was diagnosed only in hailf of the cases. The correlations between the roentgenological and histological changes were more distinct in patients with more advanced stages of the diseases. The changes of the lung picture revealed on roentgenological examinations of asbestos-sick patients were shown to be due to involvement of the small structural elements of the lungs, and primarilly, of the small bronchi and bronchioles [52].

Characteristic of the clinical picture of the first-stage asbestosis are the following symptoms:

1. Complaints of quick fatigue, dyspnea at walk, dry cough, pains in the subscapular regions, periodic exacerbations with a subfebrilic temperature.
2. Initial cardiopulmonary insufficiency.
3. Diminution of the lung vital capacity: in long-working persons this capacity is diminished by 10%, as compared with the norm.
4. Mild congestive manifestations (a slight cyanosis, an enlargement

and tenderness of the liver, pastosity of the lower extremities).

5. Morphological changes (in a number of cases) of peripheral blood picture (eosinophillia, monocytosis) [56].

Cardiovascular changes occuring in patients with asbestosis were characterized by a considerable pulse lability (a sharp acceleration at a slight effort or change of the body position). Many patients showed ECG changes that gave evidences of diffuse changes in the myocardium [39]. Roentgenological examinations of the heart of persons having the first-stage asbestosis and working at the textile shop of an asbestos-processing factory pointed out that most of the patients showed changes on the heart electrokymograms. In many patients the right ventricle zone appeared to be expanded, the fact that could be interpreted as an evidence of right ventricle hypertrophy. Most persons examined had changes of the left ventricle outlines. The transverse dimension of the heart was changed, too [53].

Capillaroscopic examinations of the nail bed vessels revealed a slight dilatation of the venous loops in 5 patients out of 14 [39].

Clinical examinations of 115 asbestosis-sick patients with a work record of 5 to 10 and more years revealed a cardiopulmonary insufficience of 11 stage (58 persons), cardiopulmonary insufficience of 11-a stage (3 persons) and that of 11-b stage (2 persons) [55].

In the patients suffering from asbestosis a deceleration of the blood flow and a certain rise of the venous pressure were noted [56].

Blood pressure measurements revealed a drop of the arterial (mainly systolic) pressure in one-third of the patients [39, 55].

Symptoms of chronic gastritis were discovered in 15% of the asbestosis-sick patients: in half of them these symptoms were accompanied with changes of the gastric juice acidity, more often towards hypoacidity [39].

Examinations of the liver functions revealed in most patients functional disturbences in the form of deterioration of the antitoxic and prothrombine-syntetizing functions [39]. On detailed examination of the liver functions in asbestosis-sick patients having a long-time contact with asbestos dust a distortion of the blood sugar curve, an impairment of the antitoxic and a disturbance of the protein-syntetizing functions were noted [54]. On examining the carbohydrate and pigment metabolism, the antitoxic and protein – synthetizing functions disturbances of the glycogen-synthetizing and glicogen-fixing functions were discovered in 34 patients out of 52. The impairment of the liver antitoxic function was revealed in 22 patients 7 of whom had the first, 11 the second, and 4 the third stage of asbestosis. Examinations of blood bilirubin, bile pigments and bile acids did not reveal any deviations from the control. Characteristic of the clinical picture of the first-stage asbestosis was a disagreement between the great number of complaints and distinct disturbances of the liver functions on the one hand and the scanty roentgenological and morphological changes in the lungs on the other [56].

Examinations of the kidney functions showed regular presence of pathological elements (protein, erythrocytes) in the urine. These elements were found mainly in patients with moderate and pronounced asbestosis [39]. The disturbances of the kidney functions manifested themselves primarily in changes of the filtration capacity: this might be due to

impaired permeability of the capillary walls [57].

Patients suffering from asbestosis showed no substantial changes of the blood picture. Not infrequently an accelerated ESR was observed, especially in patients with pronounced asbestosis [33].

On examining female workers dealing with asbestos no noticeable influence of the dust on the female sex sphere was revealed [39].

Pathomorphological examinations of the deceased showed single-type changes in the lungs characterized by diffuse infiltration of the pulmonary tissue, especially in the lower and the middle divisions. Rather manifest were sclerotic changes between the lobules, around the vessels and the bronchi. The lymph nodes were slightly enlarged. The heart was moderately or slightly hypertrophic. Microscopic examinations of the pulmonary tissue revealed diffuse interstitial sclerosis involving the alveolar and interlobular septa, peribronchial and perivascular tissue. The sclerosis was characterized by pronounced development of collagen fibres between which histocytes and lymphocytes, as well as considerable accumulations of dust were seen. Extensive peribronchial and perivascular sclerotic changes were noted. In the thickened septa giant cells were discovered. Rather frequent in the bronchi of all diameters were desquamative and desquamative-catarrhal bronchitis and bronchiectases, as well as pronounced hyperplasia of the mucous glands in many small bronchi and bronchioles. Around the sclerotized parts microfocal emphysema could be seen.

In the lymph nodes there were much dust, hyperplasia of the reticuloendothelial cells, sclerosis of the capsule, localized or diffuse sclerosis. A moderate interstitial sclerosis in the liver and trabecular sclerosis in the spleen were observed [40].

Pathomorphological examinations of the lungs resected from 19 patients with asbestotuberculosis and from 25 patients who died of asbestosis and asbestotuberculosis have shown that the changes in the lungs develop before the roentgenological manifestations. A characteristic pathoanatomical feature of asbestosis consists in interstitial diffuse and focal pneumosclerosis with deformity of the bronchvascular bundle, development of bronchiectases and emphysema, disturbances of the blood and lymph circulation. Specific for asbestosis are asbestos bodies found regulary in the pneumosclerotic lesions, the alveoli, and the bronchi. The number of the asbestos bodies found in the lungs of workers engaged in asbestos mining is lesser than that found in the lungs of people working at asbestos-concentrating and asbestos-processing factories.

The impairment of the drainage function of the bronchi in the patients with asbestotuberculosis leads to development of specifically shaped bronchiectatic caverns, and to intense bronchogenic dissemination of the tuberculous process. In the course of the formation of exudative-pneumonic lesions in asbestotuberculosis the specific productive reaction is frequently not distinct, and symptoms of non-specific fibrosis are prevailing.

Examinations of the structure of the tuberculous lesions and caverns walls in asbestotuberculosis reveal a complicate entaglement of the morphology of both diseases. In other words, a substantially new pathological process, i.e. asbestotuberculosis, develops [58].

Numerous experimets demonstrated the influence of various natural and synthetic kinds of asbestos on the respiratory system and the other organs and systems of the organism [13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26].

SPECIAL TOXICITY STUDIES

Carcinogenicity. In the last decades a number of reports has been published the subject of which were neoplasms of the respiratory ducts and mesetheliomas of the pleura developing in persons who have had contacts with asbestos. Among the deceased who suffered at lifetime from asbestosis lung cancer was diagnosed in 8%, i.e. at a rate greater than among other population. Cancer of various localization was the cause of death in 37.8% of patients suffering from asbestosis. The period between the making of the "asbestosis" diagnosis and the patient's death varied from 15 to 30 years with an exposure to asbestos dust of 16 years for females and 20 years for males.

The mortality from cancer of the lungs, the stomach and other organs was greater (as compared with other population) in groups of persons exposed for a long time to rather high concentrations of the dust. In the groups of persons who worked in contact with the dust for a relatively short time (for less than 15–20 years) and who were not exposed to high concentrations of asbestos-containing dust this difference was not observed [27].

The mortality from cancer among the asbestos industry workers was the object of a special 20-year study which was carried out from 1948 to 1967. The study was based on the data of death certificates and the information on the age, occupation, length of work and profession of the deceased. Agreement between the autopsy protocols and the case histories was also examined.

The rates of mortality (per 100,000 of population) from cancer of various localization in persons engaged in the mining and concentration of asbestos were compared with those observed in the population of the cities where the enterprises were situated. In the 40s and the 50s workers engaged in the ore mining were exposed irregularly to serpentine dust containing a small (less than 10%) of asbestos and present in concentrations of some dozens of mg/m^3. Only some professions, e.g. drillers and howers, were exposed to dust concentrations of some hundreds of mg/m^3. In 1948 to 1957 the dust content in the air of asbestos-concentrating workshops was rather high, but after 1957 it was diminished down to 5–10 mg/m^3. However, the workers of those workshops were exposed to the dust permanently during the whole shift.

The studies showed that the mortality from cancer of all localizations in males working at asbestos mines and asbestos-concentrating factories was, respectively, 3.6 and 4.5 times higher than that in the other population. The respective figures for females were 3.7 and 19.9 times.

The workers of the asbestos industry died, on the average, 8.5 to 13.2 years (males) and 3.1 to 10.2 years (females) earlier than the people of the control group. The average length of the "asbestos" work in the persons who died of cancer of all forms was 12.8 to 17.3 years for males and 8.0 to 11.1 years for females [28].

Examinations of the causes of death carried out for 25 years showed that of 143 asbestosis-sick patients who died within that period 124 were former workers of asbestos-concentrating factories. Of these persons 31.7% (versus 19.3% in the other population) died of malignant neoplasms, including cancer of the respiratory organs in 7.4% (versus 5.8% in the other population), stomach cancer in 6.1% (versus 5.8%) and uterine cancer in 5.8% (versus 3.4% in the population) [28].

In literature there are reports on experimental induction of tumours and precancerous states in animals. For example, mongrel albino rats were given inhalations of dust prepared from the chrysolite asbestos of the Bazhenovo mines. The dust concentration was 200 mg/m^3, the inhalations were given 5 times a week for 18 months. As a result, the rats developed pre-neoplastic changes in the bronchial and alveolar epithelium [50].

In other experiments the exposure of animals to various kinds of asbestos was combined with applications of benzo(a)pyrene or inhalations of tobacco smoke.

An experiment on rats was carried out in which the animals were given inhalations of asbestos from the Djetygary mines in a concentration of 230 mg/m^3 5 times a week, for 2 hours a day, during the whole life (the first series of the experiment). In the second series a single dose of benzo(a)pyrene (1 mg intratracheally) was added to the inhalations on the third day after their start. In the third series of the experiment the asbestos inhalations were supplemented with exposure to tobacco smoke which were started in 10 days, the dose of the smoke being equivalent to 1.5 "Pamir" cigarette per month. In all series of the experiment no tumours in the lungs were discovered. However, 19.56% of the rats given only asbestos inhalations and 57.14% of the animals exposed to the asbestos – benzo(a)pyrene combination developed pre-neoplastic states [29].

Influence of asbestos on elimination of ^{3}H-labelled benzo(a)pyrene from the lungs of Syrian hamsters was also studied. It was shown that asbestos delayed the elimination of benzo(a) pyrene from the animals' lungs, i.e. promoted accumulation of the carcinogen in the organ.

In other experiments the carcinogenic effect of chrysotile asbestos, benzo(a)pyrene and their combinations was studied. Both the asbestos, the hydrocarbon and their combination were applied intratracheally. In the first series of the experiment chrysotile asbestos was given alone in a dose of 2 mg once a month for 3 months. In the second series the asbestos was combined with benzo(a)pyrene: both were given in single doses of 2 and 5 mg, respectively. In the third series benzo(a)pyrene alone was given in a dose of 5 mg. In the first series of the experiment pre-neoplastic changes were revealed in some animals which died 9 to 28 months after the beginning of the experiment. In the second series, in addition to the pre-neoplastic states, one animal developed a lung papilloma without malignization, 3 rats, lung papilloma with malignization; and 2 rats, mesethelioma of the pleura. In the animals which received either asbestos or the benzo(a)pyrene alone no tumours were found [35].

In experiments on exposing animals to chrysotile asbestos with adsorbed benzo(a)pyrene (2 mg per rat intratracheally, three times at one-month intervals) and to a combination of chrysotile asbestos (2 mg)

with benzo(a)pyrene (5 mg) it was shown that the combined application of the asbestos with benzo(a)pyrene substantially increased the incidence of the neoplasms and pre-neoplastic states in the lungs and the pleura, as compared with application of the same doses of either asbestos or benzo(a)pyrene alone [30]. Experiments on intratracheal application of British chrysotile (1 mg) in combination with benzo(a)pyrene (5 mg) also pointed to a potentiating effect of the chrysotile on the carcinogenicity of benzo(a)pyrene [31].

Carcinogenic properties of synthetic asbestos were also studied. Dust of a hydrothermal synthetic asbestos with a size of 90% of the particles of less than 5 mcm was injected to albino rats intraperitoneally in the form of a suspension in physiological solution in a dose of 20 mh per rat three times at one-month intervals. Dust of Na/Mg-hydroxyamphibole was injected in a similar way.

Histological examinations of the organs of rate which received the hydrothermal asbectos revealed pre-mesotheliomatous changes. In one rat a benign fibrotic mesothelioma was discovered. In the rats which received Na/Mg-hydroxyamphibole the percentage of the pre-mesotheliomatous states and mesotheliomas was greater [32].

Mutagenicity.. The effect of chrysotile dust was studied in an experiment on a culture of the pulmonary tissue of a 8- to 10-week-old human embryo. The dust concentration was 100 mg per 100 ml. An inhibitory effect of the asbestos dust on the growth and migration of the pulmonary tissue cells was demonstrated [22].

Neurotoxicity/Behaviour. Most patients with initial asbestosis show vegetative dysfunctions. As the disease progresses, the vegetative disturbances give way to symptoms of asthenia which step to the foreground [33, 39].. Examinations of morbidity in workers of an asbestos-cement factory showed that in 1968–1970 diseases of the nervous system were the second among all cases of temporary loss of working capacity: in this respect they yielded only to respiratory diseases [34].

Potentiation. When combined with benzo(a)perene various types of asbestos were shown to potentiate the carcinogenic effect of the hydrocarbon [35, 30, 31]. It was also demonstrated that asbestos may play the role of a co-carcinogen which delays the elimination of benzo(a) pyrene from the lungs [36].

At all enterprises of the asbestos industry the dust polluting the air contains, in addition to asbestos, particles of the carrier rock (serpentine), talc, calcium carbonate and magnesite. The presence of these admixtures exerts a certain influence on the aggressiveness of the asbestos dust [22].

Sensitization. It has been shown that the phagocytic activity of the neutrophils in asbestosis-sick patients does not differ from that of healthy subjects, however, the mean level of heterophilic antibodies is lower in the patients that in the healthy people, and corresponds to the lowermost limit of the norm. The complement titre in most (60%) patients with asbestosis was much lower than in the healthy people. Additional examinations of persone who had no clinico-roentgenological signs of the dissease (67 workers) showed that the complement titre began to drop before the asbestosis symptoms appeared. In the patients with asbestosis the serum level of gammaglobulins was found to be elevat-

ed [37].

Examinations of workers of an asbestos-textile factory who were exposed for 7 to 8 years to asbestos dust concetrations of below the maximal permissible ones showed that in 83.8% of them the complement titre was within the normal limits (0.06), and so was the titre of heterophilic antibodies (1:15). Only in 12.5% of the workers examined the complement titre was below 0.0 8 [27].

In 34% asbestosis-sick patients the blood serum exhibited an anticomplement activity. In 31 asbestosis-sick patients the level of albumins was found to drop from 60.7 down to 50.1, while the level of gammaglobulins rose from 17.1 up to 20.3%. The data presented give one grounds to believe that autoimmune reactions play a certain role in the development of asbestosis [27].

In cases of asbestosis complication with tuberculosis the levels of total proteinl albumins, total globulins and gamma-globulin showed no distinct deviations from the above figures [38].

In experiments with sensitization of guinea pigs with an antigen prepared from the lungs of rats which were given asbestos dust inhalations it was shown that the antigenic reconstitution of the pulmonary tissue in various pneumoconioses was not similar: in addition to the common "pneumoconiotic" antigens those specific for asbestosis were also formed in it [49].

In mongrel albino rats which received inhalations of chrysotile asbestos dust in a concentration of 192.7 + 2.2. mg/m^3 for 5 days, each inhalation lasting 5.5 hours a day, no changes of the phagocytic activity of the blood leukocytes were noted [12].

Changes of the immunobiological reactivity caused by asbestos-cement dust were studied on rabbits and guinea pigs. The animals were exposed daily to a mean concetration of dust of 110 mg/m^3 for 300 days. The examinations of the body reactivity started from the 140th day of the exposure revealed no changed. However, in analogous experiments with chrysotile asbestos distinct changes were discovered [17].

In an experiment on rabbits to which asbestos-textolite dust was introduced intratracheally in an amount of 200 mg per kg body weight a drop of the titres of hemolysins and post-immunization antibodies was observed. After the next immunization given in a year the production of the antibodies returned to the initial level [19].

The state of the immunobiological re-activity was estimated on the basis of data of phagocytosis, artificial and natural immunity examitations. The phagocytosis was determined after intraperitoneal injection of 20 mg of chrysotile asbestos dust to mice. The asbestos particles were poorly absorbed by the macrophages. The dust was found to lower the production of hemolysins [22].

Primary irritation. On direct contact with asbestosis its particles may invade the epidermis; this leads to development of hyperkeratosis and cell proliferation with the formation of giant cells [4].

In industry workers handling asbestos manually the so-called "asbestos warts" are observed. This pathology is caused by needlelike asbestos fibres that invade the epithelial integuments and induce a proliferation reaction with subsequent keratinization. In the underlying tissue a chronic inflammatory process develops. The asbestos warts appear most often

on the fingers and toes, hands and soles. In some subjects they can be also found on the skin of the shanks [39, 40].

In many workers of the asbestos industry invasion of the eye cornea with numerous asbestos fibres was revealed. This did not noticeably affect the vision function [27]. However, some authors [33] point to a lowering of the cornea sensitivity. The eyes were affected most frequently in machine operators and crane drives [34].

In experiments on application of dusts of synthetic asbestos and natural chrysotile asbestos on a depilated area of the skin of the back no irritating action of the material on the skin was revealed within 15 days. The tissue reaction to subcutaneous injection (150 mg in 1 ml of physialogical solution) of various kinds of synthetic asbestos was different. Sodium-magnesium hydroxyaphibole and chrysotile asbestos caused a more active phagocytosis than other kinds of asbestos, and were eliminated from the injection site within a shorter period of time. Asbestos fibres may penetrate into sweat glands and be eliminated from the latter via their excretory ducts. Dusts of those kinds of asbestos caused sweat gland hypersecretion [21].

In 441 asbestos industry workers, aged from 20 to 40 years and having contacts with asbestos concentrations of over 2 mg/m^3, the state of the respiratory duct mucosa was examined. Pathological changes were revealed in 96.5% of asbestosis-sick workers, in 90.4% of workers showing no clinical signs of asbestosis, and in 56.4% of control subjects. In 79.7% of the persons examined the changes (of varying character) involved the trachea and the bronchi. In workers contacting the dust for less than 5 years the changes in the upper respiratory ducts were revealed in 79.8%, and in workers with an 11- to 15-year-long contact in 97.8% of the cases. The leading place among those changes belonged to affections of the nasal, pharyngeal and laryngeal mucosa: the most frequent was a catarrhal process which soon changed to a hypertrophic and then to an atrophic one. In the persone suffering from asbestosis these affections were observed 1.5 times more frequently than in the workers having no clinical signs of asbestosis. The affections were still graver in patients suffering from asbestotuberculosis.

A direct relationship between the incidence and the from of endobronchitis and the length of work in asbestos industry was discovered.

In experiments on frogs the effects of dust of various asbestos types (synthetic asbestos, natural chrysotile asbestos, serpentine and asbopheneoplasts) were compared. All the dusts were similar as regards the particle size (95% of the particles had a size of less than 5 mcm). It shown that synthetic asbestos caused no changes of the motor activity of the ciliated apithelium; chrysotile asbestos depressed the activity which, however, soon restored and was depressed again on the next exposure; asbophenoplast dusts caused a stable intensification of the motor activity [41].

SAMPLING / PREPARATION / ANALYSIS

It is believed that the most adequate for controlling the asbestos dust content in the air is the weighing method. It is recommended to take the air samples with the aid of membrane filters which can be (after determining the weight increment) clarified in acetone vapours and

then examined under a microscope for determining the shape and number of the particles [11, 36].

SPILLS

The wastes of asbestos industry are asbestos dust at asbestos mines, asbestos fibres and particles of the carrier rock at asbestos-concentrating factories, dust of articles containing asbestos [5, 6, 13, 19, 20, 21, 22].

TREATMENT OF POISONING

At early stages of asbestosis irradiation of the chest with suberythemal doses of UV-light (18 to 20 procedures, as a total) is indicated, especially during cold months.

If asbestosis is complicated with interstitial pneumonia inhalation therapy with antibiotic aerosole is indicated. In cases of complications with lung tuberculosis long-time antituberculous thearapy is necessary. For treating atypic pneumonia which hardly responds to antibiotic therapy use is made of corticosteroid hormones. A good effect can be attained by giving a course of inhalation thearapy with hydroaerosols of 1- to 3-per cent solutions of potassium chloride, sodium chloride or alkaline-salt mineral waters in combination with bronchodilating agents [27].

REMOVAL

Asbestos wastes have to be disposed of after cleaning the working places, emptying dust collectors, bins, etc. These wastes should be abundantly wetted with water and after that evacuated in tightly-closed labelled containers or strong plastis bags. The dusting wastes, depending on their character, should be transported without trans-shipping either to enterprises utilizing them or to burial places indicated by local sanitary officials.

Dumps of waste rock should be fixed by culturing green plantations on them.

RECOMMENDATIONS / LEGAL MECHANISMS

The principal way of preventing asbestosis and its complications is implementation of adequate technical and sanitary-technical measures of dust control. The maximal concentrations of asbestos dusts in the working zone air permitted in the USSR are:

Natural and artificial asbestos, as well as dusts of rocks containing over 10% of asbestos	.2 mg/m^3
Asbestos-cement	.6 mg/m^3
Asbestos-bakelite (voloknite) and asbestos-rubber	.8 mg/m^3