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Occupational Diseases Proposed for Revising the List Appended to Convention No. 121

Part II

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Byssinosis

Occupational disease caused by exposure to vegetable dusts

9. Uses of fibres and processes producing dusts causing byssinosis

(a) Cotton

Cotton accounting for 50 per cent of the world textile market is the single most important textile fibre in the world. It is spun into yarn which is used for making fabrics of all kinds.

(i) Cotton ginning. After harvesting, the raw cotton is deseeded in ginneries which are located in the areas where it is grown. The work is seasonal and may be continuous (7 days a week for as long as 9 months). Ginning is being increasingly mechanised with enclosure of machines which reduce dust exposure. The deseeded cotton is compressed into bales, which are delivered to mills, in the home country and abroad, to countries where it is processed.

(ii) Yarn processing. The compressed cotton from the bales is first loosened and mixed in opening machines. It is then beaten and blown to remove trash and short fibres. Usually, dust emissions are limited by enclosing the machines. When the mixing is done manually, the workers are exposed to extremely high dust concentrations. After the blowing or scutching processes, the fibres are formed into large rolls or "laps". These are fed onto the carding machines in which the cotton is drawn along the circumference of a revolving drum, between opposing sets of fine wire teeth which move at slightly different speeds. Fine trash is removed, as well as fibres too short for spinning. The output of the card is a thin fleece of cotton, which is gathered into a thick, soft, untwisted rope called the "sliver". The carding process straightens the cotton fibres and aligns them lengthwise.

(iii) Weaving. Most early studies concluded that byssinosis was not a significant risk among weavers. In most weaving the yarn is sized (i.e. coated with starch) which encloses the fibres and adhering trash. Only a few studies have been made of the extent of dust exposures during weaving and related processes. However, byssinosis does occur among weavers, and its prevalence may be high when unsized yarns are being woven to make heavy industrial cloths. Weavers may also have other types of respiratory disease, such as weavers' cough said to be due to micro-organisms growing in the size or an asthma from size materials such as tamarind seed.

(b) Flax

The flax plant is an annual, growing in temperate and subtropical climates, in Europe and Egypt and reaching a height of 1 to 1.5 m. Flax was apparently the first plant fibre used to make textiles.

The plants are first deseeded, the seeds being used to make linseed oil. They are then "retted" to separate the fibre bundles in the inner bark of the stem from the wooden core of the plant. In the production of flax fibres, the release of agents which cause disease depends on the nature of the retting process, which has to decompose the binding materials (pectins). This may be done by retting in water in tanks, rivers or ponds, or by dew-retting with the plants spread on the ground in the fields. In recent years, chemical treatment with alkali has been used to speed up the decomposition process and make production independent of the weather. It is also possible to separate the fibre bundles from the plant stem by mechanical means, using unretted ("green") flax, and to apply the chemical treatment during a subsequent process.

Byssinosis has been found only in workers exposed to the dust of biologically retted flax. Thus, the occurrence of byssinosis depends largely on the amounts of biologically retted flax processed. The causal agent(s) of byssinosis appear to be absent from dust in factories where only chemically treated flax is spun into yarn. After retting or chemical treatment, various mechanical processes are used to separate the fibres from the woody core of the plant, to remove impurities, and to separate short and long fibres. Depending upon the state of development of the industry in different countries, manual or mechanical methods may be used for each of these different stages: breaking, scutching and hackling. Even with the primitive methods of preparing flax fibres in the home, there are high dust exposures with byssinosis prevalent in both the old and young members of the family. The subsequent processes of carding and spinning are largely similar to those used in the cotton textile industry.

(c) Soft Hemp

Many varieties of vegetable fibre used for the manufacture of rope and twine are called "hemp" in the trade. Soft hemp (cannabis sativa) appears to be the only variety of hemp that causes byssinosis. Its cultivation is favoured by a warm and humid climate and it flourishes in Africa, the Mediterranean region and the Americas. The other varieties called "hard hams" will be considered later in the section dealing with non-specific irritant vegetable dusts. Cannabis sativa should not be confused with the related species cannabis indica, which is mainly grown for the production of alkaloids (hashish, marijuana). Cannabis sativa is an annual plant which reaches a height of 2-3 m. For its cultivation, it requires fertile and well-drained soil. At harvesting time, the stems are cut, spread out to dry and then retted like flax in pits of water to destroy the gums which bind the fibres together. The stems are then crushed to separate out the non-fibrous material by hand or machine. The later processing of soft hemp fibres is similar to that of flax; byssinosis has been found to occur at all stages but particularly during the early processes of preparation.

Like flax, soft hemp may be retted biologically or chemically treated. In the trade, it is said that dust of chemically treated soft hemp does not affect the workers as dust from biologically retted soft hemp does, but this apparently has not been scientifically investigated.

(d) Sisal

Sisal is a hard fibre produced from leaves. It is widely cultivated, for example in Central America, the West Indies and Africa. The plants flourish for 6-7 years and the leaves which are 1-2 m in length are harvested when the plant is about 5 years old. Sisal was not regarded as a source of byssinosis until a recent study in Tanzania revealed that workers preparing sisal fibres prior to processing, had the characteristic symptoms of byssinosis.

Effects on health of byssinosis

(i) Clinical findings. In the early stages of byssinosis, the characteristic symptom of chest tightness on the first day of work after the weekend break has widely been described. In Western countries this occurs on Mondays, usually towards the end of the shift. On Tuesdays, the worker may not have many symptoms, with the exception of dust irritation to the upper respiratory tract. As the disease progresses, the chest tightness accompanied by breathlessness worsens and extends to other days. Eventually the worker may become severely affected on every working day with permanent and severe effort intolerance, which is not noticeably improved by giving up his dusty occupation. In some instances, the chest tightness is more experienced in the last days of the working week.

In its final stages, the disease cannot be distinguished from chronic bronchitis and emphysema due to non-occupational causes, except for the past history of chest tightness characteristically worse at the beginning of the week. The patient often forgets his early symptoms and is diagnosed as suffering from a non-occupational chronic respiratory disease. Chest X-rays do not show changes specific for byssinosis, nor has any specific pathology been identified in the lungs of workers who have died of this disease. Thus, there is an acute stage in byssinosis which may progress to chronic irreversible disease. In the acute there is usually a loss of ventilatory capacity as measured by the Forced Expiratory Volume in one Second (FEV₁). This may also occur during dust exposure in workers without symptoms, more so on Mondays than on other days. The decline in ventilatory capacity during the work shift is a temporary phenomenon and is at first reversible but follow-up studies show a greater decline of FEV₁ per year among textile workers with a long history of dust exposure than among control subjects.

The decline in ventilatory capacity on a Monday also occurs in workers with long exposures to dust and evidence of permanent incapacity. It is nevertheless an early effect of exposure. The epidemiological evidence indicates that those who have severe acute symptoms in dust are more at risk of developing chronic and disabling disease than those who suffer less or not at all during acute exposures. However, direct evidence for this hypothesis is difficult to obtain, even in long-term follow-up studies.

Although it is not known how the acute stages of byssinosis progress to chronic, irreversible disease, some results indicate a relationship between the acute and chronic phases. Among older hemp workers, moderate or severe irreversible lung function loss occurred only among men who had acute Monday symptoms when they worked in the industry. Thus, people who regularly experience the acute symptoms of byssinosis were at greater risk of developing chronic disease than those who had never had these symptoms. In another sample from the same population of hemp workers, decreased lung function was found only in those who responded to acute dust exposure with decreases of expiratory flow rates.

(ii) Pathology. There have been few systematic studies of the pathological changes in the lungs of textile workers with byssinosis. Several case reports from the earlier literature, as well as some more recent observations, were reviewed in 1956 with only one additional report since then. The absence of reports of specific abnormalities in the lungs of patients with byssinosis suggests that any changes are of a non-specific nature and similar to those found in chronic bronchitis and emphysema. In particular, there is no evidence that long-term textile dust exposure leads to fibrosis of the lungs. The paucity of histological observations suggests the need for caution in interpreting the pathological process underlying "irreversible" ventilatory insufficiency in older textile workers. The loss of elastic recoil force in older hemp workers indicates that at least some of


these workers suffer from damage to the elastic structure of the lung parenchyma, similar to that in emphysema from other causes. However, in one reported case, only minor emphysema was found at autopsy of a man who died after having been totally disabled with severe dyspnea and cough during the last years of his life. It is conceivable, then, that the "irreversible" ventilatory insufficiency in these workers is caused, at least in part, by widespread small airway obstruction rather than by structural damage to the lung parenchyma. The fact that the ventilatory capacity of these workers usually cannot be increased significantly with short-acting bronchodilator drugs such as isoproterenol shows only that the small airway obstruction cannot be readily reversed with drugs which relax smooth muscle.

Mechanisms of action of vegetable dusts

(a) Dusts causing byssinosis. There are world-wide studies showing that dusts of cotton, flax and soft hemp cause byssinosis. For many years, epidemiological studies of workers exposed to sisal dust in the United Kingdom and Africa gave no evidence to suggest that this dust caused byssinosis. In a recent study, in Tanzania, workers heavily exposed to sisal dust were found to have the characteristic symptoms of byssinosis.

The pathogenesis in byssinosis is not certain. There is, however, evidence that a primary toxic reaction from a histamine-releasing substance may account for the acute signs and symptoms of byssinosis.

The evidence in favour of histamine-release as a principal mechanism may be summarised as follows:

- Cotton and other textile dusts cause airway constriction in most healthy persons when suitably exposed; no previous sensitisation is required.
- The time course of action of the cotton dust and of cotton dust extracts in vivo is consistent with an indirect effect, e.g. release of a stored mediator.
- Textile dust and bracts extracts release histamine from human lung tissue without previous sensitisation; the time course of histamine-release in these in vitro experiments is consistent with the time course of the in vivo response.
- The airway constrictor action of textile dusts in man in vivo is associated with increased excretion of a major histamine metabolite and can be prevented by a small dose of antihistamine drug.

The tachyphylaxis (decreasing response) observed after a first exposure to cotton dust extracts corresponds to the similar phenomenon observed after administration of known histamine-releasing agents under suitable conditions.

In purified cotton, this agent is absent and no byssinosis is observed. The airway constrictor as well as these histamine-releasing actions of cotton dust are caused by a water-soluble, heat-stable, small molecular compound derived from the bracts of the cotton plant. It seems probable that the two actions are caused by the same compound.

Although the histamine-release hypothesis can explain most of the acute effects caused by inhalation of textile dusts, such as cotton, flax and soft hemp, it does not explain all features of the disease. For instance, the slow progression of byssinosis in some individuals to a chronic stage cannot be explained by the histamine-liberation hypothesis.

It has also been suggested that byssinosis is caused by an immunological response to antigens in dust particles or to products of bacteria or fungi contained in the dust. Several types of antibodies have been demonstrated in the sera of byssinotic workers, but so far the relation between the antibodies and signs and symptoms of byssinosis has not been convincingly clarified. A purified antigen from cotton bracts was found to give higher antibody titres in the sera of byssinotic workers than in non-bysisnotic or in unexposed control subjects. However, the specificity of this antigen-antibody reaction has been questioned.

In guinea pigs exposed to cotton dust extracts, a correlation was found between the increase in number of leukocytes in the airways and the number of gram-negative bacteria as well as between the content of endotoxins in the dusts. A correlation between symptoms in workers and gram-negative bacteria in the air in cotton mills has also been reported. Endotoxins are capable of activating complement which leads to histamine-release and this could link the endotoxin hypothesis with that of histamine-release.

None of the hypotheses so far presented can explain all the features of byssinosis and it is likely that there is more than one type of human reaction to exposure. In addition to histamine-release, there may be an irritant effect or, in severe cases of byssinosis with an almost immediate effect on dust exposure which occurs every working day, it is possible that both antigenic and non-antigenic histamine-release factors may be important.

Exposure/response relationships and permissible levels: magnitude of problem

(a) Cotton dust. The relationships between exposure and response discussed in this section are based on epidemiological studies of cotton workers in many

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The terminology used first needs to be explained. Exposure/response relationship is the relationship between the exposure level of dust and the proportion of individuals in a defined group of workers with a specified effect, such as symptoms of byssinosis or decrement in ventilatory capacity during the shift. Exposure/effect relationship is the relationship between exposure to dust and the magnitude of the effect in an individual, that is the byssinosis grade and degree of acute and permanent change in ventilatory capacity. Exposure/effect is particularly important in making recommendations for employment for individual workers, but it is exposure/response relationships that are crucial to the setting up to permissible levels. The term "exposure limit", "permissible exposure", and "threshold limit value" are also taken to mean "maximum allowable concentration", "threshold limit value" and "maximum permissible limit or dose". A permissible level assumes that there is a level of concentration in the air of the working environment at which (below which) the worker does not incur any health impairment. It will be seen later that for cotton dust there seems to be no level of respirable airborne dust (except nil exposure) at which workers may not be affected. The recommended permissible levels in the United States and the United Kingdom are based on levels which it is possible to achieve in practice but which do not protect the susceptible worker; hence associated with these standards are recommendations for medical surveillance.

The information currently available on exposure/response relationships is imperfect in several ways, and it is unlikely that these deficiencies will be improved in the near future. The basic uncertainties concerning both the exposure levels and the response rates are due to the variable conditions in industry and to human variations in response.

(i) Exposure levels. Dust concentrations in textile factories vary in time and place, and the composition of the dust may differ. Variables include the type of production process, quality of material (grade of raw cotton), machine speeds and air management in workrooms. A separate set of additional variables are involved in the composition of cotton dust. Amounts of cotton trash and other constituents may vary. Because byssinosis is probably caused by chemical substances in textile dust rather than by dust particles per se, a case might be made for establishing exposure/response relationships on the basis of the amounts of active chemical components rather than on the basis of dust concentrations. Unfortunately, this is not yet feasible since the substances responsible for byssinosis have not been identified. The prevalence of byssinosis in cotton textile workers correlates better with the amounts of proteolytic enzymes in the mill air than with the amounts of airborne respirable dust. Analysis of the data supporting this view shows, however, that these enzyme concentrations correlate highly significantly with the dust concentrations themselves. For example, the chymotrypsin-like activity in "fine dust" from 17 cardrooms correlated positively with the fine-dust levels. Similar correlations are found between other enzymes/levels and dust concentrations. These correlations make it obviously impossible to assign more importance to one than the other. Similarly, claims that byssinosis prevalence is more closely related to bacterial contamination of dusts than to dust levels per se cannot be accepted because of the lack of valid evidence.

(ii) Response rates. The two principal methods of establishing biological responses to textile dust exposure are to determine the prevalence of symptoms of byssinosis, by questionnaire, and to measure decrements in ventilatory lung function, either during a workshift on first working days after absence from work (acute

effects of dust exposure) or over a worker's lifetime in the mills (chronic effects of dust exposure). Lack of data makes it impossible to relate chronic effects of dust to past and present exposure levels.

The questionnaire interview method is subject to several sources of error, and problems of interpretation. Questionnaire results may be affected by observer error and by variations in the perception of questions by the workers. The subjective nature of the information obtained also makes questionnaire interviews liable to error if, for instance, workers fear that they may lose their jobs as the result of answering the questions. It is important to stress that questionnaires have to be adapted to suit the cultural and working conditions of a country without loss of reliability in making comparisons between countries. Persons interviewing workers need to be specially trained in the use of questionnaires.

(iii) Exposure/response points. The first systematic attempt to define the relations between airborne cotton dust concentrations and byssinosis prevalence was made using total dust concentrations and responses estimated by questionnaire interviews. In this study, less than 8 per cent of the total dust (by weight) consisted of respirable particles ($<7 \mu m$). The authors proposed a tentative "safe limit" for cotton dust of 1 mg/m$^3$ total dust. Later investigators have often found much higher proportions of respirable dust in their samples, due in part to widespread introduction of exhaust ventilation, which decreased total dust concentrations, without eliminating respirable dust. It is now generally agreed that respirable rather than total dust concentrations in textile mills should be considered in byssinosis prevention and control programmes.

Prevalences of byssinosis (all grades) varying from 20 to 50 per cent have been reported in cotton cardrooms with "respirable dust" concentrations between 0.35 and 0.50 mg/m$^3$ (fig. 1). Prevalences of less than 10 per cent occur only in workrooms with less than 0.1 mg/m$^3$ respirable dust. However, one study in ginning reported a 19 per cent prevalence of chest tightness at the beginning of seasonal work with respirable dust concentrations of 0.11 mg/m$^3$. Thus, even when respirable dust concentrations are as low as about 0.1 mg/m$^3$, symptoms may occur in a sizeable proportion of workers after their return from an annual holiday. For cardroom workers, all dose-response points in fig. 1 fall between the high and low average prevalences shown as lines 1 and 2.
Fig. 1. Dose-response relations based on questionnaire interviews (prevalence of byssinosis, all grades)

- Solid line: initial portion ofMerchant and Albrecht's regression equation.
- This regression line is based on byssinosis prevalence as a function of respirable dust concentration, with the vertical axis representing "minimal response curve."
10. Diseases due to cadmium and its compounds

**Properties.** Cadmium is a white metal with a bluish tinge. The melting point is 320.9°C and boiling point 767°C. Nearly all cadmium is obtained as a by-product in the processing of zinc, copper and lead from ores in which cadmium is found in small amounts. Metallic cadmium and different cadmium alloys and compounds, such as cadmium oxide (CdO), sulphide (CdS) and chloride (CdCl₂), have found industrial use.

**Uses.** Cadmium is highly resistant to corrosion and therefore it is widely applied for electroplating of other metals. Cadmium is an important component of many alloys, such as cadmium-nickel, cadmium-silver-copper and cadmium-gold; cadmium-silver alloys are used for soldering. Cadmium is also used for the electrodes of alkaline storage batteries, in the manufacture of fluorescent lamps, in nuclear reactors, jewelry manufacturing and the automobile and aircraft industries.

Cadmium oxide is used for the make-up of cadmium electroplating baths, in glazes and as a catalyst.

Cadmium sulphide is used as a pigment in the textile, paper, rubber and pyrotechnic industries and in dyes, enamels and glazes.

Cadmium chloride is used mainly in the photographic industry.

**Hazards.** The fumes and dust of cadmium and its compounds (mainly cadmium oxide) are very toxic. Exposure occurs mainly in smelting and refining cadmium, welding cadmium-coated metals, spraying cadmium-containing pigments, handling materials containing cadmium, etc.

**Health effects:** Acute toxicity. Inhalation of high concentrations of fumes or dust of cadmium and its compounds results in irritation of respiratory tract, extending from a simple tracheobronchitis to severe lung edema or chemical pneumonitis.

Ingestion of soluble cadmium compounds (rarely occupational) causes severe haemorrhagic gastroenteritis.

**Chronic toxicity.** Adverse health effects of chronic cadmium exposure include local changes of the nose mucosa (anosmia), yellowish colouration of the neck of the teeth, lung emphysema, slight anaemia, osteomalacia with presence of striae on bone X-rays similar to Milkman-Looser syndrome (both usually only in subjects with nutritional deficiencies), and kidney damage.

**Diagnostic:** Acute effects. Symptoms of respiratory-tract irritation (cough, pain in the chest, dyspnea) develop with a latency of usually several hours after the occupational exposure. They may be accompanied by fever, chills and general weakness. Signs of bronchitis, lung edema or pneumonia are present at physical, laboratory and X-ray examinations.

**Chronic effects.** Among the effects mentioned, kidney damage is the most important because it may lead to renal insufficiency. It is also the most specific sign of chronic cadmium exposure as it is accompanied by proteinuria with typical electrophoretic pattern (mainly β2-microglobulin, but also albumin, transferrin, IgG, etc.). These urinary protein changes are the first detectable signs of functional disturbance of the kidneys in possibly reversible slope.

An increased excretion of cadmium in urine (limits given usually in the range from 20 to 100 μg/l or 10 to 15 μg/g creatinine, respectively) may support the diagnosis of chronic cadmium poisoning, but lower values do not exclude it. The elimination of cadmium in urine shows a broad individual variability.

**Prognosis:** Acute effects. Mortality up to 15 per cent in cases of severe exposure has been reported.

**Chronic poisoning.** When removed from the exposure at early signs of renal functional impairment, the prognosis is usually favourable. In continuing the exposure to cadmium, irreversible changes may develop causing renal insufficiency.
Legislation: Cadmium poisoning as occupational disease in different countries. According to information obtained by WHO from selected member States, diseases due to cadmium are included in the official list of occupational diseases in the following countries: Chile, Czechoslovakia, Finland, France, German Democratic Republic, Indonesia, Switzerland, Yugoslavia.

Besides, other member States, like Bulgaria, Poland, Thailand and USSR, define as an occupational disease acute and chronic poisonings and their sequelae due to any toxic chemical substance, if the disease is attributable to work. Thus, cadmium poisoning is included. In USA, 49 states enacted full coverage of occupational diseases, not limited to a list or schedule of specified diseases, enabling also the compensation of occupational cadmium poisoning. (For more details, see ILO synoptic table, Doc. MERLOD/1980/V.)

References


NIOSH Criteria for a recommended standard ... Occupational exposure to cadmium, DHEW (NIOSH) Publication, 1976.
II. Diseases due to fluorine and its compounds

Properties. Fluorine is a yellow gas, very reactive. Many of fluorine compounds are used in industry, both inorganic, such as calcium fluoride (CaF$_2$), cryolite (Na$_3$AlF$_6$), aluminium fluoride (AlF$_3$), hydrogen fluoride (HF), and organic, sodium fluoroacetate (POH$_2$000Na) and a large group of fluorocarbons, derived from methane, ethane, ethylene and other hydrocarbons.

Uses. Elemental fluorine is used only in a very limited number of special chemical operations. The basic inorganic compound is hydrofluoric acid which is made by the action of concentrated sulphuric acid on fluorspar (calcium fluoride) and used in the production of inorganic fluorides and fluorocarbons, in the refining of certain metals, as a catalyst in organic chemical reactions and in etching glass and pottery products.

The major uses of calcium fluoride are as raw materials for the production of hydrofluoric acid, in steel and glass manufacturing, iron foundries, and in special fluxes, including coatings for welding rods. Cryolite is used mainly as electrolyte in the manufacture of aluminium, aluminium fluoride in the manufacture of glass and ceramics, as a flux in metallurgy and in different chemical productions (catalyst; inhibitor of fermentation).

Sodium fluoroacetate is used as rodenticide and general mammalian pesticide.

Fluorocarbons developed to very important modern chemical compounds. They are used as refrigerants, propellants in aerosol formulations, foam blowing agents, fire extinguishers, solvents, anaesthetics (Fluothane) and as intermediates in polymer production.

Hazards. Fluorine and vapours or mists of hydrofluoric acid have strong irritative properties; hazard occurs wherever these substances are used without proper precautions.

Inhalation of airborne particulate material of inorganic fluorides is the main exposure route in mining these raw materials, in aluminium manufacturing, metallurgy, including welding with fluorine-coated rods, etc.

Hazards from fluoroacetates could arise only by improper handling and breaking basic safety rules and simple hygienic habits.

Fluorocarbons seem to exhibit toxic action only after repeated exposures.

Health effects. Health effects depend on the chemical composition of the fluorine-containing material.

Acute toxicity. Fluorine and hydrofluoric acid and aerosols of inorganic fluorides cause irritation of mucosae (acute conjunctivitis, acute bronchitis); a serious lung damage is possible but it is usually prevented by the strong irritation which makes presence in the contaminated atmosphere impossible. Hydrofluoric acid may cause chemical burns on the skin and mucosae.

Sodium fluoroacetate is a strong systemic poison of the central nervous system (convulsion with coma and respiratory depression) and of the cardiovascular system (cardiac rhythm irregularities, ventricular fibrillation, sudden death).

Fluorocarbons in repeated exposures even at concentrations which do not produce subjective adverse feelings are claimed to sensitise the cardiac receptors against endogenous catecholamines, giving the possibility of ventricular fibrillation. At least some sudden deaths of patients inhaling repeatedly broncholytic drugs from aerosol containers, where fluorocarbons are used as propellants, were explained in this way.

Controversial reports concern the possible hepatotoxic effect of fluothane used as a potent anaesthetic; whereas some anaesthesiologists consider it to be absolutely safe, others claim to have observed liver damage of patients after protracted or repeated anaesthesias.
Chronic toxicity. The typical disease caused by excessive chronic exposure to inorganic fluorides is fluorosis, which was first reported in 1933. The signs include increased radiographic bone opacity, formation of blunt excrescences on the ribs and ossification of intervertebral and other inter-osseal ligaments. This localisation of changes may lead to severe mobility reduction, especially of the spine.

The etching of hydrofluoric acid may have a detrimental effect on the dental enamel and accelerate the development of caries.

The elimination of fluorine in urine increases in subjects with recent exposure. Concentrations up to 4 or 5 mg fluorine per litre of urine are usually considered as upper safety limits. Normal values for occupationally non-exposed subjects are given usually as less than 1 mg/l urine, depending on the fluorine content in drinking water and food.

Diagnostic: Acute effects. The symptoms and signs of the irritation of mucous membranes or of chemical burns develop immediately at the exposure so that usually no diagnostic difficulties arise.

Sodium fluoroacetate poisoning is rarely to be of occupational origin if basic hygienic measures are kept when handling the substance.

As regards fluorocarbons, so far no undoubtedly documented cases of occupational poisoning have been reported.

Chronic effects. The X-ray picture of osteosclerosis and ossification of ligaments in connection with long-lasting occupational exposure to high concentrations of fluorides usually causes no diagnostic difficulties.

Prognosis. Prognosis of acute damage of the respiratory tract is usually favourable, but a severe damage may occasionally end in death or by permanent sequelae (fibrosis).

If occupational exposure is interrupted in early stages of chronic fluorosis, the disease stays at a stationary state. Continuing exposure may lead to crippling, disabling forms of the disease.

Fluorine poisoning as occupational disease in different countries. According to information obtained by WHO from selected member States, diseases due to fluorine are included in the official lists of occupational diseases in the following countries: Chile, Czechoslovakia, Switzerland, Yugoslavia.

Besides, other member States, such as Bulgaria, Finland and Poland, define as an occupational disease acute and chronic poisonings and their sequelae due to any toxic chemical substance, if the disease is attributable to work. Thus, fluorine poisoning is included. In USA, 49 states enacted full coverage of occupational diseases, not limited to a list or schedule of specified diseases, enabling also the compensation of occupational fluorine poisoning. (For more details, see ILO synoptic table, Doc. MERLOD/1980/V.)
Aromatic compounds containing halogen atoms (usually chlorine) are used as solvents, in the manufacture of dyes, pharmaceuticals, pesticides, resins, and may occur as chemical intermediates or impurities. The compounds may be classified into different groups according to their chemical relationships but in spite of their chemical similarity, their toxic effects differ considerably. Examples of only the most important groups will be given.

Chlorinated benzenes

Chlorinated benzenes are aromatic (benzene) rings with one or more chlorine atoms substituted for hydrogen atoms.

Monochlorobenzene is a colourless liquid with a pleasant odour used as a solvent, and as an intermediate in chemical production (especially dyes).

o-Dichlorobenzene is used as a solvent, fumigant, insecticide and chemical intermediate.

p-Dichlorobenzene is, in contrast to the orthoisomer, a white, strongly odorous crystalline substance, used as insecticide, chemical intermediate, disinfectant and moth preventative.

Compounds with more chlorines (trichloro- up to hexachlorobenzene) are crystalline substances, not as widely used in industry, but nevertheless used as chemical intermediates.

Chlorinated derivatives of toluene (benzyl chlorides = monochloromethylbenzene, di- and trichloromethylbenzene) are also chemical intermediates of industrial application. They have similar health effects.

Health effects. The route of entry is absorption of vapours or dust by the lungs; liquids may be absorbed also through the skin.

All chlorinated derivatives of benzene and its homologues have irritating properties to mucous membranes and eyes. In high concentrations, acute tracheobronchites may develop, and highly irritating substances may cause even lung edema. Monochlorobenzene and o-dichlorobenzene, which are less irritating, exert also a narcotic effect (feeling of instability, drunkenness, coma).

In chronic exposure, the possibility of damage to liver, kidneys and/or nervous system is suspected from the results on animal experiments, but no specific clinical picture of chronic poisoning is known in humans.

Chlorobiphenyls and derivatives

Chlorobiphenyls are biphenyl rings in which one or more hydrogen atoms are replaced by a chlorine atom. Synonyms are polychlorinated biphenyls, PCB. Most widely used are chlorobiphenyls with 3 or 5 chlorine atoms. These compounds are light, straw-coloured liquids with typical chlorinated aromatic odour.

Chlorinated biphenyl oxides are ethers. They range from clear, oily liquids to white or yellowish waxy solids, depending on the degree of chlorination.

Chlorinated biphenyls are used alone and in combination with chlorinated naphthalenes. They are stable, thermoplastic and non-flammable and find chief use in insulation for electric cables and wires, in the production of electric condensers, as additives for extreme pressure lubricants and as coatings in foundry production.

Health effects. These substances are absorbed as vapours through the lungs; percutaneous absorption of liquids is possible too.

Skin contact may cause the formation of comedones, sebaceous cysts and pustules known as chloracne. The changes are very persistent, lasting for a long time even after discontinuing the work.
Acute and chronic exposure can cause liver damage (toxic hepatitis). Fatal cases have been observed.

It is believed that chloracne develops from skin contact and/or internal absorption (inhalation, ingestion) of the substances, while systemic (liver) effects result primarily from internal absorption. Systemic poisoning may occur without the development of chloracne and vice versa.

Chlorinated naphthalenes

The substitution of one or more hydrogen atoms by chlorine in the naphthalene molecule changes the physical states of the compounds from mobile liquids to waxy solids, depending on the degree of chlorination.

Due to their stability, thermoplasticity, and non-flammability, they find uses similar to chlorinated biphenyls.

Health effects. The toxic effects are almost identical to those of chlorinated biphenyls (chloracne, liver damage).

Chlorinated dibenzo-p-dioxins

These substances have no industrial use, but they were synthesised in research laboratories and may occur as undesirable by-products of some chemical procedures in industrial preparations of 2,4,5-trichlorophenol (used for the production of herbicides such as trichlorophenoxyacetic acid or the bactericide hexachlorophene). Group poisonings occurred in workers in such factories, and large groups of general population were affected following industrial accidents or release of the substances in the environment because of the extremely high toxicity of these substances, mainly tetrachlorodibenzodioxin.

Health effects. Routes of entry are respiratory and partly also skin absorption.

Again, chloracne is the most frequent illness produced. The systemic effects reported comprise liver disease (including hepatic porphyria with skin changes typical for porphyria cutanea tarda, disorders of the lipid, carbohydrate and protein metabolism) and damage of the nervous system (polyneuropathies, psychic disturbances).

The disease has been observed in subjects exposed for no more than a few days. Fatal cases were reported.

References


Diseases due to nitroglycerin and other nitric acid esters

General

The most important substances are esters of nitric acid and glycerol or glycols: nitroglycerin, nitroglycols (ethylene glycol dinitrate, diethylene glycol dinitrate). They are pale yellow oily liquids, highly explosive. The explosion may be initiated by heat, but even by mechanical shock or by spontaneous chemical reaction.

Uses and hazards. The compounds are produced by nitration of glycerol or glycols with nitric acid. The industrial explosives (called usually dynamites) are prepared by mixing nitroglycerin and/or nitroglycols with other ingredients, like carbonaceous combustibles or nitrocellulose and oxidants, like ammonium or sodium nitrate. The final product has usually a consistency of hard wax or gelatin and is delivered in the form of rods of different shapes and sizes.

In manufacturing the explosives, there is a constant danger of fire and explosion. The final products can be handled far more safely.

Nitroglycerin and nitroglycols are readily absorbed through the lungs (as vapours) and also through the intact skin at direct contact. Acute poisonings were observed both in explosive producing industry as in use of explosives (mainly in mines). Chronic poisonings were limited practically only to the manufacturing.

Health effects. Nitroglycerin and similar nitroglycols have strong vasodilatory and slight methemoglobinising effects; the latter may be accompanied by development of small Heinz bodies in erythrocytes. These properties of nitroglycerin lead to its therapeutic use: in ischemic heart diseases because of the vasodilatation, and in acute hydrogen cyanide poisoning because of methemoglobin formation. Systemic effect on the cardiovascular system has been ascribed to the chronic exposure to nitroglycols.

Acute poisoning. Intensive headache (due to vasodilatation of intracranial blood vessels) is usually the first symptom. It may be followed by nausea, vomiting, cyanosis, fainting due to the lowering of blood pressure, coma.

Chronic poisoning. Following an exposure period of several years, sudden deaths in workers producing nitroglycol containing explosives were observed. Some of the victims suffered from anginal pains or repeated collapses, but others were without any previous troubles. The death occurred usually after a work-free weekend, before re-entering the work on Monday. Also the anginal pains showed dependency on rest. Contrary to the attacks accompanying ischemic heart disease, which are provoked by physical exercise, the angina in chronic nitroglycerol poisoning occurs usually after discontinuing the work for several days. ECG findings did not prove any organic damage of the heart muscle, and autopsies also proved negative, although in some cases coronary arterio-sclerosis was found. However, no atherogenic effect of nitro esters has been observed.

The pathogenic mechanism of sudden death in chronic exposure to nitroglycols is uncertain, since the coronary insufficiency is far from having been proved. It is suspected that the origin of heart failure may be due to metabolic changes of the myocardium, changes in the sensitivity of the heart conductivity or sudden release of catecholamines.

In addition to the dominating cardiovascular symptomatology, minor disperse changes of the central and peripheral nervous system may be found.

Diagnosis. In acute poisoning, the typical clinical picture develops after high exposure, either by inhalation of vapours (in explosive production, after blasting in mines) or by skin absorption (manufacturing, contact with mineral material contaminated by explosives after blasting).

In chronic poisoning, withdrawal symptoms as described above are the only diagnostic aid.
Prognosis. The prognosis of acute poisoning is usually favourable. In chronic poisoning, after removal of the worker from the exposure, the symptoms (angina, collapses) diminished, but in some individuals they may persist even for several years.

References

Additional references in all these papers.
14. Acute poisonings and their sequelae caused by toxic substances not included in the present list of Convention No. 121

General

The List of Occupational Diseases (Schedule I of Convention No. 121) enumerates diseases caused by a limited number of substances, the adverse effect of which — in the sense of chronic poisoning — had been indisputably proved and generally accepted. The development of the chemical industry has brought about an ever-increasing number of new chemical compounds, some of them having very dangerous properties. It is felt that diseases caused by these substances should also be covered by benefits of Convention No. 121. This draft of a new Schedule includes an enlarged number of substances and will improve the situation.

Nevertheless, none of the lists enumerating individual substances will be able to cover the hazards arising from new, and even undiscovered compounds. Therefore, some countries avoided the enumeration of specific substances and define as occupational disease any disease (acute or chronic poisoning and/or their sequelae) caused in the course of work by any chemical substance, if the disease is obviously attributable to the work performed. Such is the situation, e.g. in Bulgaria, Finland, Poland, Thailand and the USSR. Similar approaches have been used by those states of the United States which enacted several coverage of occupational diseases.

However, such broad conception of occupational diseases could lead to uncertainty if adopted in an international Convention, especially with respect to the often existing controversies of even highly qualified medical experts about the nature of some chronic poisonings. The questionable chronic carbon monoxide poisoning may be given as the example.

In acute poisonings, however, the diagnostic uncertainty is avoidable because the clinical picture together with the proven occupational overexposure (usually connected with a breakdown at the workplace) makes the diagnosis sure.

Schedule I of Convention No. 121 does not cover many very important and often occurring occupational poisonings.

The following groups of noxious agents, according to their mode of action, are proposed for inclusion.

1. Asphyxiants

Asphyxia is the diminished supply of oxygen to the lungs or to the body tissues. The organ most sensitive to lack of oxygen is the brain and the damage of the central nervous system is the most important feature.

(a) Asphyxia caused by low oxygen content in the air. It is caused by gases which are in general not toxic but which may displace the available oxygen in the atmosphere and reduce it to a level incompatible with life. A decrease of oxygen content in the atmosphere to a concentration of 17 per cent or less causes insufficient saturation of blood haemoglobin by oxygen. The same effect is caused by the low partial pressure of oxygen at high altitude, but this kind of injury could be only exceptionally occupational (in air crews). The simple asphyxiants gases include nitrogen, carbon dioxide, gaseous aliphatic hydrocarbons (methane, ethane, propane, butane), helium, neon, argon. As their effect is proportional to the extent they reduce the partial pressure of oxygen in the inspired air, they are hazardous only in high concentrations such as may occur only in confined spaces.

Nitrogen. In industry, most of the nitrogen is obtained by fractional distillation of liquefied air. It is used in large amounts in the production of ammonia and compounds derived from it, such as nitrogen-containing fertilizers. Free nitrogen is used in many industries as an inert medium to prevent accidental chemical reactions due to oxidation. Following detonation of explosives in mines, the air may contain up to 87 per cent nitrogen and about 13 per cent carbon dioxide.

Dangerous concentrations of nitrogen may occur usually after mechanical breakdown of nitrogen-holding containers within enclosed spaces or in mines as mentioned above.
Carbon dioxide. The gas is produced when carbonaceous substances burn in an excess of air or oxygen. It is a product of fermentation processes, synthetic ammonia production, limestone calcination and numerous reactions in the chemical industry. Carbon dioxide is used in the form of gas to neutralise excess alkali in many chemical industries, in shielded-arc welding, for carbonating beverages such as soft drinks. Liquid carbon dioxide is used in fire-extinguishing equipment, for refrigerated storage, etc. The solid form called Dry Ice is used for many refrigeration purposes.

Again, dangerous concentrations may develop mainly after mechanical breakdown, but also in containers for fermentation or silos (agriculture, food industry). Erroneous entering in such closed rooms has seldom been the cause of serious injuries.

Aliphatic hydrocarbons. The saturated aliphatic hydrocarbons with small molecules (up to four carbon atoms), i.e. methane, ethane, propane and butane, are gases at room temperature. They are produced mainly by the cracking, distillation and fractionation of crude oil, which is a complex mixture of hydrocarbons. Methane comprises 85 per cent of natural gas, and may be formed also by fermentative decomposition of cellulose. All these substances are used mostly as fuels, in oxy-propane welding and cutting, etc. For distribution, they are very often liquefied and stored even in large amounts of thousands of tons. Pure hydrocarbons find use also in chemical syntheses.

Mechanical breakdowns are the best known causes of injuries, as well as entering closed spaces containing contaminated atmosphere.

Helium-group gases. Helium, neon, argon are produced by cryogenic separation from liquid air. One of the major industrial uses is in metal smelting, refining and welding, where they provide an inert gas shield.

Use without proper ventilation or mechanical breakdown are again the causes of injuries or even death.

Health effects. The slight irritating effects on mucosae of some of the substances (carbon dioxide, propane, butane) in volume concentrations of up to 5 per cent is negligible in comparison to the asphyxiant effect of high concentrations which lower the partial pressure of oxygen in the air. Humans are affected when the percentage of oxygen falls below its normal level of about 21 per cent in the air to 17 per cent or less. At about 15 per cent disizziness and general weakness are noticeable and towards 10 to 12 per cent unconsciousness usually results. However, carbon dioxide exerts also specific unfavourable effects at concentrations of over 5 per cent (shortness of breath, headache), but still the asphyxiant effect of oxygen deficiency at high concentrations prevails. The brain may tolerate an insufficient supply of oxygen for a very limited period of time only. In an oxygen-free atmosphere, loss of consciousness is a matter of a few seconds, starting suddenly without any warning symptoms. Irreversible brain damage or death has been observed after several minutes stay in such an atmosphere.

Chronic poisonings due to exposure to lower concentrations than causing oxygen deficiency are not known.

Prognosis. The prognosis is dependent on the level of blood oxygen depression and on the duration of the stay in the oxygen-deficient atmosphere. If the patient is removed early (i.e. in a few minutes in case of breathlessness) complete recovery is possible. However, permanent neurological damage may be the result of prolonged exposures.

(b) Asphyxia caused by impaired transport or utilisation of oxygen in the tissues. The main causes are either changes of the oxygen-binding capacity of haemoglobin in blood (poisoning by carbon monoxide or methemoglobin-forming substances, such as nitrates or amino and nitro derivatives of aromatic hydrocarbons) or of the oxidising enzymes in the cells (poisoning by hydrogen cyanide or hydrogen sulphide). Among them, carbon monoxide poisoning is the most frequent and most important occupational poisoning.

Carbon monoxide. Carbon monoxide is a colourless, tasteless gas, lighter than air. Its mixtures with air are flammable and possibly explosive.
Carbon monoxide is formed by incomplete combustion of organic materials such as wood, coal, coke, oil, gas (high access of oxygen leads to formation of carbon dioxide). Carbon monoxide itself is used only in a very limited number of chemical procedures (reducing agent in nickel production, some organic synthesis). However, due to the widespread use of carbonaceous fuels, carbon monoxide may be encountered in dangerous concentrations in many workplaces, as a waste product of incomplete combustion. It is found in the exhaust gases of internal combustion engines, gas manufacturing plants and coke works, blast furnaces in the iron and steel industry, oil refineries, in various types of oil, coal or gas heated furnaces, in coal mines. It may accumulate in any closed room where there is an accidental fire.

Health effects. Acute carbon monoxide poisoning is the most frequent occupational poisoning.

Carbon monoxide is highly toxic. Its affinity to haemoglobin is far stronger than that of oxygen. When entering blood through the lungs, carbon monoxide combines with haemoglobin to form carbonylhaemoglobin. Blood loses its oxygen-carrying function and a state of tissue hypoxia develops. Again, the brain is the most sensitive organ.

Acute toxicity. Moderate headache, dizziness, sometimes chest tightness, muscle weakness and nausea are usually felt at carbonylhaemoglobin levels above 15 per cent. With increasing concentrations of carbonylhaemoglobin (which depend both on carbon monoxide concentration in the air and the duration of exposure) the symptoms become more serious being accompanied by confusion and indecisiveness. At about 45 per cent of carbonylhaemoglobin, collapse is possible, and loss of consciousness comes at about 50 to 60 per cent. At yet higher concentrations, the risk of death is imminent.

If the exposure to carbon monoxide is of only short duration the patient usually recovers fully even from serious poisoning. Permanent damage of the central nervous system is rare, but it may occur as a result of prolonged exposure at high carbonylhaemoglobin concentrations. Myocardial damage has been repeatedly reported, too, probably on the basis of preceding atherosclerosis of coronary arteries.

Chronic poisoning. Chronic poisoning caused by long-term exposures to carbon monoxide concentrations, in which carbonylhaemoglobin levels did not surpass 15 per cent, is claimed by some authors, whereas others deny it. Chronic sequelae of repeated slight poisonings are, however, admitted as a rule. The clinical pictures are very similar to atherosclerotic brain changes (disperse brain damage).

Chronic effects. Because of the very vaguely defined clinical picture of the chronic poisoning, no specific diagnostic signs can be recommended.

However, if chronic sequelae (brain or heart damage) develop immediately following a severe and protracted acute poisoning, there is no doubt of their causal relationship.

Prognosis has been mentioned above.

Hydrogen cyanide. Hydrogen cyanide (hydrocyanic acid) is a colourless gas with a characteristic odour of bitter almonds. It is used as a disinfectant or rodenticide and in the synthesis of chemicals, especially acrylonitrile. It is easily released from its salts, the cyanides, by action of acids. Cyanides are widely used in electroplating, steel hardening, extraction of gold or silver from ores, in manufacture of chemicals and for many other purposes.

Health effects. The highly toxic property of hydrogen cyanide depends on its ability to inhibit enzymes required for the respiration of cells, the most important being the inhibition of cytochrome oxidase. The utilisation of molecular oxygen by cells is thus prevented and tissues are damaged by anoxia. The toxicity of cyanides depends on their ability to release the cyanide ion. Some of them, like sodium or potassium cyanide, are decomposed even by action of faint acids, but the complex compounds, such as potassium ferricyanide are very resistant even to strong acids.
In spite of the high toxicity of hydrogen cyanide and the broad use of cyanides in the industry the poisonings are not frequent. They have been reported mainly in production or direct use of hydrocyanic acid or due to accidents at which cyanides (usually their solutions) were mixed with acids, e.g. in waste waters.

In acute poisoning, symptoms such as weakness, headache, tightness of the chest, confusion, nausea, vomiting may develop, followed by unconsciousness. At very high concentration, coma can occur suddenly after some seconds of exposure, without previous warning symptoms.

Chronic poisoning due to exposure to lower concentrations than causing acute symptoms is not generally accepted.

Prognosis. Prognosis is similar to that of other asphyxiants: if removal of the patient from the toxic atmosphere is rapid and proper treatment is started, complete recovery is usual. After protracted unconsciousness, permanent brain damage may persist.

Hydrogen sulphide. Hydrogen sulphide is a colourless gas with a characteristic rotten-egg odour. It is produced by reacting sulphides (usually iron sulphide) with diluted sulphuric or hydrochloric acid or by reacting hydrogen with sulphur. It occurs as the by-product of many chemical processes involving viscose rayon, synthetic rubbers, petroleum products, etc. It is also released during the decay of sulphur-containing organic material.

Uses and hazards. Hydrogen sulphide is used for the production of various inorganic sulphides or organic sulphur compounds. Health hazard is usually connected with technical breakdown or without regard to the rules of work safety.

Health effects. Like hydrogen cyanide, hydrogen sulphide inhibits respiratory cell enzymes, leading to tissue anoxia. The brain is again the main target organ.

Low concentrations are readily detected by the characteristic unpleasant smell. Irritation of the eyes (feeling of burning) and mucosae of the respiratory tract may be felt.

General symptoms of acute poisoning are dizziness, headache, nausea or vomiting, unconsciousness. Inhalation of a high concentration of hydrogen sulphide produces rapid coma and death.

At high concentrations, the smell of hydrogen sulphide is not perceived and exposure loses the warning signs.

The existence of chronic poisoning is denied by some authors, while others describe it as a variety of unspecific complaints and signs.

Prognosis. If the exposure has been of only short duration, the patient usually recovers even from serious poisoning. Permanent brain damage has been, however, observed as well as damage of the heart muscle.

2. Narcotic agents (organic solvents)

This is a group of chemically different substances, technically described usually as organic solvents. Their physiochemical characteristics are their volatility and lipid solubility.

The most important chemical groups of organic solvents are: aliphatic and aromatic hydrocarbons (halogenated hydrocarbons), alcohols, ethers, glycol derivatives, esters, and ketones.

Uses and hazards. Organic solvents are used for a variety of industrial purposes, as materials for chemical synthesis, as solvents of resins, plastics, fats, etc. They constitute the main liquid part of different paints, lacquers, inks; they are used for extracting oils, fats and other non-water soluble compounds from many vegetable or animal materials. Some of the organic solvents are used for degreasing in industry and dry cleaning. Chemicals are frequently dissolved in organic solvents to facilitate chemical reactions between them.
Acute poisoning may develop only in high concentrations, which can be reached usually only in restricted spaces, where solvents are used without proper ventilation, or after technical breakdown. However, even in the open air slight acute poisoning may occur if there is a possibility to inhale high amounts of the solvents evaporating, e.g. from large surfaces. Not only respiratory exposure is dangerous, but some organic solvents may be absorbed to a considerable extent even through intact skin.

Health effects. The only reason why a group of such chemically miscellaneous substances is put together is the common narcotic effect they exert on the central nervous system. Besides, some of the compounds have specific organotoxic effects, causing, e.g. liver or renal damage in acute poisoning (like carbon tetrachloride and other chlorinated hydrocarbons), or damage of bone marrow, like benzene in chronic poisoning. Only the acute narcotic effects will be included for consideration.

The nervous system is sensitive to organic solvents because of its high content of lipid-like substances. The narcotic effect is introduced by a feeling of drunkenness (instability of movements and walking, headache, dizziness, sometimes excitability) and may be accompanied by irritation of the eyes and mucosae of the respiratory tract. If exposure to high concentrations continues, loss of consciousness, coma and death due to respiratory arrest may develop. Prolonged contact of the skin to liquid solvents may cause also chemical burns.

Prognosis. Prognosis is usually favourable even after severe poisoning. Permanent neurological sequelae are very seldom. If the solvent has a specific organotoxic effect (liver, kidneys, etc.) the possibility of injury to these organs must be taken into account.

3. Irritating agents

A group of vapours or gases exerts an irritating effect on the cornea and conjunctiva of the eyes and/or on mucosae of the respiratory tract. Their chemical composition is again very different. The most important substances are: bromine, chlorine and its derivatives (hydrochloric acid, phosgene), sulphur dioxide, nitrogen oxides, ammonia, vanadium pentoxide, aliphatic amines, halogenated alkyln, aryl, or alkyl aryl oxides, halogenated alkyl aryl, or alkyl aryl sulphides, aliphatic sulphur-containing compounds (dimethyl sulphate), aldehydes (acrolein, formaldehyde, etc.), nickel carbonyl. However, the full list of all irritants cannot be enumerated.

Uses and hazards. Because of the widely varying chemical structures and often high reactivity, substances of this group are used in nearly all branches of industry. They can be found in chemical plants as well as in metallurgy, in laboratories, mining and ore processing, refrigeration industries, painting and varnishing shops and many others. Sometimes they are undesirable by-products of different procedures, e.g. nitrogen oxides are formed during electric welding, in contact of nitric acid with metals, or in recently filled silos; phosgene may be formed by thermal decomposition of chlorinated hydrocarbons when using carbon tetrachloride fire extinguishers.

If liquids are used, high concentrations of their irritating vapours may be found in the workplaces. Usually the irritating effect of vapours signals the danger and forces the worker to leave the hazardous room in time. Gas escaping from leaking containers or pipes and any mechanical breakdowns are the most dangerous causes of health impairment. If the gas or vapour is heavier than the air, as in the case of chlorine, it can spread over large areas and affect many persons. Particularly high concentrations may be reached in poorly ventilated workrooms.

Health effects. The painful sensation in the eyes forces the subject to close the eyelids and therefore serious damage of the eyes is extremely exceptional.

On the contrary, breathing cannot be stopped, although an inhibitory reflex may slow down the respiration. The main health risk is therefore the damage of respiratory organs, reaching from a simple acute tracheobronchitis to chemical bronchopneumonia or lung oedema.

The majority of the irritants causes symptoms immediately at exposure: painful or burning sensations in the nose, throat and retrosternal, irritation to cough and sometimes shortage of breath (due to bronchospasm and secretion).
The objective findings correspond to simple hyperemia of the mucoes to typical signs of bronchitis. In mild exposure, the symptoms of irritation cease usually until the next day; in more severe injuries, longer lasting bronchitis may develop or even bronchopneumonia.

Lung oedema is a much feared complication which develops usually several hours after the exposure, characterised by heavy breathlessness and expectoration.

Some of the irritating substances are more insidious as they have no strong immediate irritative effects, but the lung damage (especially lung oedema) appears after several hours of latency. This is especially the case of nitrogen oxides (silo-filler's disease) and phosgene.

**Prognosis.** Adequate treatment and absolute body rest are the two main measures influencing prognosis. Recovery is usually complete, but permanent sequelae (e.g. lung fibrosis, bronchoostenoses or bronchiectasias) as well as fatal course of bronchopneumonia and lung oedema have been observed.

4. **Pesticides**

The word "pesticides" denotes chemical substances used for the destruction of organisms thought to be detrimental to man or to his interests. It includes a number of other terms (e.g. insecticides, fungicides, herbicides, rodenticides, bactericides, miticides, nematocides, molluscicides) which indicate the organisms designed to be killed by the particular chemicals.

The chemical compositions of different pesticides vary greatly. Recently, mainly three classes of them have found the broadest use: chlorinated hydrocarbons (DDT, hexachlorocyclohexane, etc.), organophosphorus compounds and carbamates. However, a large number of other both inorganic compounds (e.g. arsenic, mercury, sulphur, copper, thallium compounds) and organic compounds (e.g. bromomethane, dinitro-o-cresol, coumarin, cresols and derivatives) are in use too.

**Uses and hazards.** Although the manufacturing of pesticides is connected with health hazards, the majority of reported poisonings occurred at application of pesticides. The methods of application depend on the physical state of the formulation. Most often it is a solution or water dispersion, sometimes a solid substance (dust, granules), exceptionally gas. Therefore, pesticides are applied mostly by spraying (by hand, from agricultural machines on the ground or from airplanes). The inhalation of airborne material represents the main hazard, but considerable amounts of some very toxic substances may be absorbed through the intact skin. The use of protective equipment (respirator, clothing, gloves, boots, etc.) is necessary as a rule when mixing or applying pesticides.

**Health effects.** Because of the diversity of the chemical composition of pesticides, their health effects vary considerably and cannot be summarised. After absorption, systemic general acute poisoning may develop, and the clinical picture differs according to the substance. Some compounds have irritating effects on eyes, mucoes of the respiratory tract, or even on skin. Chronic poisoning (resulting from long-term exposure to low concentrations) is known only for a few substances.

The majority of cases of pesticide poisoning falls into other items of the List of Occupational Diseases according to their chemical structure, e.g. diseases caused by phosphorus, arsenic, mercury, lead and their toxic compounds, by carbon disulphide, toxic halogen derivatives of aliphatic hydrocarbons or by toxic nitro and amino aromatic derivatives. Their number will increase after the enlarged new List is approved. Therefore, no details of health effects need be mentioned. Nevertheless, there still will be substances not on the List, which may cause acute poisonings. The best examples are carbamates or thiocarbamates, which act like organophosphorus compounds (inhibition of acetylcholinesterase) or the very dangerous bipyridyl derivative paraquat (hepatic and renal toxicity, progressive hyperplastic lung changes).

**Prognosis.** The recent pesticides try to have a specific effect on the selected organisms, and to be as low toxic for man as possible. The prognosis of acute poisoning is therefore usually favourable. However, there are some substances in use, e.g. paraquat, which always endangers life in cases of acute poisoning.
Acute poisonings as occupational diseases in different countries. Some States, such as Bulgaria, Finland, Poland and the USSR define as an occupational disease acute and chronic poisonings and their sequelae due to any toxic chemical substance, if the disease is attributable to work. In the United States, 49 states enacted full coverage of occupational diseases. Thus, in these states all acute poisonings are covered by the benefit of legislation for occupational diseases.

In other States, where there are lists of occupational diseases enumerating individual chemical causes of occupational diseases, most of the substances dealt with above are included in these lists, by the following definitions:

- Chile: chemical asphyxiants;
- Czechoslovakia: carbon monoxide, hydrogen sulphide, cyanide compounds, vanadium and its compounds;
- Egypt: carbon monoxide, hydrogen cyanide, chlorine, bromine, petroleum products;
- Finland: vanadium, halogens and their inorganic compounds, cyanide compounds, sulphur dioxide, sulphuric acid, hydrogen sulphide, nitrogen oxides, ammonia, carbon monoxide, phosgene, hydrocarbons, aldehydes, ketones and alcohols, etc.;
- German Democratic Republic: hydrogen sulphide, methanol, carbon monoxide;
- Indonesia: ammonia, carbon monoxide, cyanides, sulphur dioxide;
- Singapore: carbon dioxide, carbon monoxide, cyanides, nitrous gases;
- Switzerland: acetone, acetates, methanol, nitrous gases, hydrogen sulphide, carbon monoxide;
- Yugoslavia: vanadium and its compounds, aliphatic hydrocarbons, sulphur dioxide, hydrogen sulphide, alcohols, esters, ketones, ethers, cyanides, nitrous gases.