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OCCUPATIONAL EXPOSURE AS A FACTOR IN RESPIRATORY IMPAIRMENT

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A review of the literature data on the effect of specific substances – common in industry as environmental pollutants – upon respiratory organs is presented.

Chronic bronchitis in industry is discussed separately, especially from the point of view of long-term effects of irritant gases and inert dust exposure. A part of the review relates to the diagnostic problems of bronchitis, as well as to the study of the so-called obstructive ventilatory diseases of the lung in relation to occupation.

I. INTRODUCTION

Advances both in hygiene and in the treatment of disease, improved socioeconomic and working conditions, changes in nutrition, a prolonged life expectancy, etc. have considerably altered the morbidity and mortality pattern. In all advanced countries acute bacterial infections are no longer such a problem as they were in the past. On the other hand, chronic degenerative diseases, connected with changes in organic systems but etiologically unexplained in most cases, are growing in importance. Some of the advances themselves, such as the life in industrial communities with its attributes and developments in production have brought diseases that either were unknown or were of little significance not long ago.

This general feature includes also the diseases of the lung. A half-century ago tuberculosis was one of the most frequent causes of death. In California, for example, at that time tuberculosis accounted for 15 percent of all deaths. Now the same disease causes less than one percent.

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(1) On the other hand the so-called obstructive ventilatory disease of the lung (2) and lung cancer have become very important. In California lung cancer takes now four times more lives than tuberculosis (1). Emphysema as a cause of death has increased 400 percent from 1950 to 1957 (1). The importance of chronic bronchitis in some countries, both in the morbidity and mortality, is well known.

Studies which were carried out had shown the association of obstructive ventilatory diseases of the lung and lung cancer with certain environmental factors such as air pollution and cigarette smoking (3–9).

It has been very well known that some chronic diseases of the respiratory organs have originated from occupations. Silicosis is one of the first examples. At the end of the nineteenth century it was realized that malignant diseases of the lungs may also arise as a result of occupational exposure. This was shown in Schneeberg miners.

The discovery of the causative relationship between such chronic respiratory conditions and occupations was not always an easy and fast process. For example, it has taken a lot of time to understand the role of coal dust in the occurrence of chronic lung disease among the coal miners. Although the condition known as miners' asthma was observed very early, it passed practically unexplained for more than 100 years. The introduction of mechanical methods in mining resulted in a great increase of dust, and by 1930 the lung diseases of coal miners were again compelling attention, particularly in South Wales. In 1942 *Hart and Aslett* (10) after a long-term study underlined the importance of coal dust and not rock dust as the causative agent in pneumoconiosis of coal workers. In the United States until very recent years it was thought that coal miners' pneumoconiosis did not carry any disability or any other threat to health. But later it was concluded that the soft-coal miners here do have so-called coal workes' pneumoconiosis identical with that seen in South Wales (11).

Berylliosis has to some extent a similar history. By 1936 the dust of beryllium fluoride was suspected of being hazardous. But at this time animal experiments supported the erroneous view that beryllium ion was non-toxic (12). Only after a few years of exposure was it recognized that man could suffer even fatalities from the inhalation of beryllium compounds.

Step by step the list of occupational hazards which can cause diseases of respiratory organs has been extended. Although the presentday working conditions are much better than they used to be, there is no doubt that many of these occupational factors have still to be taken into consideration in the development of respiratory impairment. The role of occupational exposure in the occurrence of respiratory diseases is the subject which will be discussed in this review.

II. OCCUPATIONAL HAZARDS AND THEIR EFFECT ON RESPIRATORY ORGANS

Diseases of the Lung Caused by Dusts or Fumes

Many materials appear in the working environment in the form of dusts or fumes. Both dusts and fumes are composed of particles. While fumes represent a stage of attenuation of solid matter with particles of such a small size as to resemble gases, dusts consist of larger particulate matter which in general have the same chemical composition as the substances from which they are derived. When airborne, dusts and fumes can be inhaled into the lungs and cause either acute or chronic lung diseases.

In industry fumes are usually the oxides formed from hot or boiling metals. Dusts derive from different inorganic and organic substances.

From the point of view of occupational lung diseases caused by the inhalation of dusts, inorganic dusts which contain silica either in the form of free silica (SiO_2) or in the form of silicates have been given special attention. Exposure to dust with sufficient content of free crystalline silicon dioxide may result, as already mentioned, in the causation of silicosis. This dust has been most frequently met in the quarrying industry, in the pottery industry, in gold, tin, iron and coal mining, as well as in the different other processes such as grinding of metals, metal polishing, sandblasting, silica milling, manufacture of abrasive soap, etc. Mention should also be made of pneumoconiosis due to diatomaceous earth (13, 14). Diatomaceous earth consists of the silica skeletons of aquatic plants: diatoms. Flux-calcined diatomaceous earth seems to be more hazardous than the natural diatomite (15–17).

Some silicates may also produce specific diseases of the lung. Dust of asbestos, which is a silicate containing iron, magnesium and aluminum can produce asbestosis. Asbestos is used in the manufacture of a wide range of insulation products and of a great variety of building materials, especially in combination with cement. Exposure to asbestos dust also arises in mining, crushing and disintegrating of asbestos. Harmful effects have also been attributed to other silicates, such as talc (18, 19).

Specific effect on the lungs arises from metal dusts or fumes, such as beryllium, aluminum (20, 21), cadnium and »hard metals« (22, 23). These are carbides of tungsten which are usually mixed with other metals, including titanium and cobalt. Interstitial fibrosis of the lungs caused by the inhalation of the powdered substances used in the preparation of hard metals has been attributed to cobalt (22, 24).

Some metal dusts produce striking changes on X-ray but in fact they do not affect the lung tissues. That happens in the case of exposure to barium dust and to a lesser extent to iron, tin (25) and titanium dusts (26). On the other hand, some dusts are complex mixtures often containing silica. So, the disease produced is more likely to be due to the free silica content of the dust than to the dust itself. This relates, probably, to Shaver's disease (27) occurring in men exposed to the fumes arising in the process of making the abrasive corundum Al_2O_3, by heating bauxite, an ore containing approximately 80 percent Al_2O_3 and up to 7 percent silica. Although, as stated by *Hunter* (12), Riddell believed that the disease was aluminosis and not silicosis, it has been shown that ultramicroscopic sized silica particles such as are present up to an amount of 30–40 percent during the above mentioned processes are capable of producing diffuse non-nodular fibrosis (28) similar to that in Shaver's disease (26).

In the mining of certain coal with low silica content (less than 2 %) a pneumoconiosis with distinctive features may result. Simple coal workers' pneumoconiosis is considered to be due to the inhalation of coal dust. But in cases of the so-called progressive massive fibrosis an additional factor, i. e., tuberculosis of the lung seems to be involved.

Exposure to cotton dust may cause a disease which is called byssinosis. In its advanced stage byssinosis resembles to chronic bronchitis and emphysema. A history of tightness of the chest on Mondays, common in bysinosis but very rarely found in chronic bronchitis of other origin (29), usually helps to distinguish byssinosis from a non-occupational bronchitis and emphysema. During the course of exposure to cotton dust, a characteristic fall in ventilatory capacity and expiratory flow rate respectively has been noted (30, 31, 32).

Bagassosis is an acute respiratory illness due to the inhalation of dust of baggase, which are dried cellulose fibres of sugar cane after the sugar has been extracted. The disease was first recognized in Louisiana in 1937 (33). Since then bagassosis has occurred sporadically, but in 1959 a large outbreak of the disease occurred at a paper mill in Arecibo, Puerto Rico (34).

Although bagassosis has been considered as an acute disease which subsides gradually, leaving no residual lesions, evidence of sequelae with pulmonary fibrosis and functional impairment of the lungs has been demonstrated in some cases (35).

In the cases of exposure to some organic dusts it has to be taken into consideration that the changes which may occur may also result from the small amount of the free silica content of the dust such as tobacco, grain and seeds.

Pathologic Physiology of Pneumoconiosis

Pathologic physiology of pneumoconiosis has been studied intensively. This particularly relates to the mechanism of the action of silica particles.

Findeisen (36) first predicted that the particle size is a critical factor in determining to what extent dust will penetrate into the respiratory tract.

Larger particles will be retained in the nose. Experimental study made by *Pattle* (37) has shown that the retention in the nose is nearly complete for particles 9 microns in diameter. The retention for the smaller particles varies with particle size and flow rate and may be probably explained by an impaction mechanism. Among the mechanisms of absorption and filtration in the upper part of the bronchial apparatus the role of ciliary activity has to be pointed out. It is well known that the lining epithelium of the respiratory passages, with the exception of the pulmonary alveoli and the terminal bronchioles, is provided with cilia and goblet cells. The ciliary movement causes continuous flow on the layer of mucous above the cilia. If, for any reason, cilia cease to beat the transportation of mucous also stops (37).

Particles 1 to 3 microns, as well as the submicronic particles reach the smallest bronchioles and the alveoli. It is important to point out that the alveolar deposition of submicronic particles is very high: 90 percent and more of the inhaled particles. This has been shown for coal dust and also for other particles (38, 39).

Dust deposition occurs in the peripheral alveoli. From the peripheral alveoli particles are either eliminated by the alveolar clearance mechanism or conveyed to the sites of collection around respiratory air passages. In this process phagocytes seem to be involved (40). *Gross* (41) thinks that the alveolar macrophages engulf most but not necessarily all of the dust particles which have impinged upon the alveolar membrane. According to the same author (42), transport of dust particles depends mainly on respiratory excursions of alveolar walls, while the role of macrophages is passive.

Lung clearance of the dust originally deposited in the alveoli is a slow and continuous process. Inhaled dust is eliminated via the bronchi and the lymph passages. The elimination curves of various insoluble dusts are fundamentally similar (43).

The silica particles which have remained in the alveoli stimulate the proliferation of alveolar cells to form nodular aggregates or small nodules. The tissue fluid transports some of the silica particles into the interstices of the looser periadventitial tissue of vessels and bronchial passages. There, histiocytes also proliferate to form cellular aggregates

or nodules (42). According to *Schiller* (44) only free, not phagocytosed particles can enter the interstitium of the lung. The particles deposited in the lymph nodes also cause proliferation of histiocytes.

The cellular aggregates or nodules quickly develop a characteristic concentric layering of reticulin fibres, and later a process of collagenization and hyalinization follows.

Theories proposed for explaining the fibrotic reaction have included mechanical irritation due to silicic acid, then action based on piezoelectric properties of the silica crystals, and recently an immunochemical mechanism (45, 46). Although the mechanism of the action of silica particles has not been solved yet, the chemical theory has been usually considered as the most plausible.

As far as the action of inhaled asbestos fibres is considered, *Davies* (47) believes that it is based mainly on their mechanical effect rather than chemical.

In coal workers' pneumoconiosis in the simple state, the dust is found throughout the lungs in the form of black foci. Reticulin fibers develop in the foci of dust (48). Fibrosis may not proceed beyond this stage or there may be the development of collagen which however does not develop to the same extent as in classical silicosis nor does it have the concentric disposition, but runs irregularly or radially (12). In and around the coal foci the air spaces become dilated, giving a characteristic appearance described as focal emphysema. (49).

As mentioned before, superimposed massive fibrosis in this kind of pneumoconiosis seems to be due to the combined action of tuberculosis and dust (50, 51). There is some experimental evidence (52) to suggest that changes in the form of progressive massive fibrosis of the lung may also be produced in animals exposed to some other »inert« dusts such as kaolin and silicon carbide, if a tuberculous infection is present.

Pathologic physiology of some other diseases of the lung caused by the inhalation of dust, such as cotton dust and sugar cane dust needs still to be better explained. In the case of the exposure to cotton dust the narrowing of the airways has been attributed to some histamine–releasing substances contained in the cotton (53) but the exact nature of the disease is still only partly understood. The etiological mechanism of bagassosis remains also unkown. When the disease was first reported (54), it was believed that fungi were the causative agents, but this could not be proven later.

Beside fibrosis of the lung and similar chronic changes some metals may exert an acute action on the lung with cellular death and necrosis (55). These conditions are described as chemical pneumonitis (56) and may occur after an extensive exposure to fumes of manganese, cadmium, beryllium, vanadium (57), and, according to some experimental evidence, probably also to those of osmium (58).

Respiratory Diseases Caused by Gases, Vapours and Mist

Among gases which can be found in the working environment, from the point of view of respiratory diseases, the most harmful are those belonging to the group of irritants. Irritant gases are chlorine, ammonia, sulfur dioxide, hydrogen chloride, hydrogen fluoride, ozone, nitrogen dioxide as well as its polyform: N_2O_4, phosgene, nickel and iron carbonyl. Some other gases with a systemic toxic effect, according to the dose inhaled as well as to the rate of exposure, may also produce to a great extent irritation of the respiratory organs. That relates, for example, to hydrogen sulfide which is primarily a chemical asphyxiant and to methyl bromide which damages the nervous system and the kidneys.

Substances which appear in the form of vapors and mist are extremely numerous. Among such substances of outstanding importance in industry are those used as solvents. These compounds, belonging mostly to the aromatic or aliphatic hydrocarbons, can have a serious toxic effect on different organs. But as they enter the body principally by inhalation, they may also act on the tissues of primary contact and produce a local effect on the air passages.

Acids, such as sulfuric acid, chromic acid, etc., may also play an important role as irritating material in the working environment.

Fuel oil, gasoline and petroleum products affect the respiratory organs when oxidized to acids, aldehydes, ketones and other substances. Such an oxidation occurs in the general atmosphere. In the presence of nitrogen dioxide unsaturated hydrocarbons (59) but also a number of aromatic hydrocarbons (60) irradiated by sunlight form ozone which then further promotes their oxidation. During the photochemical reactions which occur in the polluted air the production of organic free radicals may also result. They may be produced by the photolysis of organic molecules, by the reactions of atoms or other radicals with stable molecules, or possibly by the reaction of ozone with unsaturated hydrocarbons (61). The free radicals are capable of removing an H atom from almost any organic substance upon contact, and thus, they are expected to be irritating and harmful to living tissue (62). Although the conditions in the working environment differ from those in the general atmosphere, such reactions might occur, at least to some extent, also in industry.

The formation of aerosols is also important in the working environment. This includes both the so-called neutral particles, and condensation nuclei which are made up of hygroscopic substances. Aerosols of different particle size composition will reach different parts of the respiratory system (63).

Aerosol forming particulates may concentrate material on their surface and in their interstices, thus producing – when inhaled – a locally high concentration of an otherwise very dilute substance. Aqueous droplets may dissolve such gases as sulfur dioxide and permit their hydration to sulfurous acid, or if oxidized to sulfuric acid (64).

Physiological Action of Irritating Chemicals

Irritants, when inhaled cause an immediate or delayed effect on the respiratory tract. This depends on the amount of the material inhaled and the length of exposure, as well as of certain characteristics of individual substances. Some of the irritant gases, such as ammonia are very soluble in water and in the body fluids and for this reason are absorbed from the inspired air by contact with the first moist tissue which they reach in the respiratory passages. As a consequence the upper respiratory tract bears the brunt of the action. The lower part of the bronchial apparatus and the lungs are relatively little affected since the concentration of the irritant which reaches them is greatly reduced by absorption in the upper passages. Thus, the solubility might be considered as the factor which determines the concentration of gases in various parts of the airways. In connection with that it is possible to accept Hunter's conclusion that the irritant gases, as a general rule, are dangerous inversely as their solubility (12).

On the other hand the effect of inhaled irritants is not only the function of their solubility. It depends also on their chemical properties as well as on the chemical and physical reactions with the tissue in contact.

In the case of the exposure to high concentrations of some soluble gases, in spite of their solubility, a considerable amount of gas will enter the small bronchioles, alveolar ducts and alveoli. This might give rise to pulmonary edema. However, the occurrence of edema in such a situation depends also upon the other characteristics of the gas. Thus the inhalation of chlorine, even in high concentrations, seldom causes edema (12), while edema of the lung seems to be relatively frequent if concentrated ammonia has been inhaled. Individual susceptibility, based probably mainly on pre-existing conditions of the respiratory organs, seems to play also an important role.

Gases such as nitrogen dioxide, ozone, phosgene, iron and nickel carbonyl usually have a delayed effect. The initial irritative symptoms may pass unnoticed especially if the concentration of the gas inhaled is low. Acute pulmonary edema can develop after a latent interval which varies from two to twenty–four hours. If very high concentrations of some of these gases are inhaled, symptoms may occur almost instantly after the exposure. In such a case clinical picture of the intoxication is usually

more complex, because some other functions, as the regulation of blood pressure, central regulation of respiration, etc., may be affected. Thus, for example, the inhalation of very high concentrations of oxides of nitrogen can cause the so-called shock-type poisoning, characterized by severe asphyxia, convulsions and respiratory failure, which is almost always fatal (65).

The action of irritating chemicals upon the respiratory organs seems to be based on different mechanisms. These mechanisms include reduction, oxidation, formation of new compounds in contact with the tissue fluids and various biochemical reactions. Thus, ammonia causes an immediate corrosive effect on mucous membranes. Chlorine probably takes off hydrogen from the water on mucous membrane and releases oxygen in a nascent state. This effect seems to be combined with the effect of hydrochloric acid formed on the tissues. The action of phosgene was also considered to be due to hydrochloric acid formed by the hydrolysis of phosgene in the presence of moisture in the airways. *Moeschlin* (66) believes that the effect of phosgene is based on a chemical reaction of a released O = C group which reacts with cellular amino-acids and enzymes. For ozone and nitrogen dioxide it has been presumed that they act as free radicals (in the capacity of oxidizing agent) (67). According to this view it is believed that ozone and nitrogen dioxide attack –SH groups important to the integrity of cell surfaces and membrane permeability. It is also possible that –SH groups of some other critical enzyme systems are involved. Immuno-chemical mechanisms have also been suggested (68) to explain – at least partly – the action of the same irritants.

There are some experimental works which led to the hypothesis that pulmonary edema caused by the action of irritant gases might be neurogenic in origin.

Meyers (69) showed in animal experiments that after an exposure to 50 ppm of phosgene for fifteen minutes, guinea pigs treated with smooth muscle relaxants (Paveril) could be kept alive twice as long as the controls (animals exposed to phosgene but not treated).

Studying the pathologic physiology of pulmonary edema and emphysema caused by irritant gases, very recently the importance of the biologic surfactants has been pointed out (70). This is naturally occurring surface – active material which lines the alveolus and controls the amount of liquid in it. When toxic agents alter pulmonary mechanics or fluid dynamics of the lung, these mechanisms, which serve to facilitate the passage of fluid laden with respiratory gases between the lumina of the capillaries and the lumina of the alveoli, may be impaired (71).

In another study on pulmonary surface phenomena, *Hackney et al* (72) found a highly significant decrease in surfactant activity in O_2 poisoned rabbits. According to these authors, this might be explained by a decrease in production of surfactant and / or by production of an

inhibitor. Using the U. V. fluorescent microscopy technique to examine frozen lung sections of a series of normal and oxygen poisoned animals, the investigators were able to show marked reduction in the fluorescent lining of air spaces in the O_2 poisoned lungs compared with brightly fluorescent linings of air spaces in the normal lungs.

The changes in the air passages produced by irritating chemicals vary from bronchoconstriction and inflammatory reaction of mucous membrane with mucosal edema, congestion and exudate to marked alteration of the bronchial epithelium and alveoli with desquamation, squamous metaplasia, obliteration and obstruction. The nature of these changes, their location and intensity depend on factors already mentioned such as the physiochemical characteristics of inhaled irritants, their concentration as well as on the duration of exposure and the susceptibility of the organisms exposed.

Inhalations of various chemical substances, as well as the inhalation of pollens and different dusts, may produce in persons with an asthmatic diathesis responses of allergic nature in the form of bronchial asthma or spastic bronchitis. These conditions are often initiated by bronchial irritation or infection.

Occupational Cancer of the Lung

Both dusts or fumes and vapours or gases may be involved in the causation of occupational cancer of the lung.

Apart from the already mentioned cancer of the lung seen in Schneeberg miners, lung cancer has been found in connection with other exposures, as well. The industries which are primarily concerned in causing or possibly causing cases of cancer of the lung are the nickel industry, the chromate industry, the asbestos industry and the manufacture of sheep dip (12). In a review on occupational lung cancer *Doll* (73) distinguishes among various occupational factors those which represent established risks, suspected risks and possible risks. According to Doll's classification the established risks are radioactivity, nickel, chromates, asbestos and gas production (exposure to fumes and dust from coal tar).

Studies which were carried out had shown that in mines of Schneeberg and Jáchymov about 40–75 percent of all men who were continuously employed died of lung cancer (74). The Schneeberg mines have been worked at first for copper and iron, later for silver and some other minerals, and then for bismuth, arsenic and cobalt. The air in the mines has been shown to be radioactive. Jáchymov mines are uranium mines. Although the exposure – as described – was combined, great suspicion rests upon radioactivity (12, 73).

In 1959 *Doll* (73) pointed out that until that year no other group of workers had been shown to have suffered a high incidence of bronchial tumors as a result of exposure to high doses of radioactivity. Since then there has been some indication that the hazard exists in some other uranium mines. A study in the mines in the Colorado Plateau in U. S. A. (75), which have had atmospheric radioactivity comparable to that in Schneeberg (76), showed that of 907 white miners with 3 years or more of uranium mining experience 5 died from lung cancer during a period when only 1.1 deaths was expected.

Cancer of the lung due to nickel was reported for the first time in 1939 (77). It has been found mostly in the production of nickel carbonyl (78). As cobalt is almost everywhere present with nickel it also might play a role in the occurrence of cancer due to this type of occupational exposure.

A high incidence of lung cancer among the workers exposed to chromates was reported from Germany (79) and U. S. A. (80, 81). It seems that the responsible agent is trivalent chromium in the form of an acid soluble – water insoluble compound (82).

The association of the lung cancer with asbestosis has been suspected since 1935 when *Lynch and Smith* in U. S. A. (83) described the first case. Since then the possible relationship of asbestosis and carcinoma of the lung has been reported several times (84–89).

In 1960 *Wagner et al* (90) described 33 cases of diffuse pleural mesothelioma in men and women who all but one had a probable exposure to crocidolite asbestos in South Africa.

As far as the production of gas from coal is concerned, there are some publications (91, 92) which support the view of the role of such an exposure in the occurrence of lung cancer. It is known that the combustion and distillation products of coal are rich with carcinogens (73).

In 1948, *Hill and Faning* (93) suggested the arsenical sheep dip manufacture as a possible source of lung cancer. There are also some other processes where the exposure to inorganic arsenic was suspected as carcinogenous (94).

A high incidence of lung cancer was also reported among arsenic-poisoned vineyardists in Moselle (95, 96). *Buechley* (97) believes that arsenic is even responsible for the occurrence of lung cancer in workes in Schneeberg mines and arsenic-high smelters. According to the same author, lung cancer associated with cigarette smoking could be due to arsenic–sprayed tobaccos used in cigarettes!

It has also been suggested that there might be some causative relationship between hot metal occupations and lung cancer (98, 99). This, as well as some other suspected risks such as commercial cooking, construction and maintenance painting, etc. (6), have not been proved yet.

Although as stated by *Breslow* (6) the number of persons in the U. S. A. known to have occupational exposure which might give rise to lung cancer is small, it seems unlikely that all the existing risks have been discovered. In addition it has to be taken into consideration that new materials and new processes are being constantly introduced in industry and, as pointed out by *Doll* (73) there is no reason to suppose that they will necessarily all be free from risk.

In spite of our knowledge about the potential and possible occupational risks in the occurrence of cancer, the real nature of the disease still remains unsolved. Occupational cancer of the lung is in most instances bronchial in origin. The exposure to nickel and to chromates may cause nasal cancer, as well. This site specificity has not been yet satisfactorily explained. Perhaps there are some differences in susceptibility of the tissues or in the amounts of the carcinogen deposited (73).

Individual differences in general susceptibility to cancer among persons with the same exposure have also remained unexplained, because the basic patho-physiology of the disease is not clear.

Some recent experimental work – using methylcholanthrene and ionizing radiation – suggested that the induction of cancer of the lung is exquisitely dose dependent (100).

From the experience it is known that lung cancer due to occupational exposure occurs after a latent period between 15 to 30 years.

Effects of Climate

Temperature, humidity, velocity of air, and irradiation of heat in the working environment – when deviated from the normal range – may affect either directly or indirectly the function of the respiratory organs. Although this has been generally accepted, the effect of atmospheric characteristics upon physiological and disease processes in the respiratory organs has been only partly resolved. Some experimental evidence (101) and epidemiological studies (102, 103) support the view that there is certain association between the temperature and humidity and respiratory diseases. One mechanism for the effect of temperature may be through the fluctuation of the blood supply of the respiratory mucosa. In a cold atmosphere the superficial blood vessels of the skin and of the oral and nasal mucosa become constricted. Thus depleted of their blood supply, these tissues are less able to resist invasion by microbial agents which enter through the respiratory route (104). Low humidity may exert a drying action on the mucosa of the upper respiratory tract and lower its vitality and resistance to infection (104).

In the working evironment the role of exposure to changes in temperature outdoor and indoor, is obviously also important.

Variations of the temperature and humidity may influence the effect of irritants upon the respiratory tract. Thus, in a physiological study of irritant aspects of atmospheric pollution, *Roth and Swenson* (105) observed that the increased humidity raised the threshold for irritation due to formaldehyde.

Temperature and humidity in particular may affect the virulence of microorganisms in the environment thus influencing not only the host but the disease agent, as well.

Occupational Diseases of the Lung Caused by Living Agents

Acute and chronic lung diseases can result from the infection by different living agents which may specifically contaminate the working environment. These agents belong mainly to the group of causes of zoonotic diseases and to the fungi. Examples are: virus of psittacosis pneumonia, rickettsia causing Q fever pneumonia; and among fungi: Coccidioides immitis, Histoplasma capsulatum and Aspergillus fumigatus.

The occurrence of diseases caused by these agents depends also very much on residence. This is particularly true for coccidioidomycosis and histoplasmosis which occur among farmers but also among the members of their families, as well as among other people living or even visiting the contaminated areas.

There is another occupational lung disease – belonging probably to this group of diseases – which is known as farmer's lung. This disease was described for the first time in 1933 by *Campbell* (106). Farmer's lung seems to be due to the inhalation of dust from moldy hay, silage or grain. The disease is characterized by a prompt response to exposure with acute symptoms such as dyspnea, cough, sputum, fever, nausea, vomiting, etc. X-ray of the lung reveals diffuse infiltrations. The resolution is rapid in the acute stages, but chronic form of the disease with fibrosis – usually of a granulomatous type – may also occur. The etiology of farmer's lung has not been definitely solved, as yet. Some authors (107–109) are inclined to believe that the disease is a true pulmonary mycosis. More recently other mechanisms, such as pulmonary tissue reaction to dust particles (110) or a blockage of the terminal bronchioles by the fungal spores or their breakdown products (111) have been suggested. There is also a view that a hypersensitivity reaction to the molds or their products of disintegration is probably the basic pathophysiological mechanism of farmer's lung (112, 113).

Farmer's lung must be distinguished from silo-filler's disease which is caused by nitrogen dioxide gas produced during the process of fermentation of the stored crops (114, 115).

III. CHRONIC BRONCHITIS IN INDUSTRY

In spite of the fact that many questions concerning the relationship of respiratory diseases to environmental factors have been answered, there are still problems which have not been solved satisfactorily. If we exclude the question of the real nature of occupational lung cancer, these problems are mainly connected with the so-called industrial bronchitis, which is in fact, chronic bronchitis of occupational origin. Although, as described earlier, the mechanism of the acute effect of irritant gases upon the respiratory organs is known, as well as the clinical picture of the disease, it is not clear enough what happens in the case of chronic exposure to the low concentrations to such irritants. Also to be resolved is the question of whether a single acute intensive exposure can result in chronic bronchitis with obstructive ventilatory disease (116). Similar questions arise concerning the relationship of chronic bronchitis to prolonged exposure to »inert« dusts.

Although it is generally assumed that emphysema or pulmonary parenchymal infection with or without fibrosis may result as a complication of the chronic inflammation of the mucosa of the air passages, the relationship between those conditions needs, perhaps, more clarification. Criteria for the diagnosis of chronic bronchitis also have to be better defined.

Reports concerning these problems, though numerous, are sometimes contradictory or controversial. Differences in results and opinion are, at least partly, due to different conditions and sometimes different methods and criteria under which experimental, clinical and epidemiological studies were carried out. However, the majority of authors agree on a possibility of the chronic effect of the prolonged exposure to various irritants as well as on possible sequelae after single intensive exposures.

Long-Term Effects of Irritant Gases Exposure

The greatest amount of published work and observations relate to sulfur dioxide and nitrogen dioxide.

In 1932, *Kehoe et al.* (117) found a significantly higher incidence of nasopharingitis, of alteration in sense of taste, and of shortness of breath on exertion in a group of subjects with a prolonged industrial exposure to SO_2 than in the controls. As the result of an investigation carried out among workers exposed to sulfur dioxide gases in an electric foundry, *Humperdinck* (118) expressed the opinion that the symptoms observed (chronic catarrh of upper respiratory tract and bronchi, cough, rales, frequently emphysema, etc.) indicate the possibility of further aggravation of the conditions; an increasing incidence of emphy-

sema or bronchiectasis could be expected. Similar experience with prolonged exposure to SO_2 is reported by other authors (119, 120).

Studying the effect of exposure for periods up to 60 minutes, to sulfuric acid mist, sulfur dioxide and various aldehydes in a large group of men, *Sim and Pattle* (121) found an increase in airway resistance and development of rales with the exposure to sulfur dioxide and sulfuric acid, as well as long-lasting bronchitic symptoms in some subjects exposed to sulfuric acid mist. The same authors expressed the view that the repeated exposure to SO_2 may increase the sensitivity to this irritant rather than cause a tolerance.

Sequelae after the acute illness due to exposure to high concentrations of SO_2 were observed several times. The conditions were described as persistent severe dyspnea due to pulmonary emphysema, bronchiectasis and bronchitis (122), increased linear densities in the X-ray, slight emphysema and bronchiectasis (123), bronchiectasis (124), bronchial stenosis (125), and late dyspneic effects occurring several months after the accidental exposure (126).

In 1937, *Dowling* (127) described a case of asthma following prolonged exposure to sulfur dioxide, but the patient involved had evidence of an allergic constitution.

Some authors (128) think, however, that there is no good evidence that either chronic exposure below certain concentrations of SO_2 (< 5 ppm) or occasional exposure to higher concentrations, so long as these are not over-powering, have any ill effect upon healthy men. *Anderson* (129) had similar results in a group of workers exposed to daily concentration of sulfur dioxide between 0 and 25 ppm for periods of time varying from 1 to 19 years. In a radiographic study of the influence of industrial gases (Cl_2, SO_2, SO_3, and some others) on the human lung, *Evans* (130) found no increase in pulmonary fibrosis in any group of exposed workers. A higher death rate from pneumonia and other pulmonary infections was not found in the exposed groups than that recorded for the other plant employees.

Some experimental studies (131) also did not show enough evidence that the inhalation of SO_3 in low concentrations produced permanent damage to experimental animals.

Concerning nitrogen dioxide there are also a number of reports on its chronic effect after a prolonged exposure. In 1937, *Vidgortschik et al.* (132) found statistically proved higher prevalence of emphysema of the lung and chronic bronchitis in a group of 127 men who had worked 3–5 years in H_2SO_4 plants and as etchers in printing shops. Although the authors pointed out that in the majority of cases NO_2 was the only injurious gas detectable, the influence of some other contaminants in the working environment in this case cannot be completely excluded.

There are some publications (133) in which were reported a high incidence of asthma and asthmatic bronchitis as the result of exposure to nitrogen oxides and certain other irritants.

Chronic effect after single exposures to high concentrations of nitrogen dioxide and other irritant gases was also described several times. *Brille et al.* (134) found, in two men severely intoxicated and in 5 men moderately intoxicated by nitrogen oxides, an obstructive respiratory disease with emphysema and some fibrosis of the lung. *Becklake et al.* (135) had similar results from a follow-up of a group of seven patients who had recovered from an episode of acute pulmonary edema following the exposure to nitrous fumes. In 1961, *Bellini et al.* (136) published the results of a follow-up study of workers previously treated because of intoxication caused by irritant gases. They found marked changes in the form of chronic bronchitis, emphysema and fibrosis of the lung and diminished respiratory function, especially in those who had been exposed to mustard gas and to nitrogen oxides. The symptoms observed were milder in those exposed to sulfur dioxide and chlorine. *DuBois* (137) found a lower airway obstruction, demonstrated by respiratory function tests, even when there was no other objective evidence of chronic respiratory disease, in a group of workers previously exposed to various irritant substances. Sequelae after acute intoxications with nitrogen oxides sometimes have been described in the form of bronchiolitis obliterans (138–141). This condition has been considered as a clinical and pathologic entity which may either follow the inhalation of irritant substances or may occur as a complication of pulmonary infection. Sometimes the etiology cannot be explained.

Bronchiolitis obliterans is characterized by marked, progressive dyspnea, variable cough, expectoration of blood stained sputum and cyanosis. The X-ray picture of the lung may simulate miliary tuberculosis. Pathologically, the lumen of the bronchioles may be partially or even entirely obliterated by granulation or young connective tissue (141).

Although it has been generally assumed that the alveolar involvement is secondary to the bronchiolar involvement, *Groll* (142) had demonstrated the role of alveoli in the organizing processes.

There are some reports which indicate the possibility of a prolonged effect after exposure to ozone (143–147).

Some experimental studies also support the view of the chronic effect of exposure to some irritant gases. Thus, *Kleinerman and Wright* (148) observed in rabbits and guinea pigs exposed to nitrogen dioxide in various exposure schedules (from 15 to 100 ppm) for periods up to twenty-two months, histologic evidence of inflammatory reaction, and epithelial hyperplasia in the region of the respiratory bronchiole and proximal alveolar duct. A form of respiratory bronchiole ectasia resembling superficially a centri-lobular type of human emphysema has been observed in a number of guinea pigs after 15 and 18 months of

exposure to nitrogen dioxide. Studying the chronic ozone exposure, *Stokinger et al.* (149) found chronic injury to the lungs, which was principally manifested in the terminal airways by chronic bronchiolitis and bronchiolar wall fibrosis, in a breed of mice exposed to one part per million of ozone for 6 hours daily, five days a week, for 268 exposures.

On the other hand *Rigner and Swenson* (150) in Sweden in a follow-up study of 27 men who suffered acute intoxications with nitrogen oxides, could not find any clinically important symptoms or signs of chronic respiratory conditions.

From the point of view of late consequences of exposure to high concentrations of irritant gases, the groups of persons poisoned by gases in World War I are very interesting. In the opinion of *Brille et al.* (134) the follow-up of these persons have shown the evidence of chronic effects after such an acute intoxication.

Long Term Effects of Inert Dust Exposure

Exposure to »inert« dust has also drawn a lot of attention. *DuBois and Dautrebande* (151) measured the physiological effect in men of inhaling chemically inert dust particles. *Cummins and Rayner* (152) studied the health of employees engaged in gas production exposed to coke or coal dust and some other potential hazards. *Fletcher* (153) studied the disability and mortality from chronic bronchitis in relation to dust exposure; *Becklake et al.* (154) studied the nature and etiology of respiratory disability in goldminers free of radiological silicosis. Experience from some of these studies (152, 153) showed that persons exposed over a long period of time to the inhalation of dusts may get notable respiratory impairment. Some authors (154) suggest the combined action of other occupational factors such as inhalation of fumes and rapid changes in temperature, humidity and altitude.

A survey of chronic bronchitis (155) was carried out in Newcastle-upon-Tyne - an industrial city in England. The results obtained from this survey suggest that infection is the dominating factor in chronic bronchitis, but this is maintained and aggravated by other environmental agents. Such environmental factors seem to be cigarette smoking, atmospheric pollution and dust, draughts and extremes of temperature at work. The authors expressed the view that in a considerable proportion of cases of chronic bronchitis a constitutional predisposition to the disease exists, which may be hereditary.

To emphasize the possibility of a combined role of this last factor and the environmental exposure in the causation of chronic bronchitis and emphysema, it is perhaps worthy to mention at this point the Caplan's syndrome (156) which illustrates nicely the collaboration of genetic and environmental factors in disease (157).

Caplan's syndrome is a special type of pneumoconiosis, with multiple globular masses 5 mm to 5 cm in diameter which evolve rapidly, that occurs in persons exposed to silica or coal dust, but who have a »rheumatoid« genetic predisposition.

In a study of the clinical pattern of chronic bronchitis *Oswald et al.* (158) states that almost any dust seems to be a potential irritant. They found dust to be one of the principle factors at work which the patients considered as aggravating their bronchitis. The role of dusts as an occupational factor in the incidence of fronchitis was pointed out by *Gregory* (159). This author arrived at the conclusion that among dust, temperature and humidity and gases and fumes as principal occupational factors, dust was undoubtedly the most important in the incidence of bronchitis.

There is a recent work written by *Carstens* (160) that is also very interesting from the point of view of »non-specific« effect of dust. It relates to the observations obtained among the coal miners in Germany. From the clinical and epidemiological observations – using appropriate statistical methods – it has been concluded that pneumoconiotic fibrosis represents only a small portion of the lung disease processes, an impairment of pulmonary function due to bronchospasm with or even without clinical signs of chronic bronchitis and emphysema being of much greater importance. Some previous studies made by other outhors (161, 162) also confirmed this view.

In a hospital study of thirty soft-coal miners with respiratory disease, *Stoekle et al.* (163) arrived at the conclusion that disabling pulmonary disease of soft-coal miners is not only a pneumoconiosis. It is – in these authors' opinion – rather a disease of the respiratory tract which may be due to inhalation of harmful chemicals and dust during work, interacting with other individually determined factors, such as physiological aging, smoking habits, and infection.

It is important to point out that there is also experimental evidence (44) which supports these epidemiological and clinical observations. Dogs exposed over a period of three years to 50 mg of bituminous coal per cubic metre of air developed a remarkable hypertrophy of the bronchial mucosa and its goblet cells. After a six year's stay underground epithelial hypertrophy as well as hypertrophy and hyperplasia of the goblet cells have ceased for an atrophic epithelium only 14 microns high that in some place was denuded of cilia.

On the other hand it is interesting to mention *Gough's* (22) opinion that, whereas dust or other environmental conditions in mining may contribute to the development of bronchitis, the latter will have a negative role in respect to pneumoconiosis. Gough thinks that a person with bronchitis and increased sputum is less liable to accumulate dusts in the lungs than a person whose lungs are otherwise healthy.

As already stated, from the literature citations quoted above one could conclude that respiratory impairment with irreversible changes of pulmonary function are likely to be found as long-term effects both of irritant gases and of the inert dusts exposures.

However, the whole problem doesn't seem to be definitely solved, yet. First of all the negative results – although reported by a minority of authors – have to be taken into account. On the other hand, positive findings, at least to some extent, may be criticized especially because of inadequate controls and inappropriate statistical confirmation of the results obtained. One example from Great Britain illustrates very well how careful one must be before making conclusions, even when the facts seem to be obvious: National Statistics for England and Wales showed that miners and foundry workers had an excessive mortality and morbidity from bronchitis compared with other workers, but *Higgings et al.* (164) noted that wives of these workers also had a high incidence of bronchitis. In other words simple asumption that the difference in mortality and morbidity from bronchitis among those workers is related to occupation would be inaccurate.

Although the question of whether occupation contributes to the incidence of chronic bronchitis and emphysema might be still under suspicion, remains the fact that the role of irritants as well as that of the inert dusts has to be seriously considered in the evaluation of – most likely – complex causation of such diseases.

Diagnosis of Industrial Bronchitis

The diagnosis of so-called industrial bronchitis is based on the history of adequate exposure, symptoms after exposure and exclusion of some previous pulmonary conditions. *Higgings* (165) thinks that neither X-rays of the chest nor lung function tests help much as diagnostic methods for such a disease. In his opinion the diagnosis must be made clinically and the individual symptoms and various combinations of them indicating different levels of bronchitis can then be used to evaluate possible etiological factors. Discussing the question of diagnosis of industrial bronchitis *DuBois* (137) on the contrary stresses the necessity of the pulmonary function testing. Among other things, this author says: »Whereas the chinical and radiological examinations are essential in order to rule out other diseases which might have produced equivalent changes of pulmonary function, the X-rays of the lungs do not reveal the basic cause of dyspnea on exertion because the lower airways are not visible on an ordinary X-ray. Hence, the burden of objectively demonstrating the alterations of pulmonary function in cases of suspected industrial bronchitis rest on the pulmonary function tests.«

Although for the early stage of the disease it is probably possible to say that »chronic bronchitis implies no more than chronic inflammation of the bronchi,« as the disease progresses the bronchiolar and alveolar disruption becomes widespread and far surpasses the bronchial change in functional significance (166). For this reason DuBois' view of the diagnostic value of functional testing seems to be reasonable, because this is the best way to make the diagnosis of the disease more objective. On the other hand it must be taken into consideration that in certain cases of occupational exposure only the lower part of the bronchial apparatus and alveoli are involved from the beginning in the pathological processes. In such cases some classical symptoms of chronic bronchitis are usually absent but the impairment of pulmonary function might be very evident. However, it is obvious that there is no specificity in the results obtained by the lung function tests.

In recent years in Great Britain attempts have been made to standardize the diagnostic criteria and grade the symptoms of chronic bronchitis and related respiratory conditions (167–169). As a result of this work a standard form of symptomatic questionnaire* has been created. It has been shown that by using such a questionnaire observers' variation as well as the errors in diagnosis may be kept small (170, 171).

In the same time a great deal of work has been done to standardize the pulmonary function tests and improve the methods. According to the nature of changes which can be expected, the emphasis of the testing of pulmonary functions has been on the mechanics of breathing and to a lesser extent on pulmonary ventilation, distribution of inspired gas and diffusion capacity of the lungs. The detection of the obstruction of the airways has attracted particularly a lot of attention.

In 1951, *Gaensler* (172) introduced a device for simultaneously measuring the vital capacity and the timed volumes. *Gaensler* emphasized the ratio of the particular forced expiratory volume to the total vital capacity as a very valuable index of ventilatory obstruction (173). It has been shown later that either the $FEV_{0\cdot5}$ or the $FEV_{1\cdot0}$ expressed in liters and compared to predicted normal values are more sensitive in detecting early impairment than such ratios (174). Suitable prediction formulae for both the half second and one second forced expiratory volumes have been provided (175, 176). Recently, waterless portable instruments have been introduced which record the forced expiratory spirogram (177).

Devices for single-breath measurements which indicate a type of peak flow have also been developed. These are Puffmeter (178) ind Wright peak flow meter (179).

* Questionnaires and instructions can be obtained from Dr. C. M. Fletcher, M. D., F. R. C. P., Department of Medicine, Postgraduate Medical School of London, Ducane Road, London, W. 12.

Although there is generally a good correlation among the values obtained by using these different measurements, it is still uncertain which portion of the forced expiratory spirogram provides the earliest indication of abnormality. For this reason *Kerry* (174) thinks that it seems advisable for adequate screening that a recorded forced expiratory spirogram be obtained.

Screening surveys – using the above mentioned ventilatory testing techniques – were undertaken by several investigators. Examples are 1961 and 1962 study carried out by Queensbore Tuberculosis and Health Association, in New York City (180), a survey for chronic respiratory disease in an industrial city (181) as well as some other studies (182).

There seems to be considerable agreement that for such purposes a single breath spirometric test such as the forced expiratory volume (forced vital capacity) is easier to perform and more reproducible than conventional spirometric measurements of maximal voluntary ventilation (maximal breathing capacity).

The results of the validation examinations made during some of these studies (182) indicate that such simple tests of pulmonary function »are capable of detecting persons in whom the probability of having chronic respiratory disease is high«.

The introduction of body plethysmography has further facilitated the detection of ventilatory obstruction, but it is still difficult to use this method for surveys and screening purposes.

IV. STUDY OF OBSTRUCTIVE VENTILATORY DISEASES OF THE LUNG IN RELATION TO OCCUPATIONS

When approaching the problem of bronchitis in industry interaction of many other possible etiological factors must be carefully evaluted. This might include the smoking habits, generalized air pollution, fog frequency, temperature and humidity, economic class composition and its attributes such as housing, economic status, nutrition, adequacy of medical care, size of family, size of community, density of population per area, aging. The role of all these factors, as well as of the infection, must be resolved by appropriate epidemiological methods before judging about the influence of any individual noxious agent in the working environment upon the respiratory impairment.

There are various possibilities for studying respiratory disease from the point of view of occupational exposure.

One of the possibilities is to study a working population with a defined exposure. Such a study has some disadvantages. First of all in this case it is difficult to find the appropriate controls. If only a working population is studied those who have retired from work are excluded and

so far as ill-health is one reason for premature retirement, this exclusion may bias the group in favor of fitness (165). The same happens when those are excluded who have left their work for a similar reason and have moved to a different occupation. Conversely, as pointed out by *Holland* (183), if the occupation is physically light it may attract individuals with chronic bronchitis.

Another type of study might be to study the whole population or a random sample of it, in a geographically defined community. By using this method some disadvantages connected with a working population study can be overcome (165). On the other hand, general population study implies many problems from the point of view of organization, size of sampling, planning and statistical control. In such surveys valid conclusions will not be possible if the number of subjects in each subgroup is too small (178). These kinds of studies are also very costly. However, a certain number of such studies has to be done. This is not only in order to get more valuable information about the role of the environment in the occurrence of the obstructive ventilatory disease of the lung, but to allow comparison of the results obtained in different areas and countries at different time. The results obtained by a population study may also help as an orientation for selective studies of the individual enviromental factors such as different agents in occupations.

The relationship between diseases and some environmental factors, such as occupation, can also be studied by the use of data collected for other purposes. One of the possible sources are data of sickness absenteeism. The information obtained from this source may be very valuable although there are always problems of the selectivity of groups, of different diagnostic criteria, as well as of the lack of some information such as about previous occupational history.

During the last few years a standardized type of survey has been developed and used for comparative international studies of the rates of respiratory (and cardiovascular) conditions. Such a survey was conducted among telephone workers in Great Britain, Norway, the Eastern United States and recently in San Francisco and Los Angeles.

As far as respiratory conditions are concerned, this type of survey includes a standardized questionnaire and one or two simple tests of respiratory function. In addition volume and character of the sputum have been estimated. In some of these surveys X-ray of the chest was also made.

The questionnaire used was either – already mentioned – the British questionnaire or a slight modification of it. The basic idea in using such questionnaires has been »to define various levels or grades of severity of symptoms and to find means by which the same subjects are likely to be placed into the same, appropriate grades by different, independent observers.« The basis of the technique that has been recom-

mended is to use questions about the presence or absence of symptoms of various degrees of severity and to record the answer to each question as either »yes« or »no«. In the instructions for the use of questionnaire standard procedures are provided for probing as well as for dealing with indefinite answers. As a check of the accuracy of the interviewer, recording of interviews on tape have been used during the survey.

The questionnaire enables one to make an arbitrary definition of chronic bronchitis such as persistent cough with phlegm (for at least three consecutive months of the year), and at least one period of increased cough and phlegm lasting for three weeks or more in the last three years. Comparison of the prevalence of individual symptoms, as well as the use of other combination of symptoms for defining chronic bronchitis are also possible.

In addition to questions designed to elicit the presence of symptoms of respiratory conditions, the questionnaire also includes questions concerning age, smoking history and past illnesses as well as a short residential and occupational history.

The purpose of the use of ventilatory function tests is to get an index of lung function. As for the tests applied, the preference has usually been given to a single breath spirometric test such as the forced expiratory volume in a short time period (FEV_t). In spite of the fact that there is still some uncertainty as to the value to be measured from the tracing obtained, $FEV_{1\cdot0}$ (or $FEV_{.5}$) have been considered more sensitive in detecting early impairment than $FEV_{3\cdot0}$.

In some surveys maximal expiratory flow – using Puffmeter and Wright flow meter – were also measured.

Pulmonary function tests have been performed in a standing position and the same instructions on procedures are always given to the subject. To avoid bias in the interpretation and comparison of the lung function measurements, sitting height and sometimes the weight of the subjects, as an index of their physical size, is also recorded.

For the collection of sputum a standard size container has been provided. Each subject is instructed to bring or send the bottle the next day with phlegm from the chest or nose brought up during the first hour after getting up.

It has been shown that for the above mentioned procedures about 15 minutes per person are needed. A team of two trained interviewers and a technician, together with a receptionist, may examine a group of 60–70 persons a day.

The goal of such surveys has been to get information about the incidence of chronic bronchitis and to allow geographical comparison of the results obtained. Basically the same techniques can be used to study the effect of occupational exposure to respiratory impairment. This was used recently, for example, among the workers in a borax mine and borax refinery plant in California.

However, in order to elicit the possible role of occupations in the occurrence of respiratory impairment special attention has to be paid on history of exposure. Consequently, questions concerning occupational history have to be expanded. It must be pointed out that in addition to the present and previous occupations it is necessary in such cases to collect information about precise nature of exposures as well as their duration. The information about the actual exposure is useful because the exposure may vary much among men in the same occupation but in different plants. On the other hand questions concerning occupational history have to be designed in such a way that the information obtained can be coded and used for tabulation and analysis.*

Analysis of data obtained by different studies has been greatly faciliated by the development of computer techniques. In addition to conventional statistical methods, mathematical models might be used with the electronic computer to estimate the relative contribution of various factors such as age, smoking habit and occupation operating simultaneously.

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* A proposal for the part of the questionnaire concerning occupational history is presented in Appendix.

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Sadržaj

ULOGA PROFESIONALNE EKSPOZICIJE U OŠTEĆENJIMA RESPIRATORNIH ORGANA

Prikazan je problem profesionalnog oštećenja respiratornog trakta. Nakon kratkog uvoda, u kojem je istaknuta sve veća uloga kroničnih bolesti organa za disanje u pobolu stanovništva, iznijet je sistematski pregled profesionalnih štetnosti koje mogu uzrokovati bolesti pluća i bronha. Posebno su obrađene bolesti pluća izazvane udisanjem prašine i dimova, zatim para, magle i plinovitih spojeva. Opisana je patofiziologija tih oštećenja. Zatim je prikazan profesionalni rak pluća. Na kraju tog poglavlja raspravlja se ukratko o utjecaju klimatskih faktora u pojavi respiratornih bolesti, kao i o profesionalnim bolestima pluća uzrokovanim živim agensima.

Drugi dio prikaza odnosi se na problem kroničnog bronhitisa u industriji. Razmatra se posebno pitanje kroničnih učinaka ekspozicije kemijskim nadražljivcima kao i ekspozicije inertnoj prašini. Na temelju ocjene literaturnih podataka iznosi se mišljenje da jedan i drugi faktor mogu imati određenu ulogu u – najvjerojatnije kompleksnoj – etiologiji kroničnog bronhitisa. Posebna pažnja obraćena je ocjeni dijagnostičkih kriterija kroničnog bronhitisa.

U posljednjem poglavlju razmatraju se metode istraživanja kroničnog bronhitisa odnosno obstruktivnih bolesti pluća u industriji. Ističe se naročito važnost standardizacije anketnih upitnika i funkcionalnih testova pluća. Ukazuje se posebno na problem uzorka i zaključuje se o potrebi kontinuiranog korištenja pojedinih grupa radnika kao i uzoraka opće populacije. Zatim se iznosi kratak opis jednog modela koji se može koristiti za takva ispitivanja.

Na kraju prikaza prikupljena je najvažnija literatura iz područja koje se obrađuje s ukupno 183 bibliografska podatka.

Institut za medicinska istraživanja i medicinu rada, Zagreb

Primljeno 15. XII 1968.

OCCUPATIONAL HISTORY*

Name _____ Identification Number _____

1. What occupations have you worked in? (List in order by year, starting with present position.)

Occupation	Dates of Employment	Industry

2. Do you work, or have you ever worked, where you were exposed to dusts such as:

	Length of Exposure (Years)	Occupation
☐ Sand dust		
☐ Hard stone dust		
☐ Asbestos		

* Questions concerning present and previous respiratory conditions, smoking habits and etc., can be used from a standard questionnaire such as Questionnaire of the British Medical Research Council's Committee on the Aetiology of Chronic Bronchitis.

☐ Talc _____ _____
☐ Coal dust _____ _____
☐ Metal dust(s) _____ _____
☐ Cotton dust _____ _____
☐ Crop or grain dust _____ _____
☐ Sawdust _____ _____
☐ Tobacco dust _____ _____
☐ Cement dust _____ _____
☐ Clay dust _____ _____
☐ Any other dust (spec.)

_____ _____ _____
_____ _____ _____
_____ _____ _____

3. Do you work, or have you ever worked, where you were exposed to irritating chemicals such as:

	Length of Exposure (Years)	Occupation
☐ Ammonia		
☐ Sulfur dioxide		
☐ Chlorine		
☐ Nitrous fumes and other welding fumes		
☐ Hydrogen sulphide		
☐ Methyl bromide		
☐ Acetaldehyde		
☐ Acetone		
☐ Fuel oil		
☐ Gasoline		
☐ Petroleum		
☐ Cooking fats		
☐ Acids		
☐ Solvents		
☐ Any other dust (spec.)		

_____ _____ _____

(If the subject is not certain of the irritant he was exposed to, record only the lenght of exposure and occupation.)

4. Do you work, or have you ever worked, where you were exposed to the following environment(s):

	Length of Exposure (Years)	Occupation
☐ Damp		
☐ Wet		
☐ Hot		
☐ Dry		
☐ Sudden temperature variation		

5. Have you ever suffered from any acute illness caused by an irritating chemical?
☐ Yes ☐ No
If yes: Specify the irritant and the date when illness occurred.

Irritant	Year	Irritant	Year

6. Have you ever left any position because your chest was affected by the working conditions?
☐ Yes ☐ No
If yes: Specify the type of work you were doing and the year you left the position.

Type of Work	Year	Type of Work	Year