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OCCUPATIONAL FACTORS IN PULMONARY DUST DISEASE.[1]

By GORDON C. SMITH, M.B., B.S.,

Industrial Health Unit, School of Public Health and Tropical Medicine, Sydney.

THE Third International Conference of Experts on Pneumonokoniosis, held recently in Sydney under the auspices of the International Labour Organisation (1950), adopted the following definition of pneumonokoniosis: "a diagnosable disease of the lungs produced by the inhalation of dust, the term 'dust' being understood to refer to particulate matter in the solid phase, but excluding living organisms" (the term "diagnosable" indicating the presence of signs or symptoms, but not always loss of function).

The implications and scope of this definition are very wide. Whilst pneumonokoniosis in the etymological sense means "dust in the lung" and therefore is applicable to the pulmonary reactions due to all types of dust (and fumes), in practice it has been associated chiefly with silicosis, asbestosis, and in recent years with the lung disease due to coal dust—diseases in which permanent fibrosis occurs and with characteristic radiographic abnormalities.

Of late it has become evident that acute, semiacute and chronic pulmonary conditions may result from inhalation of a variety of dusts, both organic and inorganic, other than silica, asbestos and coal. Although the nature and severity of the tissue reaction vary according to the type of dust and conditions of exposure, the term pneumonokoniosis can be applied to these conditions.

[1] Read at a meeting of the Section of Public Health, Tuberculosis, Tropical Medicine and Industrial Medicine, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

The Occupational History in Pneumonokoniosis.

Whilst the diagnosis of pulmonary dust disease should never be made without proper consideration of the occupational history, medical history, clinical examination and radiographic findings, the first is of special importance. Unless there has been exposure to dust of a harmful nature there cannot be a pulmonary dust disease, no matter what the medical history or clinical examination might indicate, and no matter how closely the radiographic appearances of the lungs simulate those of pneumonokoniosis.

Thorough and detailed inquiries should be made about any process in which the patient may have been exposed to dust—the nature and conditions of the work, the type of dust and duration of employment at the process. Unless these particulars are obtained accurately, the industrial history may be rendered invalid, with resultant error in diagnosis. Negative evidence as to dust exposure is as important as positive; pneumonokoniosis has been diagnosed in certain individuals with suspicious radiographic signs, evidently on the film appearances alone, for a proper inquiry as to the nature of their work would have revealed no history of industrial dust exposure in these cases.

Properties of Dust in Relation to its Disease-Producing Capacity.

Although all dusts, if inhaled in sufficient quantities, may be regarded as being potentially harmful, some are known to be considerably more injurious than others. Important information concerning the potential capacity of a dusty environment to cause disease of the lungs can be obtained from a study of the following factors: (i) the composition and nature of the dust, (ii) the size of the dust particles, (iii) the intensity of exposure, as determined by (*a*) the atmospheric concentrations of the dust, (*b*) the duration of exposure.

The Composition of Dust.

An analysis for the chemical or mineralogical constituents should be carried out on any dust suspected of causing pneumonokoniosis, and, preferably, the air-borne dust should be examined.

The most injurious component of an industrial dust is crystalline free silica (uncombined silicon dioxide—SiO_2). The pulmonary reactions from amorphous or non-crystalline free silica are less severe, while silicates, that is, silica in a combined form, with the notable exception of asbestos (a hydrated magnesium silicate), are less harmful than free silica.

Most minerals which contain silicates also contain some proportion of free silica; compounds of other elements, such as iron, aluminium, calcium, magnesium and potassium, may also be present. Because of the possible modifying or inhibitory influence of certain substances on the action of free silica, the analysis should be not only for free and combined silica, but also, where indicated, for other likely constituents, especially if these by themselves are capable of producing a lung reaction.

In regard to metallic dust and fume, even though there may be no question of the presence of silica, the concentration and chemical analysis of the atmospheric contaminant should be determined in order that a proper appraisal of the suspected hazard may be made.

In the case of siliceous materials, as a general rule and other conditions being similar, the higher the percentage of free silica in a dust, the greater is its capacity to produce disease. When pulmonary disease is caused by dusts which contain lower percentages of free silica than, say, sandstone or quartz, greater exposures due to higher atmospheric concentrations of dust, or to a longer working period, or both, are necessary. Thus pneumonokoniosis has been found in men engaged in "dragging" bricks from continuous or patent kilns. Tests in New South Wales have shown these men to be exposed to very high concentrations of dust containing about 25% of free silica.

The pulmonary changes in coal miners are out of proportion to what one would expect, having regard only to the free silica content of coal dust, which Smith and Whaite (1950) found averaged 1·8% in the under 10 micron fraction of sixteen samples collected from the coal face of five collieries in New South Wales, but a long period of exposure seems necessary to produce disabling pneumonokoniosis.

Asbestos, which contains no free silica, produces a severe pulmonary disease, but the mode of action of the asbestos fibre differs from that of the silica particle.

The pneumonokoniosis conference agreed that an analysis of material from which dust is derived does not of necessity represent the composition of the dust inhaled in an industrial process. The free silica content of air-borne dust may be similar or may be appreciably increased or decreased, compared with that in the parent material, according to the nature of the material and type of industrial process.

The problems associated with exposure to mixed dusts, for example in foundries, are of interest. Foundry dusts may comprise a mixture of carbon, free silica, silicates, iron oxide and other metal particles, as well as particles from carborundum and other abrasive materials. Iron oxide may play some part in the production of the nodular shadows seen in radiographs and may influence the action of silica in the lungs of certain foundry workers apparently affected with silicosis. One may also question whether any of the nodulation seen in films of grinders with apparent pneumonokoniosis is due to particles from metals or synthetic abrasives.

The physical nature of substances, whether hard and dry or soft and wet, influences their capacity to produce dust. Thus the fact that work is being done in sandstone does not necessarily mean that there will be a serious dust hazard, despite the high free silica content of the rock. If the rock was wet and soft, little or no dust would be [illegible] breaking it up, whereas if it was of a dry, tightly cemented type, dust high in free silica would be generated, because more energy would be required to rupture it.

The Particle Size of Inhaled Dust.

The pneumonokoniosis conference concluded that in the case of fibrous dusts, such as asbestos and certain vegetable dusts, the larger sizes appear to be more harmful, whereas in many mineral dusts the smaller sizes seem to be the most injurious. In general, whilst particles of mineral dusts (except asbestos) exceeding five microns in size are of minor importance, the lower limit of the size of particles causing pneumonokoniosis has not yet been determined.

Particles of the harmful size range cannot be seen with the naked eye, and it is well to remember that a dangerous concentration of dust of this particle size may be present in the air without being seen. A visible dust cloud may contain fine harmful particles as well as those of larger innocuous size.

The actual number and size distribution of dust particles depend upon the method by which dust is produced and the nature of the parent material from which the dust is derived. Experience in New South Wales, like that of investigations elsewhere, is that at least 70%, and in some cases over 90%, of dust particles generated in industrial processes are up to three microns in size. About 5% of coal dust particles and only about 1% of sandstone particles are over five microns in size. Thus not only is there a relatively small production of the larger particles, but such particles settle out from the atmosphere more rapidly than the smaller sizes. Further, it is known that only a portion of inhaled dust is retained permanently in the lungs, most particles of about five microns and larger being removed by the physiological mechanisms of the nasal and upper respiratory passages.

In a recent review of this subject Hatch (1950) suggests that particles smaller than one micron in size may be the principal contributors to silicosis production. Vorwald (1950) showed in animal experiments that "amorphous" silica of particle size down to 0·005 micron produces profound lung changes, and states that the rate of tissue reaction to dust is inversely proportional to particle size.

On the other hand, in regard to asbestos, it appears that the larger particles are more harmful than the smaller and that in the lungs they act as mechanical rather than as chemical irritants (Gardner, 1938). Wyers (1949) states that dangerous fibres measure about 20 microns.

Dust Exposure.

In general, exposure is measured in terms of concentrations of air-borne dust and the period of time during which the individual is exposed to those concentrations; the higher the average concentration of air-borne dust, and the longer the period of exposure, the greater will be the risk of contracting pulmonary dust disease.

In the case of silicosis and related types of pneumonokoniosis, including asbestosis, the disease is usually of gradual onset and slow development. It does not become evident until after exposure for a period of years, the actual time varying according to the type of dust and industry.

Meiklejohn (1949) states that under present conditions in Great Britain silicosis in a diagnosable stage seldom occurs in under fifteen to twenty years' exposure to the risk, and in regard to coal miners, McVittie (1949) gives a similar period for the development of pneumonokoniosis.

My own observations on a group of 416 New South Wales coal miners showed that the average period of exposure in the men with disabling pneumonokoniosis was thirty-four years, their average age being fifty-four years.

Asbestosis apparently occurs after a shorter exposure. Thus Wyers (1949), who investigated 115 fatal cases of asbestosis, found the average exposure had been 10·4 years.

Under conditions of intermittent exposure it is more difficult to predict the likely effect on the lungs than if the exposure was continuous, but the severity of any dust

hazard is usually lessened in such circumstances. This, however, might not apply with very high intermittent exposures.

In the case of metallic and vegetable dusts, the exposure period necessary to produce a pulmonary disturbance is generally much shorter than with other dusts—sometimes months or weeks, or even less, rather than years. In some instances a single exposure may be sufficient to cause a reaction.

The literature contains reports of the occurrence of "acute" or "rapidly developing silicosis" after short exposures to high concentrations of silica dust, and the following notes about a Sydney case are of interest.

In April, 1942, a man, aged thirty-eight years, died three weeks after he had collapsed at work. The significant details of his industrial history were as follows: From 1917 to 1919 he was engaged in coal mining (Cessnock) for two years. From 1931 to 1937 he was with the Water Board, including fifteen months on rock work. From 1937 to 1940 he was for two years a builder's labourer, excavating in sandstone in the sub-basement of a building. Latterly he had been using a "water pick", but earlier had been using a "dry pick" for six months.

His chest had been examined by X rays in 1937, 1940 (on two occasions, the second being some weeks after he had left the basement excavation work), and again in 1942, 17 days before his death. It was only in the film from the last examination that the radiologist reported evidence of "bilateral nodular fibrosis".

At autopsy, aortitis, congestive cardiac failure, and silicosis of the "rapidly developing" type were found. Microscopically the lungs showed generalized, somewhat immature fibrosis, mainly diffuse, but with some tendency to nodular arrangement.

In October, 1939, the New South Wales Division of Industrial Hygiene had investigated the working conditions in the basement of the building where this patient had been employed. Water jets were fitted to the pneumatic picks and an exhaust ventilation system was installed. Nevertheless concentrations of sandstone dust, ranging from 1150 to 10,000 particles per cubic centimetre (by Owens's dust counter) were obtained. The dust conditions in 1937 were probably worse than in 1939; there is little doubt that this patient contracted silicosis as the result of dust inhalation during the period 1937 to 1940.

Occupations, Dusts et cetera, Associated with a Pneumonokoniosis Hazard.

The most important risks are those where exposure to dust containing free silica is likely to occur. Among the commoner processes involved are tunnelling, excavating, mining or quarrying in sandstone or quartz; cutting and surfacing of sandstone or granite; sandblasting, dressing and moulding in foundries, especially machine moulding in iron foundries if siliceous parting powders are used; milling siliceous ores; certain pottery processes; in the brick industry, chiefly "dragging" in patent kilns; manufacture of abrasive soaps and powders; manufacture and handling of refractory materials, and metal mining. In several of these instances, and in a number of others not specially mentioned here, the dusts are of a mixed nature but still contain significant percentages of free silica.

Pneumonokoniosis of coal miners has been discussed in other papers and will not be further considered. In regard to asbestosis, which is of very infrequent occurrence in this country, all the evidence from overseas indicates that it is much more common in workers engaged in the processing and fabrication of asbestos in factories (for example, textile work) than in asbestos miners.

In recent reviews of the literature, Perry (1947), Doig (1949) and Middleton (1950) have described disturbances of the respiratory organs due to mineral dusts other than silica, coal and asbestos, and have discussed the reactions due to vegetable dusts.

I propose briefly to mention the more important of these lung disorders, because, although to my knowledge most of them have not been reported in this country, the industries or processes involved in their causation are carried out here or may be conducted in the future, and so we should be familiar with the possible occupational risks.

Before referring to these, however, I wish to make a few comments about the lung changes which have been observed in basalt workers and biograph operators in New South Wales.

Basalt Work.

Basalt (or "blue metal") is a basic igneous rock, composed essentially of a lime-bearing feldspar with smaller amounts of one or other of the ferro-magnesian minerals and a little iron oxide. Free silica is typically absent but may be present up to 2%. Thus basalt is a silicate, with 40% to 50% of silica in combined form (Whitworth, 1947).

Badham (1927) described a case of fine fibrosis of the lungs in a man, aged sixty-one years, who for twenty-five years had worked in a blue metal quarry and crusher house and was exposed only "to the dust of orthoclase basalt, which contains no free silica". This man died, but an autopsy was not performed.

In recent years I have seen two men, aged forty-nine years and fifty-three years respectively, who, as far as could be ascertained, had been exposed only to basalt dust. The first man had been a crusher attendant for fifteen years at the same quarry as Badham's patient, and the second had worked chiefly on the crusher and screens for thirty-five years at another blue metal quarry. The radiographs of both men showed a well-marked, generalized, fine type of nodulation throughout the lung fields; in the film of the first man, who was totally disabled, there were also large shadows suggesting consolidation.

Biograph Operating.

Two brothers, whom I first saw in 1947, were aged forty-one and forty-six years respectively and had both worked in the projection booth at the same suburban cinema—the elder for ten years and the younger for twenty-three years. There was no history of exposure to dust in other employment. The elder man complained of shortness of breath and loss of weight; his brother, of pains and tightness in the chest. The chest radiographs of both men were similar and showed a generalized fine nodulation throughout both lungs. Films of both men taken in 1949 showed no significant change since 1947.

In 1946 the New South Wales Division of Industrial Hygiene had found that the concentration of dust in the air of the biograph box in the cinema concerned was approximately 6000 particles per cubic centimetre of air by the Owens dust counter; the dust particles were mostly about half a micron or less in size. The ventilation in the box was faulty. A sample of dust obtained from the biograph room in 1946 on analysis gave the following result.

Total SiO_2	6·0%
Rare earth oxides ..	48·0%
$Al_2O_3 : Fe_2O_3$	17·0%
CaO	6·7%
Loss on ignition ..	2·0%
Copper	Trace
Fluorides	Present
P_2O_5	Present
SO_3	Present

Following the discovery of the unusual pulmonary condition in the two brothers, seventeen biograph operators from fourteen other theatres were submitted to X ray examination. Their period of employment ranged from seven to thirty-seven years, and some had worked in badly ventilated booths. However, no evidence of a lung reaction resembling pneumonokoniosis was found.

Several authorities have investigated gases, fumes and dusts from carbon arcs and the ventilation of motion picture booths. MacQuiddy and others (1939) showed that some of the high-intensity carbon arc ashes appear to cause mildly proliferative reactions when injected intraperitoneally in the albino rat. They state that it might be deduced that, if inhaled in sufficient quantities over long enough periods of time, these ashes might cause a proliferative type of pneumonokoniosis.

On the limited evidence at present available, and in the absence of autopsy studies, it is not possible to say whether the lung condition present in these basalt workers and pulmonary biograph operators is due to dust. Further (with the exception) the

responsible, the question arises whether it has caused fibrosis or is merely present as inert deposits.

It seems reasonable to associate the radiographic abnormalities of the basalt workers with inhalation of dust at their employment, but in the case of the biograph operators, whilst occupational factors cannot be ruled out (indeed, one of these men was compensated for "silicosis"), as they were brothers, and as examinations of other operators gave negative results a non-industrial ætiology has to be considered.

Although these cases are incomplete, I mention them in order to emphasize that with present advances in industrial activity and with increasing knowledge of pneumonokoniosis, we must constantly be watching for lung conditions due to dust, not only from new processes, but also in industries and occupations where previously they have not been suspected.

Talc.

Until comparatively recently asbestos was considered to be the only harmful silicate. Now, however, there is evidence of several investigators that talc, a hydrated magnesium silicate which occurs in flaky and fibrous forms and is used in a number of industries, may cause significant lung changes. McLaughlin, Rogers and Dunham (1948) described a case of pneumonokoniosis confirmed at autopsy in a man, aged fifty-one years, who had worked for thirty-seven years in a rubber tire factory, where he had been exposed to a "fair concentration" of talc dust, and who died primarily from rheumatic endocarditis. There was much fibrosis in the lungs and curious bodies similar to absestosis bodies were also present. These authors conclude that talc pneumonokoniosis and asbestosis are similar diseases, and that pneumonokoniosis is probably caused only by the fibrous varieties of talc.

Graphite.

Doig (1949) and Middleton (1950) refer to recent reports by other authorities in Britain, chiefly Dunner and Gloyne, concerning pulmonary disease in graphite workers.

According to Harding and Oliver (1949), graphite (plumbago), which is a crystalline form of carbon, may be mixed with up to 10% of free silica and with other minerals. They point out that grinding of natural graphite produces the greatest dust risk and can lead to a disabling and fatal pneumonokoniosis, which they describe as resembling, both radiologically and histologically, that of South Wales coal workers.

Other Silicates.

Abnormal reactions in the lungs of animals or man have been described following inhalation of dust from china clay, mica, sillimanite and fuller's earth.

Cement.

Cement is mentioned here because of its very widespread use and of questions which periodically arise as to its possible harmfulness. The raw materials from which cement is made are chiefly limestone, which has not more than about 1% of free silica, and shale, which may contain from 20% to 40% of free silica. The finished product has practically no free silica. Gardner and others (1939) examined 2278 employees from 11 cement-making plants in the United States of America. Only eight persons showed evidence of nodular fibrosis attributable to dust, and in six of these exposure to silica dust in previous employment was presumably responsible. Baetjer (1947) showed that in rats exposure to high concentrations of cement dust did not lower resistance to lobar pneumonia and did not produce any acute or chronic pathological changes in the lung tissue.

Metal Fume Fever.

The well-known example of a minor pulmonary reaction, [illegible] fume fever, due to fume rather than to dust, mostly If the [illegible] exposure to zinc oxide fumes, for example, in form[illegible]in b[illegible]s, or from welding on galvanized iron. It is said to be caused also by fumes of other metals. When it is due to zinc there is an acute febrile reaction or chill, of short duration, and with no permanent lung damage. If other metallic oxides, such as cadmium or manganese, are involved, a more serious pulmonary condition may ensue.

Cadmium.

Johnstone (1948) states that "cadmium has probably more lethal potentialities than any other of the metals". The inhalation of dust or fumes from cadmium compounds, especially the oxide (for example in welding on cadmium-plated surfaces, from the ignition of cadmium and from the use of cadmium in solder) produces irritant effects, which may be severe, in the respiratory tract, perhaps with pulmonary œdema and death.

Beryllium.

The literature on the pulmonary (and other) lesions affecting workers exposed to beryllium is now extensive, many reports having been published during the past five years. Abstracts of the various papers and articles were recently made in Australia by Taylor (1949). Two principal types of pulmonary lesion have been described: an acute pneumonitis and a chronic diffuse granulomatosis. A number of fatal cases have occurred.

It appears that the acute form which may follow a single exposure to toxic fumes occurs mainly among workers exposed to compounds in the extraction and refining of beryllium from the ore beryl, usually within a few weeks of the first exposure.

The chronic or delayed form, which resembles sarcoidosis, is more persistent. A period of from several weeks or years may elapse from the time of the last exposure to the onset and recognition of the disease. This type usually occurs in persons who have been exposed to dust from fluorescent powders containing zinc beryllium silicate, notably in the fluorescent lamp industry. However, eleven cases of the chronic type were recently reported amongst residents in the vicinity of a beryllium-producing plant. There was no history of occupational exposure (Eisenbud and others, 1949).

Manganese.

Doig (1949) mentions that since 1921 a relationship between manganese and pneumonia has been suspected, following reports from Norway and Germany. In England, Lloyd Davies (1946) found that men exposed to dust of oxides of manganese experienced a high incidence of a condition which he called "manganese pneumonitis". No permanent lung changes were observed. Three years later Lloyd Davies and Harding (1949) reported that pneumonitis has continued to affect manganese workers.

Firket (1950) states that chemical pneumonia which follows inhalation of dusts of manganese, vanadium, osmium and beryllium resembles virus pneumonia both clinically and histologically, and he questions whether the dusts themselves provoke the disease or whether they activate viruses.

Iron Oxide.

Doig and McLaughlin (1936) described a fine nodulation in the lung fields of welders who were apparently in good health. Later, following further clinical and radiographic studies by them and other investigators, it became evident that the pulmonary changes were due to the deposition of iron oxide dust, which is present in welding fume and which is relatively opaque to X rays, and that they were not associated with symptoms, abnormal physical signs, or lessened capacity for work. A similar condition has been reported in other workers than welders exposed to iron oxide.

Enzer and Sander (1938) made an autopsy examination of the lungs of a welder whose chest radiograph showed fine nodulation and who died following an accident; there was no fibrosis in the lung tissue.

According to Doig and McLaughlin (1948), the changes due to iron oxide are not necessarily permanent. In one case described by them the abnormal lung shadows completely disappeared some years after cessation of welding, and in another the shadows became less intense.

Dusts from compounds of barium and tin, which are more radio-opaque than iron, are believed to behave similarly to the latter in the lungs.

Aluminium.

Since Denny, Robson and Irwin (1937) showed that experimental animals exposed to quartz dust *plus* 1% metallic aluminium powder did not develop silicosis, considerable interest has been taken in the question whether aluminium dust damages the lungs, and conflicting reports have been published.

The recent pneumonokoniosis conference decided that there was no conclusive evidence that the inhalation of aluminium in any form prevents the development of silicosis in man, or that aluminium powder is of value as a therapeutic agent in human silicosis or that it is harmful when used for this purpose. There was some evidence that under certain conditions the inhalation of aluminium in industrial processes may be harmful and further there was experimental evidence that in animals the inhalation of aluminium dust aggravates pulmonary tuberculosis.

Synthetic Abrasives.

Shaver and Riddell (1947) described an unusual lung disease, with fibrosis, in some cases fatal, in workers engaged in the manufacture of corundum (aluminium oxide). The process involves treating in special electric furnaces a mixture of bauxite, iron and coke at a temperature of 2000° C. Dense white fumes are given off, which, according to Jephcott (1948), consist mainly of alumina and silica.

Riddell (1948) states that the lung condition appears to be induced by an irritant of a chemical nature, but that the precise agent has not been determined.

These findings are of interest because artificial abrasives (for example, carborundum, that is silicon carbide, and aluminium oxide) have such widespread use in industry. The observations of Smith and Perina (1948) and of Bruusgaard (1948) indicate that dust from synthetic abrasives, when inhaled, may produce nodular shadows in the lungs.

Vegetable Dusts.

A variety of respiratory disturbances, some only of a minor nature, have been attributed to dust from vegetable materials, such as cotton, flax, hay, grain, bagasse and timbers. Three examples may be cited.

Cotton: A condition termed byssinosis (chronic bronchitis and emphysema with a tendency to asthma) has been reported, particularly amongst card-room and blow-room workers, in the cotton-spinning industry in Britain and elsewhere. There are no specific X-ray or autopsy findings. The literature has been well reviewed by Caminita and others (1947).

Bagasse: This is of interest to Australians because of the sugar industry here. Bagasse (or megass) is sugar cane after the sugar has been extracted, and is used in the manufacture of wall boards. Since 1941 cases of respiratory disease (bagassosis) have been reported in workers handling bagasse—in England the persons affected were those engaged on a machine ("shredder") which broke up the bales of bagasse. Hunter and Perry (1946) reviewed the literature fully and described eleven cases (one of the patients subsequently died) of acute bronchiolitis and pneumonia, arising from the inhalation of bagasse dust. They considered the disease was most likely caused by fine vegetable dust; however, its exact ætiology has not yet been determined.

Wheat Dust: Investigations have indicated that exposure to wheat dust may result in asthma (Duke, 1935) and an increased liability to respiratory disease (Smith and others, 1941). The fine hairs of the grain are believed to be responsible, at least in part, for these reactions.

Pulmonary Carcinoma.

Where an occupation, which is associated with an apparently high incidence of lung cancer, has involved dust exposure, it is reasonable to consider carcinoma in a discussion of pulmonary dust disease.

The problem has been reviewed briefly by both Doig (1949) and Perry (1947). They mention the high incidence of lung cancer in the Schneeberg miners in Saxony and in miners in the Joachimstal area in Bohemia. Arsenic and radioactive ores are both considered likely causes in these cases. They also refer to reports by various investigators which indicate that workers in chromate factories, and those exposed to arsenic and asbestos, have a high incidence of pulmonary cancer.

In a personal communication Dr. J. H. Blakemore, of Concord, advises that in a chromate factory in New South Wales, which has been operating since 1944, and which at present employs about 100 persons, all workers engaged in the various stages of production have an X-ray examination of the chest before starting work and at three-monthly intervals thereafter. To date, nothing to suggest pulmonary cancer has been found. In its early years this factory had a number of cases of nasal septal perforation and skin conditions due to chrome, and relatively high concentrations of chromates were found in the atmosphere.

Prevention of Pulmonary Dust Disease.

The measures to be applied in the suppression and control of dust, and for the protection of the workers, are essentially the same, irrespective of the industry and type of dust. Briefly, they include the following:

1. The abolition of processes creating harmful dust. If that is not possible, the total enclosure of such processes in mechanically ventilated structures, or their segregation from other operations in the factory. Isolation can be applied either geographically or by time, that is, by carrying out a dusty process when no other workers are in its vicinity.

2. The use of a harmless material in place of one that is dangerous.

3. The removal of dust at its point of origin by local mechanical exhaust ventilation.

4. The use of water or other wetting agent to prevent dust from certain processes becoming air-borne.

5. Electrostatic precipitation.

6. The provision of adequate general ventilation to dilute contaminated air.

7. Maintenance of a high standard of plant cleanliness and housekeeping.

8. Personal respiratory protection of the worker. Respirators should be worn only when other methods of protection are not practicable or for jobs of short duration. They should be well fitting, comfortable and efficient and should be kept clean.

9. Preemployment and periodical medical examinations, including a radiograph of the chest, of workers exposed to a dust hazard.

If these control methods, or such of them as are applicable to a particular industry or process, are conscientiously applied and maintained, workers would be protected against hazardous exposures to dust.

Summary.

The term pneumonokoniosis is defined and its scope briefly outlined; the need for a complete and accurate occupational history in the diagnosis of pulmonary dust disease is stressed.

The potential capacity of a dust to cause disease is discussed, with special reference to its composition and nature, particle size, and intensity of exposure. In addition to dusts containing high percentages of free silica, others, in which free silica is absent (for example, asbestos and certain metals), are capable of producing severe pulmonary damage. In most dusts (asbestos being an exception) the

harmful particles are those less than about five microns in size. Intensity of exposure, which varies for different dusts, depends on the atmospheric concentrations of dust and the duration of exposure. Brief notes of a case of "rapidly developing" silicosis are given.

The main occupations and dusts associated with a risk of pneumonokoniosis are briefly discussed. In addition to diseases caused by silica, coal and asbestos, reference is made to the radiographic abnormalities of certain basalt workers and biograph operators, and to the pulmonary effects of talc, graphite, cement, zinc, cadmium, beryllium, manganese, iron, aluminium, synthetic abrasives, cotton, bagasse and wheat.

The occurrence of pulmonary carcinoma in workers exposed to chromates, arsenic and asbestos is mentioned.

Measures for the control of dust and protection of workers are stated.

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THE CHANGING FACE OF OBSTETRIC INFECTION.[1]

By ARTHUR M. HILL, M.D., D.G.O. (Melbourne), F.R.C.S.E., F.R.C.O.G.,

Gynæcologist to In-Patients, The Women's Hospital, Melbourne.

THE last fifteen years of the half-century now ending have seen the greatest advances of all time in the prevention and treatment of obstetric infection. This is due predominantly to the introduction and development of modern chemotherapy and antibiotic therapy; but important contributory factors are improved bacteriological knowledge and methods of investigation and a clearer appreciation of the place of maternal resistance in combating infection.

It is proposed to review the major effects of these advances on puerperal morbidity, on the death rate of the main bacterial types of puerperal and abortional infection, and on modern obstetric practice, at the Women's Hospital, Melbourne, over the past ten years. During that period 41,643 women, 80% of whom had attended our own antenatal clinics, were confined; and 18,285 cases of abortion, of which 5175 were diagnosed as "septic", were dealt with. The annual morbidity following the confinements is shown in Figure I.

Sulphonamides were in use in the earliest of these years, although the compounds employed were in general more toxic and less efficient than those now available. In addition, they were not always given in adequate dosage, and their use was largely restricted to the therapy of established infection. However, when it is remembered that less than 10% of all cases of obstetric infection are likely to be of a bacterial type responsive to sulphonamide

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