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CLINICAL ASPECTS, DIAGNOSIS AND TREATMENT OF PNEUMOCONIOSIS*

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THERE are two types of dust which may cause some degree of pneumoconiosis; these have been termed inert and active dusts. The determination as to whether or not a dust is active or inert has been reached by pathological examination of the lungs of those dying of pneumoconiosis, by clinical studies, especially x-ray, and by animal experimentation.

Review of Animal Experiments

The study by animal experimentation has been particularly valuable as it provides a method of determining the effect of a given dust upon the living tissues and a comparison with the effect of similar exposure to other dusts. The animals used have been white rats, rabbits, guinea pigs, monkeys, fowl, cats, and, for some experiments, tadpoles and fish (1, p. 44).

The methods used have been:

1. *Dusting.*—This consists in exposing the experimental animal to a "high concentration of dust (up to 10 or 12 billion particles per cu. ft. of air, dark-field) to produce maximum effects in short time." This method might be called the normal breathing method.

2. *Injection of suspensions of dusts.*—This consists in injecting suspensions of dusts into various parts of animals to detect the capacities of different dusts to provoke reaction in the tissues. Results are obtained more rapidly than by dusting but do not correctly show the natural development of disease in the lungs.

The dust suspensions may be introduced into the animal by:

1. Intratracheal or bronchoscopic injections.

2. Intraperitoneal injections (Miller-Sayers method (2)).

3. Subcutaneous injections.

4. Intravenous injections—showing the effects of the dust on the extrapulmonary viscera.

5. Intracutaneous injections—for gross reaction.

6. Intra-lymphatic injections—for reaction of lymph node cells (3).

All animals tested either by dusting or by injection showed a tissue reaction to silica. This reaction was typified by the formation of fibrotic nodules except in the cold blooded animals where there was necrosis followed by some fibrosis. Changes in susceptibility of animals to tuberculosis as a result of the effects of various dusts have also been studied, and the

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activating effect of silica demonstrated.

The effect of the inert dusts upon the animal tissue was found similar to that of any foreign body. There was reactionary enlargement of lymphatic nodes, slight fibrotic reaction immediately surrounding the injected dust but no continuation of this fibrosis and no formation of nodules. The exact pathology noted was scattered or clumped cells containing the inert dust lying in the alveoli, slight inflammation or no inflammation of the adjacent walls, a slow accumulation of dust-containing cells in the lymph nodes with enlargement of the nodes, and a deposition of dust about the lymphatics of the lung or pleura. When inert substances were injected intravenously, intraperitoneally or subcutaneously, there was no reaction beyond that of any foreign body (2).

As a result of such experiments which have been repeated in many parts of the world, it is believed that of all suspected dusts only silica has a specific action upon animal tissue which results in fibrotic nodulation. Asbestos fibres produce a peculiar reaction in the lung which is different from silica. Following the inhalation of asbestos dust there are formed "cuffs of more dense connective tissue about the terminal bronchioles" (1, p. 46). Contraction and collapse of the alveoli supplied by the affected bronchioles occurs. This is followed by induration and fibrosis of the collapsed alveoli, presenting the picture of diffuse fibrosis of parts of the affected lung with persistent foci of normal air spaces.

Silicosis

The Committee on Pneumoconiosis and the Committee on Standards of the American Public Health Association (4) adopted in 1932 the following definition: "Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis, (some or all of which symptoms may be present) and by characteristic x-ray findings."

I. Pathology of Silicosis

Dust particles after reaching the lung alveoli may remain quiescent for a considerable period as is the case with the inert dusts, or they may penetrate the lymph spaces in small numbers. The bulk, however, are phagocytosed, that is engulfed, by wandering endothelial cells which at first, lining the wall of the alveolus, become detached, and then, having taken up the dust particles, pass by ameboid movement through the walls of the alveolus into the lymph spaces. The cells now slowly migrate to the minute lymph islands which guard the entrance to the small lymphatic vessels, pass through these, thence to the lymph vessels, and finally are caught by the large group of lymphatic glands which form part of the hilus or root of the lung. These wandering cells are commonly called dust cells.

The piling up of cells and the reaction of the glands blocks the free circu-

lation of lymph so that the dust cells tend to move slowly toward the lung roots, blocking the lymph vessels and later the minute lymph masses which guard the entrance to the lymph vessels. The dust cells which are killed by the silica disintegrate and free the silica dust for further injury to the neighboring tissues. The reaction of the tissues to silica is the production of fibrous tissue. Thus fibrous tissue forms along the lymphatic vessels which accompany the blood vessels of the lung, invades the septa between the lobes, and spreads its shoots into the lung tissue itself. The minute masses of lymphatic tissue become fibrotic nodules and these, as time goes on, increase in number to such an extent that, combined with the fibrous tissue of the former lymph channels, conglomerate masses of fibrous tissue are formed, blocking off large portions of the lungs. The blocking of the lymph flow toward the hilus increases the spread of dust cells to the pleura and eventually leads to fibrosis of this structure.

The fibrotic nodule is characteristic of silicosis. It is a small, discrete hyaline mass surrounded by apparently normal lung. It does not exceed 6 mm. in diameter. "Occasionally some of these nodules may show microscopic foci of central necrosis" (1, p. 146).

This increasing amount of fibrous tissue obliterates the alveoli and provokes a compensatory enlargement of the alveoli elsewhere. This condition of enlargement is called emphysema. As the amount of air space is insufficient to permit the normal oxygen-carbon dioxide interchange of the lungs required by the body, the effort of the heart to pump blood fast enough may promote enlargement of that vital organ. This, however, if it occurs, is a terminal condition infrequently observed, as the patient usually develops pulmonary tuberculosis or dies of an intercurrent disease before this stage is reached. It must be remembered that only one-fourth of one lung is necessary for life, and that the amount of fibrosis must be enormous to restrict the lung capacity to this extent.

II. Symptoms

The effect of the underlying pathology upon the workman is at first too slight to be detected by physical examination and the worker shows no symptoms. While there is a pathological condition present, it is to all intents and purposes harmless, in that if it does not progress rapidly it may not provoke symptoms during a worker's life, or only during the last decade. Even respiratory disease of another nature, such as bronchitis or even pneumonia, may occur with recovery during this period.

As the condition progresses, however, more and more of the lung tissue is converted into fibrous tissue, and the patient begins to show certain symptoms. He complains of shortness of breath when he goes up-hill and says that he notices his heart beat on exertion. He also mentions a cough which is at first dry and infrequent, but which gradually becomes moist and frequent. With the cough he raises scanty, stringy sputum which is sometimes discolored. The sputum is never large in amount unless he catches cold and develops bronchitis. At this

stage an attack of bronchitis does not disappear but becomes chronic. During this period the silicotic may complain of pain in the chest due to pleurisy or to epigastric pain, anorexia, and morning vomiting. While he is able to work, the victim cannot carry on his usual tasks and seeks lighter work.

Slowly the shortness of breath increases, the patient has poor lung expansion, and his color becomes pale and his lips bluish.

At this stage pulmonary tuberculosis is a frequent complication. When this occurs, the cough becomes more severe and continuous, the sputum free and moist, areas of dulness to percussion are noted, râles are heard over the chest on physical examination, and cavitation may be detected. Tubercle bacilli may or may not be present in the sputum. Many of these cases are able to carry on light work for years before finally succumbing. Hemorrhage from the lungs occasionally occurs.

During the early stages when perivascular, peribronchial, lymph node reaction is present there are only indefinite physical signs. Irvine (5) describes these as follows:

> 1. A certain lack of elasticity of the chest wall during movements of respiration together with
> 2. A somewhat reduced air entry, and
> 3. A characteristic alteration of the inspiratory murmur from the normal vesicular character to a higher pitched or "harshened," thinned and commonly somewhat shortened type, the expiratory murmur although somewhat prolonged remaining fainter than the inspiratory.

There is little change in these signs as the disease progresses. "The percussion note is somewhat flattened without being definitely dull especially posteriorly. Breath sounds have more definite characteristic thinning, the expiration being longer and fainter" (6). With the advent of tuberculosis the physical signs are those of that disease.

III. X-ray

The most important evidence of silicosis is obtained by x-ray. While a flat film gives valuable information, an accurate diagnosis requires a stereoscopic set of films. In many cases a lateral film is desirable in order to determine the amount of emphysema present and the size of the heart. Fluoroscopic examination is of interest in cases where diaphragmatic adhesions are suspected or shown on the film.

The old classification into first, second and third stages has been recently changed to a more descriptive nomenclature. The conditions noted on the film are now called:

1. Stage of perivascular, peribronchial, lymph node proliferation. Irregular exaggeration of linear markings.
2. Stage of nodulation.
3. Stage of fibrosis with conglomerate masses.
4. Any of above stages complicated by shadows characteristic of tuberculosis.

The first is not considered as diagnostic of silicosis as it may occur in persons who are in good health or in a number of pathological conditions which have nothing to do with silicosis.

The stage of nodulation is pathognomonic of silicosis but may be confused with films of miliary tuberculosis.

The stage of fibrosis with conglomerate masses may be confused with fibroid phthisis.

IV. Diagnosis

Diagnosis is made upon:

1. Occupational history with estimated length of exposure, quality of dust and quantity of dust.
2. Physical examination.
3. X-ray examination.
4. Presence or absence of tuberculosis as a complication.

Of these measures the first and third are most important. Probably the most difficult thing to decide is whether or not tuberculosis is present. Absence of fever and maintenance of weight, with absence of physical signs suggesting active inflammation in the chest, is indicative of simple silicosis.

The absence of tubercle bacilli from the sputum is inconclusive, as many cases of silico-tuberculosis fail to show bacilli in the sputum.

V. Progress

Silicosis once established in the lung has a strong tendency to progress. This appears to be owing to the toxic properties of the silica particle. What causes this toxic action is still in doubt. "Experiments and human pathological material indicate that in high concentrations silica is toxic and kills tissue; prolonged action of lower concentrations cause proliferation and fibrosis" (1, p. 46).

In spite of the fact that quartz is generally recognized as relatively insoluble, there is great rapidity of development of body reactions to silica. "Possibly unexplored properties of silica, electrical charge, adsorption etc., are concerned" (1, p. 48). This progressive tendency has been noted by Irvine, Böhme, Russell and others (1, p. 28).

The question is not what is the present condition of the silicotic, but how rapidly it will advance and how far. This is a serious problem in industry, for a worker having no symptoms and an early stage pathology by x-ray, may develop a disabling silicosis or a complicating tuberculosis. It is, therefore, possible for a worker to contract his disease while working for one employer and to develop symptoms many years later while working for another. If the work he is doing for the second employer involves exposure to an inert dust the employer may have difficulty in proving that this was not the cause of disability.

One of the striking differences between the effects of the inert dusts and silica dust is the lack of progress of pathology in the former as compared with the latter. Böhme (7) found that silicosis progressed after removal from exposure in 20 per cent of the cases diagnosed as having silicosis grade 1, in 40 per cent of the cases in grade 2 and in practically all the cases in grade 3. The experience of the State of Wisconsin, however, suggests that silicosis detected at an early stage in many cases will not progress if the worker is transferred to non-dusty work (8) though the reports of Watkins-Pitchford (9) and Britton and Head (10) make this somewhat doubtful.

Infection may play a part in the development. In fact, it is the frequent and serious complication of silicosis, and of the common infections, pulmonary tuberculosis is by far the most serious.

Tuberculosis may develop early or late in the disease. In those who have an inactive focus in the lung, the silica dust may change the condition to one of activity, or in cases of well-developed silicosis, tuberculosis may be superimposed upon the silicotic fibrosis.

Thus Kettle writes, "harmful dusts if inhaled into the lung may excite to activity a latent tuberculosis infection; they may exaggerate an active tuberculosis lesion or a coincident infection; and they may render the lung less able to cope with a superimposed infection" (11).

The course of silicosis with a secondary tuberculosis is usually slow and without the usual symptoms of intoxication and may be carried for years without serious impairment of working capacity.

While tuberculosis is the most frequent complication and cause of death, other infections do occur. On the Rand pneumonia is a common cause of disability and death. Pope and Zacks found pneumonia occurring with great frequency among foundry workers in Massachusetts. Proske and Sayers have confirmed Cummings' discovery of fuso-spirochetal organisms as a cause of infection among miners in Picher, while chronic bronchitis with asthma is fairly common (12, p. 47).

Collis and Yule (13) compared the mortality experience of an occupational group exposed to silica dust with that of the general population, and with that of an occupational group exposed to dust not containing silica. As a result of the study, they concluded that silica is a body poison like lead. Even though it exerts its primary injurious effects on the respiratory tract, the silica slowly invades the body, involving the circulatory system, the nervous system, the digestive organs, the kidneys and liver, and eventually causes death through its harmful effect upon one or another of these organs.

Asbestosis

"Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis both in its pathology and clinically. Asbestos is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water and traces of iron and nickel" (14).

I. Pathology of Asbestosis

Asbestos dust differs from other inhalable dusts in that it exists in thread-like fibres in which the diameter may be very small (5 microns or less) but which may be many microns in length.

These dust fibres do not appear to enter the alveoli. They are stopped at the neck of the alveolus where they are phagocytosed or penetrate the tissues. Acting specifically or merely as a foreign body they become surrounded first by mononuclear phagocytes, later by giant cells and last by fibrous tissue. There is no migration in dust cells to the lymphatics and lymph nodes as is seen in silicosis. The newly formed fibrous tissue contracts, constricting the neck of the alveolus so that no air can enter. Collapse of the alveolus follows and subsequent fibrosis.

In this way the lower parts of the lungs are filled with interstitial fibrosis and the air space markedly dimin-

ished. A compensatory emphysema may occur in the upper part of the lungs. Thickening of the pleura over the lower half of the chest is almost always present. Tuberculosis as a complicating factor is not common.

The asbestos fibre after it has become fixed at the neck of the alveolus frequently develops into a "curious body," the asbestosis body of Cooke (15). This is a long, narrow, cylindrical body, golden yellow and translucent, often with rounded ends like a dumbbell. These asbestosis bodies are found on autopsy and occasionally in the sputum. As similar bodies may occur in lungs not exposed to asbestos dust, the finding of these bodies is not diagnostic of asbestosis unless accompanied by a definite history of exposure to asbestos dust.

II. Symptoms of Asbestosis

The symptoms of asbestosis, like those of silicosis, are cough and dyspnea. With the cough there is sputum which may or may not contain asbestosis bodies. Hemoptysis is rare. There is loss of weight in the advanced cases, and marked reduction in the vital capacity and in chest expansion. The patient's face has an "earthy" look.

In advanced cases the dyspnea is out of proportion to the physical signs, being very severe, and is accompanied by blueness of the lips and occasionally by clubbing of the fingers. The fingers frequently show "asbestos corns" from the penetration and irritation of fine spicules of asbestos which are detached from the asbestos in handling.

III. Physical Examination

The patient, if an advanced case, shows loss of weight. The chest is emphysematous, and respiration is shallow, expansion being frequently less than 1 inch. The percussion note is one of dull tympany but with no definite dullness. The breath sounds are distant and expiration prolonged. The heart is normal in size, but lateral x-ray may show an anteroposterior enlargement.

IV. X-ray

This shows a fine mottling of a "ground glass" quality over the lower half of the chest. There is often an obliteration of the costophrenic angle and pleural thickening, shown particularly in the interlobular pleural on the right. The lateral view may show well-marked compensatory emphysema. A spot of tuberculosis at either apex is occasional but rare.

Still later in the course of the disease the appearance is that of a very fine stippling that obliterates most of the natural markings. The pleural shadow is definitely thickened. In some of the advanced cases the heart is enlarged and radiating from it into the lung fields is a series of heavy fibrous bands. This picture has been referred to as "porcupine heart."

Anthraco-Silicosis

This disease occurs among hard coal miners and is commonly known as miners' asthma. It is caused by the inhalation of large amounts of dust consisting of a mixture of anthracite coal and quartz, the quartz coming from the rock in which the coal is imbedded.

The pathology consists of a clogging of the lymph spaces with carbon particles which invade the upper lobes especially. Accompanying this is a linear or nodular fibrosis due to the

inhaled silica particles. When large areas of the lung are involved there is a compensatory emphysema.

I. Symptoms of Anthraco-Silicosis

The cardinal symptom of anthraco-silicosis is shortness of breath, which explains the term "miners' asthma." With this there is cough and sputum. The cough may be dry and the sputum scanty but if infection in the form of bronchitis is present, the sputum is muco-purulent and colored with coal dust. As in silicosis, tuberculosis is a frequent complication.

The physical signs are similar to those of simple silicosis or of silicosis with infection. Diagnosis is made largely by x-ray which gives a picture very similar to that of simple silicosis. The U. S. Public Health study on this problem (16) suggests that the true lung injury is due to the silica inhaled rather than to the carbon particles. Carbon has been found to be harmless when inhaled in the form of smoke and by animal experiment.

Inert Dusts

I. Pathology

The inert dusts are relatively harmless. They may increase the frequency of respiratory disease when inhaled in large amounts, but this has not as yet been proved statistically. Workers inhaling these dusts develop a mild fibrosis which follows the course of the lymphatics along the broncho-arterial tree and is accompanied by enlargement of the trachial lymph nodes. This reaction which is very slow, after a number of years may progress to a moderate amount of interstitial thickening. The pleura may be thickened and there may be diaphragmatic adhesions. These later manifestations only appear after many years of constant exposure to a high concentration of dust.

II. Symptoms Caused by Inert Dusts

The symptoms of the pneumoconiosis of inert dusts are negligible. If the exposure has been long and the dust discharge abnormally heavy, there may be some dyspnea on moderate working. This is not usually severe enough to interfere with the worker's normal activities and may be caused by degenerative changes of the heart due to advancing age, as much as to the pathology of the lung. There is no evidence that the inert dusts, unless mixed with an active dust, can produce disability.

III. X-ray Picture, Physical Examination and Diagnosis

The x-ray picture is that of perivascular, peribronchial, lymph node thickening which does not progress. Some diaphragmatic adhesions may show in cases with long exposure to heavy concentrations of dust. Serial pictures fail to show nodulation or the massive areas of fibrosis which characterize silicosis.

The physical examination of those exposed to inert dusts is usually negative. In a few cases where there has been prolonged exposure to great quantities of dust there may be some restriction of chest expansion and signs of emphysema.

The diagnosis is made by physical examination, occupational history, and x-ray.

A typical inert dust is that of the artificial abrasive, aluminum oxide. This material is widely used in grind-

ing wheels and for polishing, sand paper, etc. In the year 1929 over 71,000 tons were sold or used in plants in the United States and Canada (17).

The use of aluminum oxide creates a certain amount of dust. The effect of this dust has been studied by Clark (18) and Simmons clinically and by Gardner (19) on the experimental animal.

In a factory where artificial abrasives and grinding wheels are manufactured, Clark has studied the effects of inhalation of this substance for 25 years. He believes that in factories which provide proper dust removal, the continuous inhalation of artificial abrasive dust extending over many years of work does not produce the symptoms or present the x-ray findings of crippling fibrosis of the lung; that the number of cases of pulmonary tuberculosis does not greatly exceed the number normally present in the community; and that workers using wheels made of artificial abrasive or using this substance for polishing or other purposes run but slight risk of pneumoconiosis if excessive dust is removed by exhaust systems.

In spite of the fact that clinical and experimental evidence points to the relative harmlessness of the inert dusts, an effort should be made to keep the dust count around 20 million particles per cubic foot of air. This will make working conditions much more satisfactory to the worker and will reduce the hazard of respiratory disease.

Treatment of Pneumoconiosis

The only method of treatment of the pneumoconioses is prevention. Dust in quantity must be eliminated from industry wherever possible, and in most operations this is feasible. In a few where it is not, protective devices such as respirators or positive air pressure helmets must be used by the worker.

When a worker is found upon examination to have lung fibrosis it is wise to keep him at work in a nondusty department. While his lung condition, if it is due to silica or to asbestos dust, will progress slowly, many years of profitable work are before him unless infection intervenes. As dyspnea increases, lighter work must be provided. If tuberculosis complicates the picture and the patient develops tubercle bacilli in the sputum, he must be isolated from other workers, but even then may be able to do light outdoor work. In more severe cases where there is temperature, all work must, of course, be stopped.

Exposure

The rapidity of development of silicosis and of asbestosis depends upon the amount of dust inhaled and the percentage of free silica (SiO_2) or of asbestos in the dust.

In men exposed to heavy dust clouds with a high concentration of silica, silicosis has developed in as little as 2 years, but under the usual exposures of mining and of industry where the free silica usually varies from 13 to 35 per cent, the development is slow and symptoms do not appear for 15 or more years.

In the case of asbestosis, the development of symptoms is more rapid, 5 to 8 years being the usual required time of exposure.

Efforts are now being made to correlate the exposure with the pathology, as shown by x-ray, and with the physi-

cal signs. In other words, an effort is being made to determine standards of permissible amounts of dust in the working air for both the inert and the harmful dusts. Such standards will be of the greatest value to industry in its effort to reduce its dust hazard to a minimum.

Epidemiology

I. Silicosis

The danger of inhaling inorganic dust has been recognized for centuries, but careful study of the effect of such inhalation was first instituted in South Africa and culminated in the International Conference on Silicosis held in Johannesberg in 1930.

In 1933 Van Siclen (20) published an estimate of the health hazard from dust in the mines and allied industries of the United States. In his study he found that of 7722 men examined in the Tri-State zinc-lead district of southwest Missouri in 1927–28, 5704 were classified as negative for lung disease, 1362 had signs of first stage silicosis, 253 had second stage, and 32 had third stage. The remainder showed signs of tuberculosis with or without silicosis.

In Butte mines (Montana), of 1018 miners 42.4 per cent showed definite signs of dust injury to the lungs.

In the Lead-Deadwood district mines in South Dakota the sickness rates per thousand for respiratory disease was two and a half times greater than in general industry, while the tuberculosis rate was almost ten times greater.

Grouping the various industries studied, Van Siclen found that in metal mining about 62,228 workers were exposed while among those engaged in non-metallic mining or quarrying, 23,665 had a respiratory hazard.

The prevalence of silicosis in the general population was studied by Lanza and Vane in 1934 (21). They cite the following occupations as constituting a definite silicosis hazard:

1. Anthracite-coal and metal mining, quarrying.
2. Certain manufacturing industries such as potteries, glass works, and plants manufacturing granite, sandblasting.
3. Construction work—rock drilling, handling sand and gravel.

Their rough estimate of the number of workers exposed to silica dust to a harmful degree in the United States is upward of 500,000.

A careful study of 2,600 granite and foundry workers has recently been made by Pope and Zacks (22) in which correlation of the duration of exposure to dust and the incidence of silicosis was determined. Their conclusions are as follows:

1. In representative groups of both granite and foundry workers in Massachusetts the frequency of silicosis and of silicosis with tuberculosis was found to be positively correlated with the duration of exposure to dusts containing free silica and to concentration of such dusts in the occupational environment.
2. Among the granite workers examined silicosis alone was found in 15.2 per cent and silicosis complicated with tuberculosis in 7.6 per cent.
3. Tuberculosis is the cause of death in over one-third of all granite workers, a proportionate mortality three times that in foundry workers and four times that in all males of 20 years and over.

In 1907 Summons of the Miners' Phthisis Committee of Australia reported that gold miners there who contracted silicosis died of tuberculosis

(23). "The initial studies of silicosis by workers in South Africa were started by a demand made upon health authorities to determine the cause of the excessive mortality from tuberculosis which was increasing at a rapid rate among the miners there."

Russell found an excessive number of deaths from tuberculosis in his Barre, Vermont study of the health of granite workers (24) and Gardner (25) believes that at least 75 per cent of those who develop silicosis contract tuberculosis.

berculosis is so often associated with asbestosis that it seems probable that the association is more than accidental".

Gardner (1, p. 51) says that many autopsies show a combination of tuberculosis with asbestosis and other silicate dusts but "that surveys of living American workmen show no great excess of this infection", while Lanza (28) in a study of dust conditions in asbestos mines and mills in Canada and in fabricating plants along the Atlantic seaboard found in his study

TABLE 1

SILICOSIS AND ASBESTOSIS IN GREAT BRITAIN, THROUGH 1934*

	NUMBER OF DEATHS	AVERAGE AGE AT DEATH	DURATION OF EMPLOYMENT IN YEARS		
			Maximum	Minimum	Average
Silicosis	261	55.4	60	2.3	34.8
Silicosis with tuberculosis	315	52.5	67	2.0	32.0
Asbestosis	41	41.0	27	1.5	12.9
Asbestosis with tuberculosis	26	38.0	29	0.8	9.9

* J. C. Bridge: Report of Chief Inspector of Factories and Workshops, London, 1934, chapter 3.

II. Incidence of Asbestosis

The health hazard of asbestos dust has only been recently recognized. The Regulations for the Asbestos Industry in England have been in force for only 4 years. Bridge (26) says: "Over a number of years" (number not specified) "the deaths from asbestosis reported to the Department and compiled in 1934 numbered 41 while those from asbestos and tuberculosis numbered 26". (See Table 1.) Wood and Gloyne began their studies on pulmonary asbestosis in 1928 and published results of the study of 100 cases in 1934 (27). Most of these cases worked in the same factory. According to Wood, "pulmonary tu-

of 126 workers by x-ray that 67 were diagnosed as having asbestosis in some form, but that no predisposition to tuberculosis due to asbestos dust was indicated. Clark and Drinker (29) in summing up present beliefs say that while a secondary tubercular infection is not uncommon in asbestosis, it is far less frequent as a complication than in silicosis.

SUMMARY

1. Pneumoconiosis is a disease resulting from the inhalation of inorganic dust.

2. The two pneumoconioses which produce disability are silicosis and asbestosis.

3. The pathology of silicosis is characterized by the presence of fibrotic nodules scattered through both lungs, and that of asbestosis is characterized by an interstitial fibrosis involving the lower half of both lungs.

4. The symptoms of silicosis and asbestosis are similar, the most important being dyspnea.

5. The most common complication is pulmonary tuberculosis which in silicosis is a frequent cause of death.

6. The diagnosis of both silicosis and asbestosis is made largely by a correlation of history and symptoms with the x-ray examination.

7. The treatment of pneumoconiosis is preventive and symptomatic.

8. The course of silicosis and of asbestosis is slow but eventually leads to incapacity due either to the disease itself or to a complication, frequently chronic pulmonary tuberculosis.

9. The number of workers exposed to harmful mineral dusts in mining and in industry in the United States has been estimated as 500,000.

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