SILICOSIS AND ASBESTOSIS

A. J. LANZA

OXFORD MEDICAL PUBLICATIONS

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Silicosis and Asbestosis

BY VARIOUS AUTHORS

EDITED BY

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and Pendergrass published the first book to appear on this subject.[61] These authors, together with Sampson and others, have carried on experimental work in technique and in elaborating standards of diagnosis.[62]

The determination of disability in silicosis has proved vexatious to both clinicians and workmen's compensation officials. At the University of Rochester, McCann and his associates have been working on physiologic methods of estimating pulmonary impairment.

Harrington of the United States Bureau of Mines, Philip Drinker at the Harvard School of Public Health, Cummings at Saranac, Bloomfield and Dallavalle of the Public Health Service, Fehnel and his associates at the Metropolitan Life Insurance Company, have contributed a very large amount of work to the engineering and chemical methods of dust determination and control. Here again, the reports and publications are far too numerous to detail and the reader is referred to the original sources.

In addition to all the publications made available by all this field and laboratory work, the Harvard School of Public Health, the Saranac Laboratory, and the Division of Industrial Hygiene of the United States Public Health Service have undertaken direct educational activities. A recent organization is the Air Hygiene Foundation, sponsored by various industries to encourage research into atmospheric pollution and pulmonary disease and to carry on educational work.

2. HISTORY OF ASBESTOSIS

Asbestos was mined and the fibres were separated from the mineral and used in the making of fire-proof fabrics several centuries B.C. Attention is called by Cooke [63] to a cremation cloth described by Herodotus in 450 B.C. Materials made of asbestos have been used ever since but in the past three decades, the industry has grown tremendously owing to the

greatly increased use of fire resisting and insulating materials as well as brake linings in the automobile industry.

Asbestos is a magnesium silicate, containing a small amount of iron and a trace of aluminum. The fibres are long and flexible and for many purposes are combined with cotton to undergo regular textile processes of spinning and weaving. Most of the asbestos used in the United States comes from Canada.

The first report of a death resulting from asbestos dust inhalation was presented by Murray in 1900.[64] A second case was reported by Cooke [65] in 1924 and again in 1927.[63] At that time, Cooke described the unusual and characteristic asbestosis bodies. In 1927, Oliver [66] reported 2 cases, to be followed by McDonald [67] and Seiler.[68] In 1928, Simson [69] reported 2 cases in South Africa. Other cases were reported by Wood and Page [70] in 1929, Haddow [71] in 1929, and Wood and Gloyne [72] in 1930; in that year appeared the comprehensive report of Merewether and Price.[73]

In the United States, Lynch and Smith [74] reported the first cases in 1930. The first investigation of the asbestos industry in the United States was made in 1930 and 1931 by Lanza, McConnell, and Fehnel.[75] In the last few years, much attention has been given both to the clinical aspects and diagnosis of asbestosis and to its pathology. In 1936, Shull reported a roentgenological review of 71 cases.[76] Recently the United States Public Health Service [77] has published several reports of dust control methods in asbestos plants and of the industry in general.

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tention to silicotic fibrosis as an etiologic factor in cardiac enlargement and break-down. While this may be true, it is very difficult to prove, and one raises the question as to whether there may not be some other etiologic factor operative coincidentally. If such were true, silicosis might be regarded as a major factor without sufficient evidence.

The Erythrocyte Sedimentation Reaction in Silicosis

There is some evidence [78] to show that the sedimentation rate in silicosis is increased. I have not had sufficient experience to allow me to state whether it is increased in individuals without active infection. Preliminary observations would seem to justify the following statement, however. In the absence of clinical or roentgen evidence of active infection, in workers showing roentgen evidence of silicosis or of silicosis with healed infection, I have rarely found an increased sedimentation rate. If this observation is subsequently substantiated, the sedimentation rate will become an excellent test to use in conjunction with other clinical studies to evaluate the activity or quiescence of a given lung lesion.

Asbestosis

The Asbestos Industry.[49] — This industry has grown extensively during the past twenty years. This has caused attention to be centered around it as a factor in industrial disease, mainly, however, because of some very unusual features found in the lungs of those exposed to the inhalation of the dust. The first important one is the fact that pneumoconiosis is produced in the workers apparently by silicates instead of directly by silica. This fact will, no doubt, lead to further interesting studies of industries in which silicates predominate in the dusts. There is some confusion in regard to the action of silicates in the production of pneumoconiosis, es-

pecially as certain of them, like clay and kaolin, are supposed by some authorities to retard the action of silica. In addition to these, talc, French chalk, soap stone, pumice, and many other silicate substances are extensively used in various industries. Another unusual feature of pneumoconiosis in asbestos workers is, first, the ante-mortem and autopsy finding in the lungs of some very interesting, unusual, and characteristic mineral deposits, and still another is a progression of fibrosis and roentgenographic appearances differing from those of most injurious dust producing industries. Numerous articles have appeared on the subject during the past four years. The most comprehensive one has been that of Merewether and Price,[79] supplemented by Merewether,[80] in 1930, and again in 1934,[81] in a general review of all phases of the asbestos industry in Great Britain and a resume of the results of their examinations of 363 asbestos workers. Prior to 1928, there were available records in medical literature of only 2 deaths among employees in the factories, but since then, a considerable number have been reported. The importance of the condition and its differences from other forms of pneumoconiosis seem to warrant a general acceptance of the term, 'asbestosis.'

Lynch and Smith,[82, 83] in their reports, have been the first in this country, at least in medical literature, to deal specifically with the subject. One of their cases, a negro, died as a result of a gunshot injury after working twenty-eight months in a mill during a period of three years. His lungs showed definite interlobular, perivascular, and peribronchial fibrosis, and also the various asbestosis bodies. The second case, also a negro, died of lobar pneumonia after four and a half years of continuous work in a mill. His lungs showed, everywhere, a definite increase of young fibrous tissue, in addition to the various asbestos bodies.

In view of the meagre number of complete autopsy re-

ports in this country, the following report of Lynch and Smith[83] is given in its entirety:

Autopsy

The body had been embalmed prior to the autopsy. There was marked subcutaneous edema, especially of the feet and legs to above the knees, and some of the hands and forearms. The toes and fingers were stubby, but there was no cyanosis, perhaps interfered with by the embalming. The sternum was flattened above and definitely depressed over the epigastrium.

The pericardium and heart were definitely displaced to the left, the apex well outside the nipple line at about the fifth interspace. The heart weighed 343 gm. empty, was definitely broadened but not lengthened, the right heart furnishing the breadth, the left, not enlarged. The right cavities were well enlarged and open, the left ventricle closed. The right myocardium measured 8 to 10 mm. in thickness well up from the apex toward the pulmonary orifice. Microscopically the muscle fibres were not of uniform size, and there was generally a distinct enlargement of those of the right. They were of indistinct outlines and striations, and those of the right ventricle were frequently vacuolated. There was an increase of stroma, and the veins, particularly of the right, were definitely distended with blood.

The aorta was the seat of a minor atheroma, and the whole vascular system, outside the heart, was in good condition.

Both pleural sacs were completely obliterated by old adhesions, usually very dense, especially over the whole right lung and the upper and lower left. The diaphragm was up to about the fourth rib on the left but was low on the right. The left lung was retracted well over to the outer wall and high, occupying about one-half to two-thirds of the usual space. The pleura generally was thick and cartilage-like, especially over the apex and base. The lung was coarse,

leathery, nodular and lumpy over the upper half and the base, the middle being more spongy and air-bearing. The vessels and bronchi were very prominent emphysematous bullae in the parenchyma especially in the apex where there was a group or chain of honey-combed sacs. The interlobar pleura was obliterated. The bronchi appeared congested. The hilum lymph nodes were inconspicuous, did not appear enlarged, and were smoky black. There was no evidence of tuberculosis.

The right lung was large, filling the whole right chest and encroaching some toward the left. Its whole pleura was very thick and cartilage-like. The interlobar pleura was sealed outwardly, but was open, a clean membrane presenting, between the adjacent lobes. The lung was very much like its fellow generally, the upper lobe densely fibrous and lumpy, vessels and bronchi prominent, and a mass of emphysematous bullae in the apex. The middle lobe was not so fibrous, nodular or lumpy, and had prominent bronchi and air bullae. Both lungs, especially the right, bore much frothy fluid, which, expressed and examined, revealed numerous asbestosis bodies, free, in sheaves or clumps, and ingested by giant cells. The two ends were often engulfed by different giant cells. There were also a few dust cells, with fine black granular pigment, and masses of a yellowish amorphous substance, of the color of the asbestosis bodies, in cellular debris.

Microscopically there was an extreme grade of hyalinizing fibrosis of the lungs, universally but irregularly distributed. The pleura was thick and fibrous, and there was marked interlobular fibrosis. Scattered here and there were irregularly rounded areas of hyaline fibrous tissue, in somewhat laminated form, within which were masses of greenish-black granular substance. Here were also areas of liquefaction and calcification in the centre of these hyaline nodules. A large part of the alveoli were obliterated or virtually so. Some

lobules remained open, the sacs having thick fibrous walls. In these open alveoli, the epithelium was sometimes cuboidal and there were fairly numerous large round phagocytes, some with a group of nuclei, some mononuclear. These macrophages contained black or greenish-black or brownish granular pigment and an occasional asbestosis body. Where the lung was less fibrous, there were young connective-tissue cells and lymphocytic accumulations. The bronchioles were dilated and their walls thick and fibrous. In some areas, the lobules of alveoli showed marked emphysema, large empty sacs with thin walls. Asbestosis bodies in typical forms, with a variety of architectural figures, yellowish-brown, clubbed, dumb-bell and rod forms, were to be found widespread, singly or in groups within giant cells in the alveoli of less fibrous areas, singly in the alveolar walls and interlobular tissues showing the younger fibrosis. Associated with them was much granular substance of the same color, as if from disintegrated asbestosis bodies. Besides these pigments, there was much ordinary black anthracotic material around the vessels of the interlobular tissues. The interlobar pleura was especially thick. The large bronchi were practically normal. The peribronchial lymph nodes were the seat of marked fibrosis, edema, atrophy of follicles, and accumulation of masses of black and yellowish-brown granular pigment, of the same order as that in the lung.

The liver was grossly and microscopically in a state of extreme chronic passive congestion, which state, in lesser degree, was conspicuous in spleen, kidneys, and other viscera.

More and more data of this type are becoming available as recorded in the more recent contributions of Merewether,[81] Lanza, et al.,[84] Lanza,[85] and Shull.[86]

Symptomatology. — The symptom-complex of pulmonary asbestosis may be of interest to roentgenologists, especially because the condition is a rather new one in the group of

pneumoconioses, or, rather, an old one come to light and extensively studied. We have made a composite grouping of symptoms assembled from the reports of Wood,[87] of his 15 cases, Haddow,[88] Stewart and Haddow,[89] Soper,[90] and Wood and Gloyne,[91] including 22 cases additional to those reported by Wood, as follows :

(1) *Onset.* This is variable and depends upon the exact occupation and the quantity of dust inhaled. Both are important, for the individual may not be engaged in a very dusty duty, but may be working in a room made very dusty by another procedure in the manufacturing process. Wood states that symptoms may appear comparatively early and gives, as the time limits, one and fourteen years. Haddow places the average period at about five years, and states that the condition is usually recognized first during or after an attack of influenza or winter cold. Cases are usually better in summer.

(2) *Cough.* This is a very variable symptom, sometimes nearly or entirely absent and seldom excessive.

(3) *Expectoration.* This is moderate when present, but is usually lacking except during bronchitic attacks.

(4) *Anorexia.* This is a rather constant late symptom. Haddow regards it as an indication to stop work. Individuals may then live for several years, but become progressively weaker, more emaciated, and exhausted, until pneumonia or bronchitis brings death.

(5) *Dyspnea.* This is the most striking symptom and practically the most important one. It is progressive as in other forms of pneumoconiosis, whether the individual stops work or not. Wood and Gloyne speak of it as 'a terrible tightness of the chest,' which is very expressive. While dyspnea is due primarily to inelasticity of the lungs, which is always beautifully demonstrated by roentgenoscopic observations of the diaphragmatic excursion, it is also, in a measure,

a result of interference with blood supply. Therefore, it is slow and insidious in its development, until, finally, the lungs are able to accomplish no more than just sufficient oxygenation of the blood to sustain life.

(6) *Cyanosis.* This is a very frequent late manifestation.

(7) *Weight Loss and Emaciation.* These manifestations are very striking in the late stages. They are out of all proportion to the physical signs, and thus differ from tuberculosis alone.

All of these symptoms are more or less those found in other forms of pneumoconiosis, and a symptomatology largely applicable to all forms is placed here for emphasis on this particular one. The last three symptoms are to be emphasized in connection with the same pulverizing industry.

There is an interesting skin lesion found in connection with the asbestos industry known as the asbestos corn. It has been mentioned particularly by Gloyne and by Soper, among the writers on pulmonary asbestosis. The fine asbestos fibers penetrate the superficial layers of the epiderm like thistle thorns and produce small corns or hypertrophies of the epiderm around them. No asbestosis bodies have ever been found in these lesions.

Roentgenologic Considerations.[49] — Comparatively few of the report on pulmonary asbestosis contain roentgenographic findings, and in many of those which do, the descriptions are not so scientifically made as to permit of easy comprehension or interpretation. Most writers who describe the appearances or mention them casually seem to regard them as most unusual or characteristic of the occupation, notably Wood,[87] Wood and Gloyne,[91] and Merewether.[80] It is true that they are unusual, but we would hardly regard them as characteristic for the reason that other conditions induce quite similar roentgenographic appearances. In all industrial pneumoconioses in which the roentgenographic appearances deviate

from the stereotyped description first given to those of the hard rock miners, one must become familiar with those of the industry and correlate them with diaphragmatic excursion, clinical findings and degree of disability represented before being able to interpret the findings for that particular industry. For example, from the roentgenograms of 17 asbestos factory employees, examined by Pancoast in 1917 and reported by us in 1926,[92] the appearances were not regarded as of any particular moment, but simply as minor changes such as are frequently found in connection with slowly fibrosing dusts. On reviewing these roentgenograms with Dr. J.V.Sparks,[93] who made the roentgenological studies of Wood's cases, referring to our records of the diaphragmatic measurements, and with a knowledge of the peculiarities of the asbestos industry, particularly from the standpoint of roentgenographic appearances, we at once realized that our first interpretations were based upon an entirely erroneous conception. It is questionable whether anyone viewing a number of roentgenograms of atypical pneumoconiosis representing several industries, could pick out the ones of asbestosis subjects alone. All of the cases represented in the series just mentioned were only moderately advanced. In some roentgenograms of asbestos factory workers seen with Dr.Lanza,[94] we noted that there was a direct progression into a terminal diffuse fibrosis without any nodular predominance. This terminal state seemed to have been reached in about twenty years, on an average. Nevertheless, in some instances, the nodular fibrosis was quite evident.

Wood [87] has given a very excellent description of the roentgenographic findings in the 15 cases examined, and this has been corroborated by Soper [90] in 1 case, by Oliver [95] and by Wood and Gloyne.[91] Our interpretation of these findings was that there had been a rather predominant early interstitial fibrosis, with, later on, an associated soft nodular fibrosis.

The transition to the third stage, if we should adhere to that term, or at least to the terminal stage, would seem to have been mainly through a continuance of the interstitial predominance with very little prominence of nodular fibrosis. The early interstitial predominance was described as resembling a ground glass appearance.

On reviewing our own roentgenograms with Dr.Sparks and comparing them with the excellent roentgenograms of some of Wood's cases, we found a great similarity in the appearances. The early evidence of the condition was a faint homogeneous haze in the lower part of the mid-lung field which seemed to correspond to the appearance we have previously suggested as an early predominance of interstitial fibrosis. This haze spreads through the lower one-half or two-thirds of the lung field and may almost obscure the domes of the diaphragm later on, with very little evidence of nodular fibrosis. With it, there is a very decided restriction in movement of the domes of the diaphragm without the appearance of pleural adhesions, which seems to be proof of the general fibrotic nature of the process. Thickened pleura does exist, of course, as in any fairly well advanced case of pneumoconiosis. The most unusual feature of the condition seemed to us to be the fact that, in both sets of roentgenograms, the appearance seemed to begin more often on the *left* side than the right, and progressed more on that side. This is the reverse of all other occupations that we have studied. Merewether [80] states that it begins on the right side, as one would expect, and the cases we observed may have been examples of the exceptions which are occasionally observed in any industrial pneumoconiosis. We noted the same tendency to obliteration of the left cardiac border as mentioned by Wood, because of the diffuse fibrosis and the prominent trunk shadows. Wood states that the late stage may or may not show consolidations. Wood and Gloyne [91] state that

the ground glass appearance in late cases, on careful analysis, resolves itself into a fine mottling and linear shadows which, toward the base, may look like cobwebs.

It is evident that the nature of these changes in the lungs requires the best type of roentgenograms for their portrayal. Also, the seriousness of what may seem to be minor changes to the uninitiated requires a roentgenoscopic study of diaphragmatic movements to make one realize the actual state of disability present. It is evident also, as Merewether states,[80] that an opinion as to the degree and intensity of an asbestosis fibrosis, based upon a comparison of roentgenographic changes with those shown in standard silicosis films, will be an underestimate.

More recently, Merewether [81] and Shull [86] have not emphasized on which side the process is most evident in the beginning or late in the disease. This coincides with my more recent experience. The disease process may be diffuse, bilateral, or unilateral, but is practically always limited to the lower half or lower two-thirds of the lung field.

Of the 36 cases examined by Merewether and Price, 133 had roentgenographic studies. Of these, 52 were said to have presented appearances of diffuse fibrosis and 22 were suspicious.

Merewether [81] says that approximately seven years must elapse between the commencement of exposure and the production of a serious degree of asbestosis. This space of time includes not only the trapping period of the fibrosis-producing dose of dust, but also the maturation period of the fibrosis, which periods, of course, overlap. The existence of this fibrosis-producing period accounts for the fact that no appreciable number of asbestosis cases are discovered until the second five years of employment. Relatively few cases mature in this minimum period of seven years ; many, even in the more dusty processes, require eleven years. When

fibrosis of serious import has matured, the worker is unduly short of breath on any extra exertion, has a slight cyanosis of the lips, and a little dry cough, mostly in the mornings. He still, however, is quite able to work and, usually, is not concerned about his state of health. Many workers, in fact, will not admit symptoms of any kind at this stage.

Discussion of the Nature of the Roentgenologic Appearances seen in Asbestosis. — From my experience, the roentgenologic findings in asbestosis are largely limited to the lower half or lower two-thirds of the lung fields, whereas in silicosis, the opposite obtains. In asbestosis, the lesion may be bilateral or largely unilateral. Emphysema, blebs, or bullae always accompany well established changes in asbestosis and occur in the upper portions of the lung fields; whereas, in silicosis, the lower lung fields, to a large extent, are the site of these changes.

I have found it extremely difficult to determine the early manifestations of asbestosis. I believe an explanation for this would include the lack of an adequate pre-employment examination, in which a careful fluoroscopic study of the respiratory and cardiac movements is made. It seems to me that fluoroscopy, directing special attention to costal and diaphragmatic expansion, is essential in the determination of early changes in asbestosis.

If one can assume that the pathological process is due to changes secondary to the lodgment of asbestos fibers in the smaller bronchioles, the roentgen findings might be explained as follows. Disturbance in the roentgen appearance would not be seen except following the lodgment of the fiber in the smaller bronchioles. Sufficient time would have to elapse until a definite collar of fibrosis had become manifest so extensively that it would limit the ingress and egress of air into the lung areas distal to the point of obstruction.



FIGURE 27

Asbestosis, early manifestations indicated by the beginning obliteration of the costophrenic sulcus. Moderate hypoventilation of the lower lung fields due to thickening of the parietal pleura and slight thickening of the interlobar pleura.

In such damaged bronchioles, it is justifiable to anticipate that the lack of ventilation and drainage, distal to the point of obstruction, might become an ideal site for the development of infection or atelectasis. The infection, in turn, would increase the amount of serum protein in the tissues and assist, or stimulate, a pre-existing tendency for fibrosis. The fibrosis, itself, might limit the amount of normal lymph flow and, in succession, the infection would extend to and involve neighboring areas. When a sufficient amount of tissue had become involved, one might be able to demonstrate beginning changes in the roentgenogram, such as beginning obliteration of the costophrenic sulcus and fluoroscopically, some limitation of costal and diaphragmatic expansion. (Figure 27) I should assume, when such an appearance is observed in the roentgenogram, that the process might be regarded as moderately advanced.

The next step in the pathological process would seem to include an extension of the affection to the periphery. With involvement of the periphery, there is a definite limitation in the respiratory movements, which would be extremely slight in the early case of asbestosis but marked in the more advanced degrees of the disease. (Figures 28 and 29) The ribs are somewhat fixed and in thin individuals, they are dependent. In fat persons, they change very little. The interspaces may become quite narrow.

In the consideration of the healthy chest above, I called attention to the importance of the respiratory movements in lymph flow, and also to the fact that any restriction of these movements would tend to limit the flow of lymph. Any limitation of the flow of lymph increases the development of pre-existing pathologic changes. It was, likewise, emphasized above, that thickening of the pleura occurs to a greater extent in the lower thoracic region than it does in the upper, the explanation for this being the powerful costal and dia-

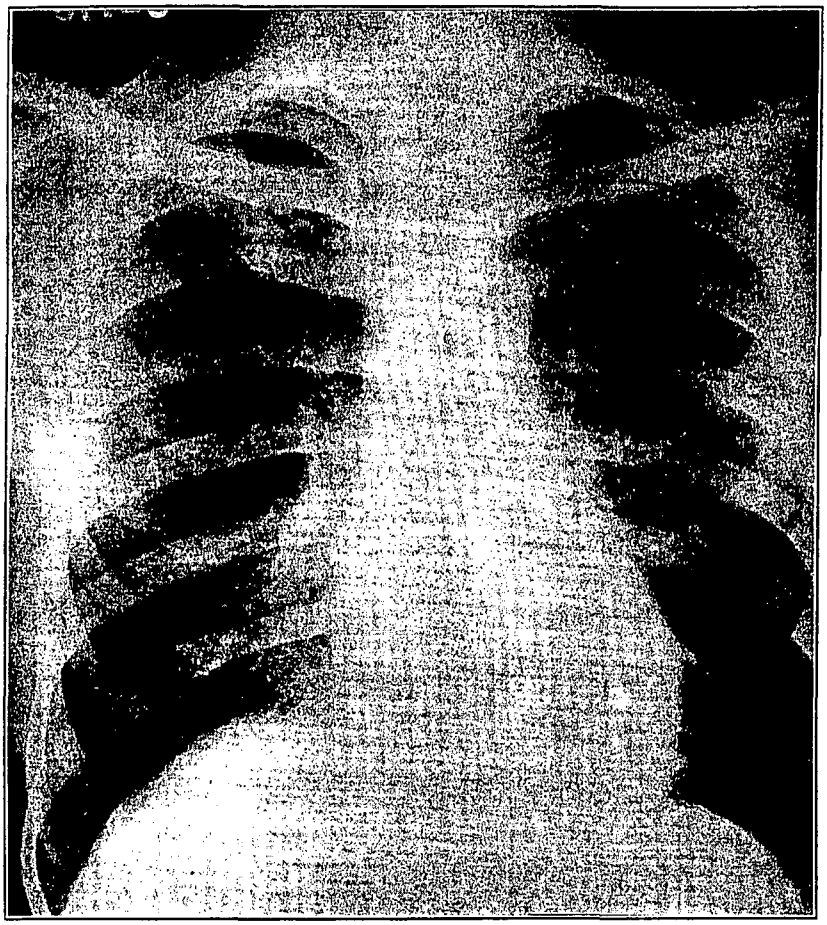


FIGURE 28

Man aged 41 ; silk weaver for twenty years, asbestos weaver for five years. The roentgenographic findings are within the limits of a healthy chest.

phragmatic movements in the lower chest and the relative fixation of the upper chest.

Almost every autopsy protocol has called attention to the

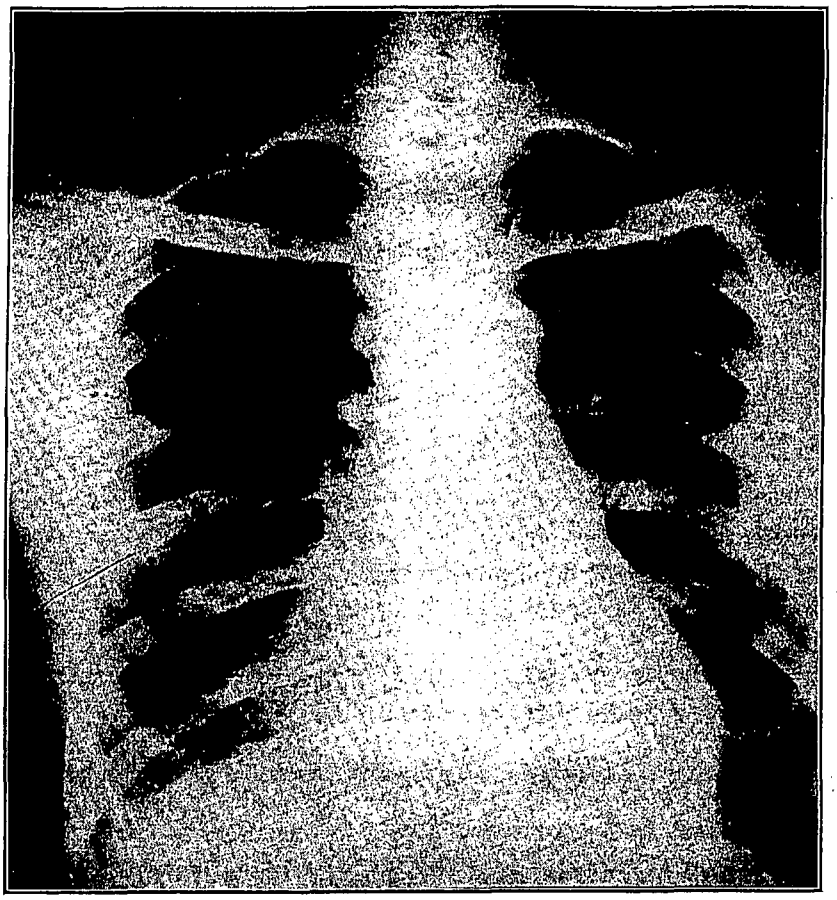


FIGURE 29

Asbestosis moderately advanced. Same individual as illustrated in Fig. 28. Examination made five years after that illustrated in Fig. 28. Note the relatively marked changes in the right lower lobe, which include thickening of the parietal and diaphragmatic pleura, obliteration of the costophrenic sulcus, slight thickening of the interlobar pleura, some narrowing of the interspaces and enlargement of the cardiac silhouette. There is evidence of hyperventilation in the upper lung fields.

marked thickening of the pleura and occasional thickening of the pleuropericardium. It is my feeling that these definite changes in the pleura can be adequately explained upon the basis of an underlying infection of the lung, extending to the pleura with subsequent thickening of the structure, due to the powerful respiratory movements and cardiac pulsations. (Figures 30 and 31)

The haziness of the lung fields, called attention to by almost every writer, can be explained by pleural thickening and a lack of ventilation. One should be careful in distinguishing between the shadow caused by pleural thickening and that resulting from the subcostal muscles. This has been discussed under the healthy chest.

I have been forced to place less and less importance upon observations concerning the trunk or vascular markings. These shadows are so changeable with variations in phases of respiration and cardiac pulsations that, unless one is reexamining patients and employing critical technics, such as making exposures during forced inspiration and during the systolic or diastolic phase of the cardiac cycle, one is absolutely confounded with changes in the size of the shadows, entirely within physiologic limits.

However, when changes develop sufficiently, one can see disturbance in the movements of the domes of the diaphragm and of the ribs, and obliteration of the costophrenic sulci. From this point on, the pathologic process in the lungs seems to advance fairly rapidly, exactly as one would expect it to do. This can readily be demonstrated by comparing the vertical diameter of the thorax in serial examinations of an individual who has a well developed asbestosis. If a period of at least five years is allowed to elapse, the vertical dimension is materially reduced. This observation corresponds to the limitation of the movement of the domes of the diaphragm, and, in a rough way, indicates the ability of the individual to



FIGURE 30

Asbestosis moderately advanced. Close-up of the right lower lung field in the individual illustrated in Fig. 29.

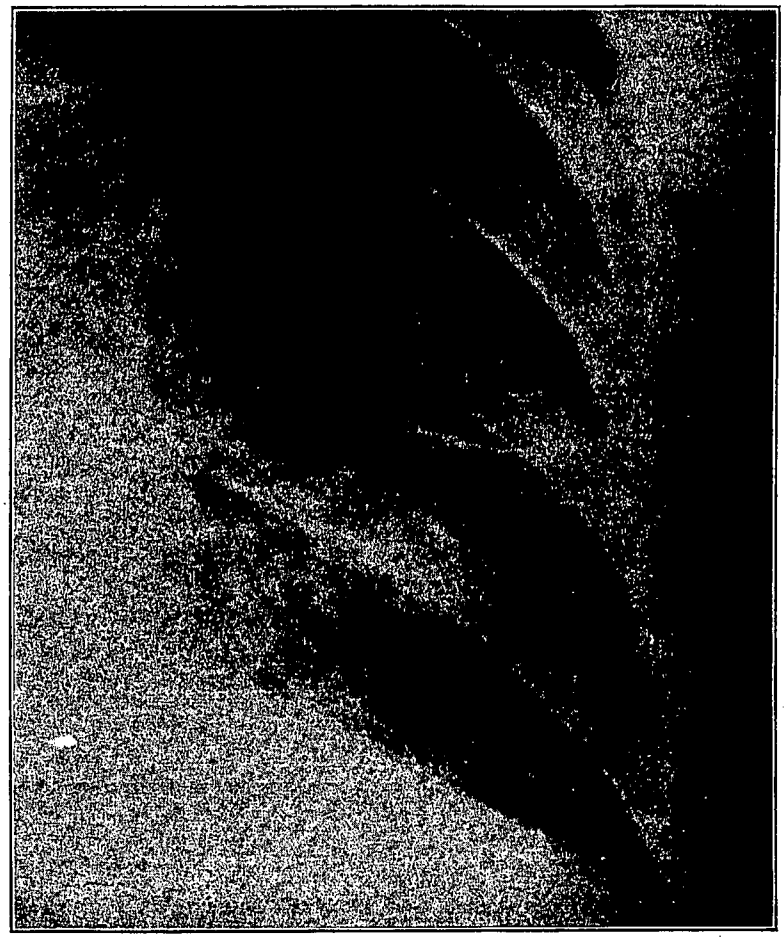


FIGURE 31

Asbestosis moderately advanced. Note the position of the ribs, the hypoventilation of the lung fields and thickening of the parietal pleura, which, in turn, are responsible for the ground-glass appearance of the lung fields. The interlobar pleura between the upper and lower lobes is thickened. From its position, one can readily see the extent of scar contraction already existent in the right lower lobe. The vascular trunk shadows have largely lost their identity.

ventilate the lung fields. The shadows are more pronounced in the bases and diminish as they approach the upper third of the lung field.

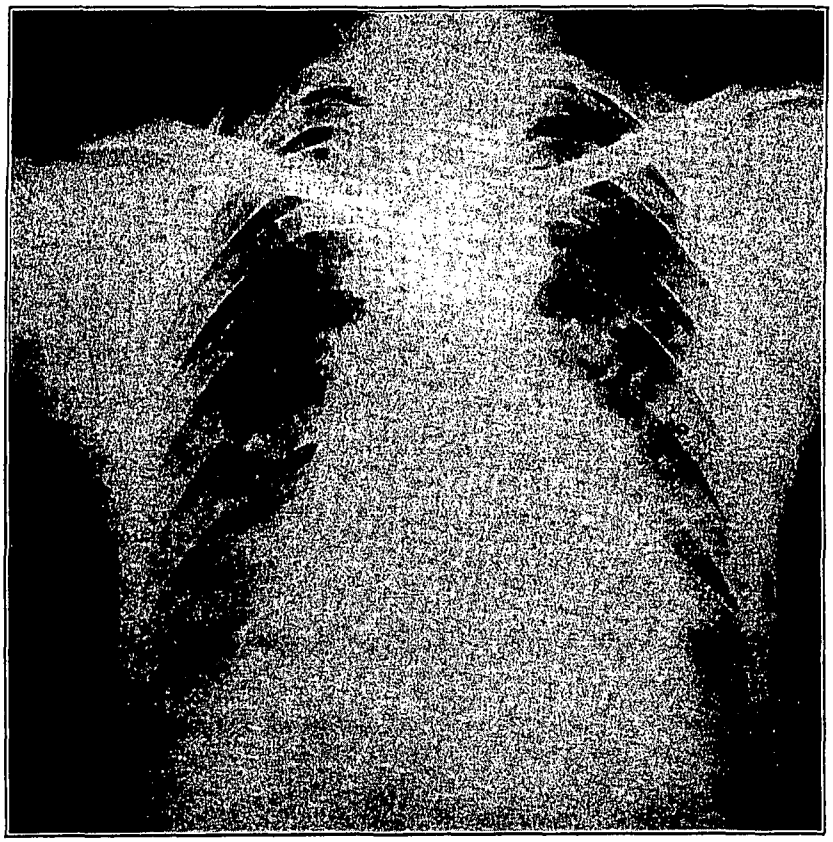


FIGURE 32

Asbestosis markedly advanced. The lesion occupies the lower lung fields and there is hyperventilation in the upper lung fields. Note the indistinct outline of the cardiac silhouette. This is probably due to thickening of the pleuropericardium and superimposed shadows in the lung fields. Note the dependent position of the ribs.

My comments thus far have been limited entirely to the lungs and pleura. The heart and pericardium also suffer in asbestosis, much more so in this occupational disease, in fact, than in any dust hazard with which I am acquainted. With a moderately advanced degree of asbestosis, the shadow of the cardiac outline is obscured by the shadows of superimposed lung structures and involvement of the pleuropericardium. (Figure 32) One rarely sees the cardiac shadow sharply demarcated, as observed in ordinary roentgenograms. The pleuropericardium becomes thickened and, in some instances, markedly so, for the same reason that the pleura over the lower lung fields and the domes of the diaphragm become thickened. Pulsations of the heart and infections extending to the pleura in this region play a major role, I believe, in producing the marked thickening of the pleuropericardium. These changes and those of the lung probably account for the right-sided hypertrophy and ultimate decompensation of the heart. In some instances, the cardiac silhouette is quite enlarged. Realizing, therefore, that ventilation of the lungs is seriously interfered with and that cardiac action is impaired, one can readily understand why these patients are embarrassed with dyspnea, weakness, and cyanosis.

In my present state of knowledge, if I were called upon to make a classification of asbestosis from a roentgenographic standpoint, I should include only two stages ; asbestosis moderately advanced, and asbestosis markedly advanced ; or asbestosis, first and second degree. I feel quite certain that the roentgenologic diagnosis of the clinically early stage of asbestosis is not entirely reliable. (Figure 33)

Prognosis. — It seems to be the rather general opinion that the prognosis in pulmonary asbestosis among factory workers is to be regarded as grave without adequate protection, in a large number of cases, among those who work



FIGURE 33

Asbestosis, an unusual type. This may be due to the fact that the individual has had some silica exposure. The shadows in the middle third of the right lung field are not typically nodular.

where there is great concentration of dust. The condition is apparently progressive, even after cessation of occupation. Merewether and Price [79] state that, with continued exposure to high concentration dust, fibrosis may be fully developed

in from seven to nine years, and that death may result in about thirteen years. With less concentration, fibrosis may not be fully developed for fifteen, twenty, or twenty-five years. Inhalation of dust in high concentration results in a more marked degree of fibrosis in a shorter time than when the concentration is lower.

There is no disease among the industrial hazards in which it is more important to have serial examinations than in the asbestos industry. Almost every investigator has been hampered by either no pre-employment roentgenologic examinations of the chest or relatively few opportunities for re-examinations of the individual exposed to asbestos dust.

Shull [86] states that it would seem that improvement could be expected in the early cases, but that, as the disease progresses, improvement is less likely. Sparks [96] feels that it is too early in the study of this disease to say very much on the subject. Lanza [85] believes that it is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor that infection seems to be so closely and intimately associated with asbestosis as with silicosis.

My experience in this condition is also limited, but I feel quite certain that, when asbestosis can be diagnosed by roentgenologic examination, the condition is likely to become progressive, due to the important role exerted by fixation of the lung structures secondary to the pleural involvement. This can be readily demonstrated in serial roentgenograms, even in the absence of fine lung detail. The striking feature is the reduction of the perpendicular diameter of the chest.

Predisposition to Tuberculosis. — The condition of pulmonary asbestosis being a new child in the pneumoconiosis family, a great amount of interest centers around the incidence of tuberculosis. Among the early case reports referred to in the beginning of this occupational section, pulmonary tuberculosis was reported as an accompanying condition in a

few instances. Cooke's case had advanced tuberculosis. Simson reported 1 case which died with tuberculosis and 1 without evidences of the infection. He thought that we could not as yet formulate any definite opinion as to the frequency of the predisposition. Simson quoted Hoffman [97] as reporting 13 deaths among asbestos workers, 3 of whom had tuberculosis. He also quoted Collis [98] as reporting 5 deaths from tuberculosis among less than 40 workers at a factory. Wood and Gloyne [91] referred to 3 cases showing evidences of tuberculosis at autopsy. Haddow [88] observed many asbestosis cases, among whom 4 died. He did not believe that the condition predisposed to the infection, at least not as a rule. Merewether and Price [79] stated that asbestosis differed from silicosis in the distribution of the fibrous tissue, in more rapid development, in roentgenographic features, and, possibly, in a *lessened susceptibility to tuberculosis.* Merewether [80, 81] states that, out of 374 cases examined, evidence of active tuberculosis was found in 3. He thinks that, while there is no outstanding susceptibility proved, the question has not been fully settled as yet. I believe, however, that when tuberculosis does complicate asbestosis, that one can readily suspect or diagnose the tuberculous lesion with a fair degree of accuracy, due to the fact that the tuberculous process often occurs in the upper lobes, where there is usually very little evidence of asbestosis. If a basal tuberculous process were superimposed upon asbestosis, it would be very difficult. if not impossible, to diagnose it. (Figure 34)

Differential Diagnosis. — Passive Congestion of the Lungs as a Result of Cardiac Decompensation. — In this condition, the enlargement of the heart shadow, the abnormalities of the cardiac silhouette, especially in cases of mitral stenosis, and the clinical picture serve to establish the presence or absence of passive congestion. If asbestosis is absent, the appearances are likely to disappear with restoration of com-

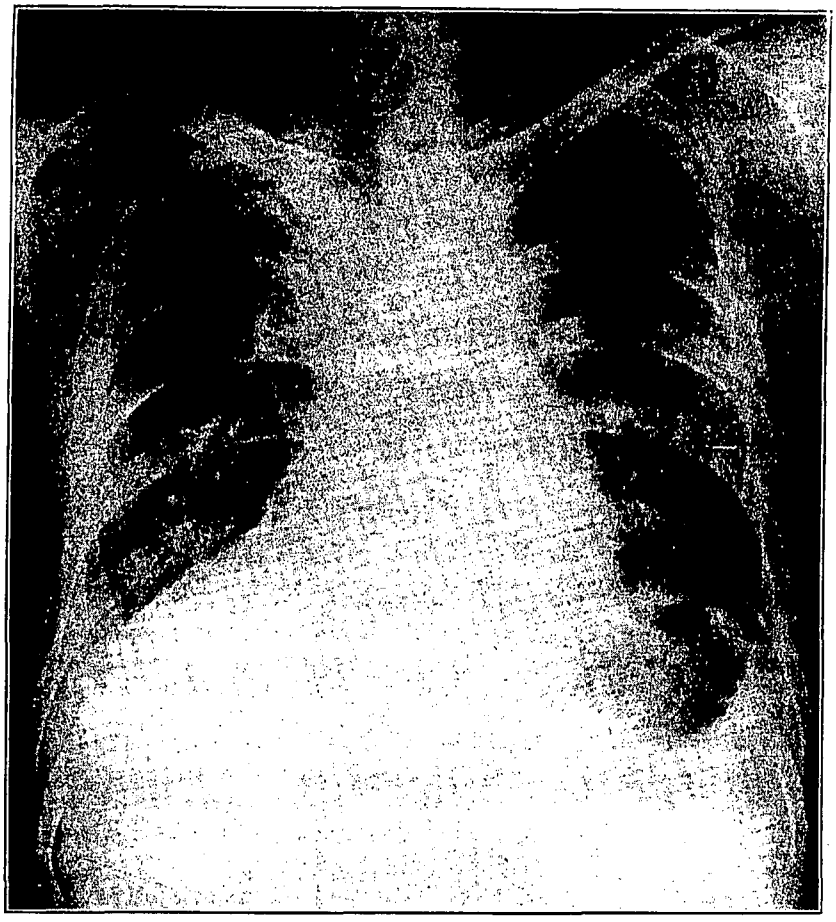


FIGURE 34

Asbestosis with a complicating tuberculous lesion in the right upper lobe. It is entirely possible that tuberculosis may account for the entire appearance found in this individual, but it was subsequently shown that this individual not only had tuberculosis but also had asbestosis in the lower lung fields.

pensation, but not altogether in long-standing cases. Marked emphysema is usually associated with asbestosis and is apt to cause a certain amount of rotation of the heart, so that the sagittal roentgenogram does not convey a direct impression of cardiac size or shape. The lateral view is always necessary to clear up the diagnosis. Any phase of asbestosis is likely to have its roentgenographic appearance greatly accentuated by passive congestion.

Advanced Bilateral Bronchiectasis. — The dilated bronchi, retained secretions and associated tracheobronchitis characteristic of this condition will produce the appearance of greatly accentuated trunk shadows, which may simulate that of the moderately advanced phase of asbestosis.

Other conditions that simulate asbestosis include polycythemia or erythremia and infiltrating malignant metastases, especially those extending from the mediastinum.

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The author has drawn rather freely from a number of the writings in which he has collaborated, in view of the fact that their observations largely represent his present views. When present opinions have differed, a discussion of the reasons for the change is included in the text.

IV. PATHOLOGY

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INTRODUCTION

DEFINITIONS

THE term *silicosis* will be used in this chapter to denote the changes which take place in the body tissues as a result of the inhalation of dust containing silicon dioxide, generally referred to in this connection as "free silica." The International Conference held at Johannesburg[1] in 1930 proposed in interpreting this term that the following conditions be satisfied, viz., that the silica must reach the lungs (a) in a chemically uncombined condition, although it might be mixed with other dusts; (b) in fine particles of the order of less than 10 microns in diameter; (c) in sufficient amount, and over a certain period of time.

For some years pathologists regarded free silica as the only siliceous dust likely to produce the disease, but recently "combined silica" or various mineral silicates found in industrial dusts have been included in the indictment and a new term, *silicatosis,* has come into use to denote the types of disease thus produced.

The chief example of this new group of diseases has already become sufficiently important to receive a special designation, *asbestosis,* a disease resulting from the inhalation of the dust of asbestos, which contains the silicates of iron and magnesium.

As the diseases caused by these various substances differ

in their pathology, it will be necessary to describe them separately, but before doing so certain general considerations from a pathologist's point of view require discussion. Just as a chronic infective disease such as tuberculosis is regarded from the three-fold point of view of the organism, the portals of entry and the tissue reaction, so in the study of a pulmonary dust disease the nature of the dust, the conditions of its entry into the body, and the tissue reaction which ensues all need separate consideration.

Nature of the Dust. Mavrogordato [2] (1930) has classified the dusts which enter the lung into four types: (1) Dust that is harmless; (2) dust that is usually harmless but which may contribute to the lung mischief when inhaled in association with a harmful dust; (3) dust that is usually harmless and may actually have a prophylactic value when inhaled in association with a harmful dust; (4) dust that is harmful. The first group does not concern us here. In the second group may be placed such dusts as iron ore and coal dust in association with silica. The third group is a doubtful one. At the moment one would not feel justified in placing any dust in this category with certainty, for the pneumoconioses take many years to develop and surprises are not uncommon. The fourth and last group must include nowadays not only free but also combined silica.

Free silica occurs mostly in the form of mineral quartz. In the British Silicosis Compensation Scheme of 1928 it is referred to as silica rock, quartzose sand or 'any dry deposit or dry residue of silica.'

Combined silica includes a large number of silicates which tend to increase with industry. The commonest of those in use which are under suspicion as possible producers of pulmonary changes are enumerated by Middleton (1936).[20]

The size of the dust particle is important. Only very small particles reach the lung tissues. Probably the majority, as

Mavrogordato suggests, are about the size of the common pathogenic micro-organisms.

The shape of the particle has also a bearing on the resultant lesion. Broadly speaking there are 3 morphological types : (1) Small particles with irregular surfaces and sharp edges and diameters approximately equal. This is the common shape for quartz particles. (2) Long fibres of asbestos, which may extend up to 100 microns in length. Even if we regard the particle as inert, size and shape are of great importance because they determine the penetrating power of the particle. The long needle-like asbestos fibre readily "engages" in the respiratory bronchiole and easily comes to rest athwart the lumen of any narrow passage it may enter, whereas the small silica particle whose diameters do not differ greatly can readily penetrate to the peribronchial lymph spaces and the mediastinal glands. (3) Minute needles of fibrous sericite — a hydrous silicate of aluminium and potassium. These vary from 2 to 5 microns in length and have been described by W.R. Jones [3] (1933) in silicotic lungs as occurring in 'piled-up bundles of needles,' hundreds of which are present, he states, for every grain of quartz that can be recognised. Although sericite had been previously described, it was not regarded as of great importance until the work of Jones. He regarded it, rather than the uncombined silica or quartz, as the important causative factor in silicosis. At the present moment the matter must still be regarded as sub judice. Obviously a series of carefully controlled pathological observations is needed in workers who have been exposed to the dust of quartz particles alone and sericite particles alone, but according to Drinker and Hatch [4] (1936) quartz free fibrous sericite is difficult to obtain. Middleton has pointed out, however, that in Britain workers exposed to sericite without uncombined silica are found in

the china clay industry and that so far there has been no high incidence of silicosis in this industry.

The experimental evidence in laboratory animals falls outside the scope of this chapter.

Non-siliceous particles are found in practically all cases of silicosis and may cause confusion. Carbon, for example, is generally present in the shape of very fine amorphous particles, but coal dust may occur as elongated sharp jagged particles often up to 10 microns in length.

In the course of recent investigations on dust sampling, Briscoe, Matthews, Holt and Sanderson[5] (1937) noted that dust which had been exposed for some time to the moist atmosphere of mines lost an unusually large amount of water on ignition, in striking contrast with the relatively small loss of weight on drying at 100°C, indicating that the dust had taken into firm combination a relatively large amount of water. In other words the comminution of the minerals had exposed fractured surfaces on which, at numerous points, there existed free and unsatisfied valency forces whereby water was attracted and held as water of constitution in the hydrated particle. Hydration of this nature can apparently liberate bases held in the crystal lattice. The writers found for instance that alkali was liberated in amounts equivalent to about one-sixth of the total alkalis present in the minerals. Obviously important new possibilities are opened up by these researches. Freshly formed dangerous dusts yield to water both alkali and soluble silica in larger amounts than do 'dead dusts' — i.e., dusts which have been exposed to air for some time.

The Portal of Entry. Probably only a small fraction of the inhaled dust reaches the pulmonary alveoli. The greater portion is deposited in various parts of the respiratory tract and expelled therefrom. In the case of asbestos workers, for

instance, the fibres can be found in the nose and throat and even occasionally in the conjunctival sac. The histological examination of the walls of the larynx, trachea and large bronchi in cases coming to autopsy usually reveals no lesions of asbestosis. Probably the same is true of silicosis though the writer knows of no investigations on the point. It may be assumed, therefore, that mechanical forces come into play to drive out the invading dust. Such are the muscular movements of the eyelids, the cleansing effect of the lachrymal secretion, the action of cilia and the outward flow of mucus in trachea and bronchi. In the case of cilia these protective phenomena have been demonstrated by Leonard Hill [6] (1928). Using the trachea of horse, ox and sheep, he found that a suspension of lamp black in Ringer's solution was carried along the mucous membrane by the cilia at a rate which might be as fast as 3 centimeters in one minute and that fine particles such as those of iron and aluminium filings were readily passed along the trachea in this way. If a small area of ciliated epithelium was damaged by injury, cautery, or various chemicals (including chemical fumes) the transport of suspended particles was greatly reduced or even inhibited altogether. Irvine [7] (1930) has suggested that repeated inhalation of silica dust may lead in the course of time to a dry bronchitis and bronchiolitis with denudation of epithelium. In this case the portal of entry is opened wide, as it were, and the penetrating dust will meet with little or no resistance. No doubt, too, some types of particles may be more readily expelled than others. These various factors may explain why different dusts seem to have a predilection for different parts of the lung.

The Tissue Reaction. Obviously the tissue reaction must, to some extent, be dependent on a variety of factors, viz., the duration or intermittent nature of the dust exposure, the size of the dose, the admixture of dusts, the age and sex

of the worker, and the condition of the respiratory passages prior to exposure. These factors take long years to observe in conjunction with employment histories and post-mortem findings and in many industries the necessary data for assessment are not yet available. Meanwhile the interaction of the dust particle and the tissues may be briefly and tentatively summarised as follows : —

(1) Many dust particles are doubtless expelled en route, but if a particle reaches the end of the journey in the distant parts of the portal of entry it "stays put." It does not multiply or become bacteriolysed like a micro-organism but remains as a more or less insoluble and irremovable irritant particle.

(2) The end of the journey for the silica particle is the peribronchial lymphoid tissue, but in the case of the asbestos fibre it is the respiratory bronchiole and the alveolus ; hence the difference in the pathological picture.

(3) Although bacteria may be found mixed with the dust, they are for the most part non-pathogenic organisms ; the dust particle can therefore be regarded as a non-infective irritant, and the tissue reaction is fibroblastic, ending in the production of the collagenous fibre.

(4) The amount of plasma exudation is minimal. The exudative reaction in the lung is characteristic of the bacterial diseases.

(5) Although we speak of the silica particle and the asbestosis fibre as being practically insoluble, many investigators believe that a colloidal silicic acid is actually formed by a slow disintegration of the particles in the lung and that the morbid processes are the result of a toxic action. There is indeed experimental evidence in the work of Gye and Purdy [8] (1922) in favour of such a view in the case of silica sol. Asbestos fibres are known to persist in the lungs of persons who have not been exposed to the dust for more than twenty years and any disintegrating action must therefore

be extremely slow. Sundius, Bygden and Bruce,[9] (1936) indeed, consider that this chemical theory of slow disintegration of dust particles is still an open question.

(6) It follows that if the dust particle is practically insoluble or only very slowly soluble, the lung must in the course of time become choked with irremovable foreign matter. This dust accumulates chiefly in the lymphatic drainage system.

(7) The process of phagocytosis, so prominent a part of the tissue reaction to bacterial diseases of the lung is much modified. Many of the particles, notably the asbestos fibre, are too large to be engulfed by a single phagocyte and a group of these cells can often be seen surrounding but not engulfing the meal which is obviously too large for them. Again, the particle even if engulfed cannot be digested and disposed of in the same way as the more soluble protoplasmic bacterium. This may be one reason why the phagocyte appears to remain so long in situ in the silicotic tissue in experimental animals. Lastly the phagocyte is for the most part a large mononuclear macrophage and not the small polymorphonuclear leucocyte from the blood stream. The latter is the phagocyte, par excellence, of pyogenic cocci in the lung.

(8) Lymphoid hyperplasia. This is one of the striking features of the tissue reaction. Small collections of lymphoid tissue are normally situated on the outside of the walls of the small bronchi but they are so small as generally to escape notice. These minute lymphoid aggregations absorb much carbon pigment and are a striking feature in the lungs of all persons exposed to the dust of large towns irrespective of silicosis. They should be distinguished from true lymphatic glands. They become markedly hypertrophied as the result of the drift of the silicotic particles into them. In the course

of time collagenous fibres are laid down and constitute the first simple silicotic nodules.

SILICOSIS

Macroscopical Appearances

As a rule the body of the silicotic is not markedly wasted unless tuberculosis has supervened. Cyanosis may be present. Deformity of the chest wall is rare, whereas in the fibroid disease of the lung of non-industrial origin which begins generally in childhood, flattening and deformity are common.

Lungs and Pleurae. On opening the thorax the lungs as a rule do not collapse. They are heavier than normal and frequently bulky. Pigment is variable in amount. In the British cases it is generally pronounced owing to the carbonaceous dust of the industrial towns and may indeed be extreme in amount. Emphysema is almost the rule, bullae at the apices being especially common, whilst all the free borders may be affected.

The essential feature of the silicotic lung, however, is the nodular fibrosis. This begins with small raised hard nodules or islets of fibrosis immediately beneath the pleura and in the peribronchial regions of the lung. The former can be felt by running the fingers lightly over the pleura and are less pigmented than those in the lung. At first the nodules are no bigger than miliary tubercles but as the disease progresses adjacent nodules coalesce into composite nodules or small irregularly shaped masses which look rather like pieces of hard black rubber embedded in the lung. They can be detected by the touch as the knife cuts through the lung substance and they stand out prominently on the cut surface as the crepitant lung tissue collapses, but a true 'gritty' feel is

only imparted to the knife, as a rule, by extreme cases such as the stone mason's lung. In this case small particles of quartz can actually be dug out of the nodule with the point of the knife. The nodules are usually more plentiful in the upper lobes where they readily tend to run together into masses of large size.

As the presence of the nodule is the essential feature for diagnosis, attempts have been made to arrive at a classification in terms of distribution and severity of the lesion. Simson and Strachan [10] (1930) in describing simple silicosis as it exists in the South African mines give the following classification which is used by the Miners Phthisis Bureau :

(1) Slight — lesions small and moderately numerous, or medium-sized to large but sparse ;
(2) Moderate — lesions numerous and small or moderately numerous and large ;
(3) Well marked — lesions numerous and large.
(4) Very well marked — lesions very numerous and large.

These authors state that in estimating the degree of nodulation present the whole lung substance has to be considered for the obvious reason that the distribution may be limited or unequal. The term small is used for islets of fibrosis with diameters up to 2 millimetres, medium from 2 to 4 millimetres, and large from 5 millimetres up to a centimetre. The term sparse is used to describe the distribution of nodules on the cut surface of the lung of the order of one nodule in a square with a side of 5 centimetres ; moderately numerous signifies one nodule per square with a 3 centimetres side, numerous when the square has a side of 2 centimetres and very numerous when the square has a side of less than 2 centimetres.

The nodule must be distinguished from calcified deposits and from the small fibrotic patches of obsolescent tubercu-

losis. As noted above, the silicotic nodule in the British cases is usually darkly pigmented, whereas calcified deposits and patches of obsolescent tuberculosis are much less so.

In long standing cases the aerated lung tissue between the nodules collapses, thus causing the groups of composite nodules to run together into the large hard, dark pigmented masses already described. These masses are commonest in the upper lobes ; they may be wedge shaped with the apex of the wedge towards the hilum like deposits of caseous tuberculosis ; they are frequently subpleural, and beneath the apical pleura are associated with emphysematous bullae, and eventually they may occupy the greater part or even the whole of the lobe. (Figures 1 and 2)

In addition to this characteristic fibrotic nodule the pleura generally shows evidence of old pleurisy in the shape of thickening, usually patchy, with obliteration of the interlobar fissures, and the production of sessile adhesions closing portions or the whole of the pleural sac. A layer of soft plastic fibrin, the result of a recent terminal pleurisy is often found at the autopsy, covering the old thickened pleura. Pleural effusion is rare.

In the lung the most important change apart from the fibrotic nodule, is the presence of emphysema. This is so common as to form almost an integral part of the picture. It occurs in the shape of bullae beneath the apical pleura, and along the thin anterior margins of the lungs, similar to the emphysematous lesion in other lung diseases. But it is seen also in another characteristic form, viz., a compensatory emphysema of the deeper portions of the lung tissue giving rise to an irregular honeycomb appearance.

The later stages of the disease are often characterized by a septic bronchitis with muco-pus plugging the bronchi, and a terminal reddening of the lower lobes from congestion



FIGURE 1

Silicotic lung showing massive nodules best marked in upper lobe.

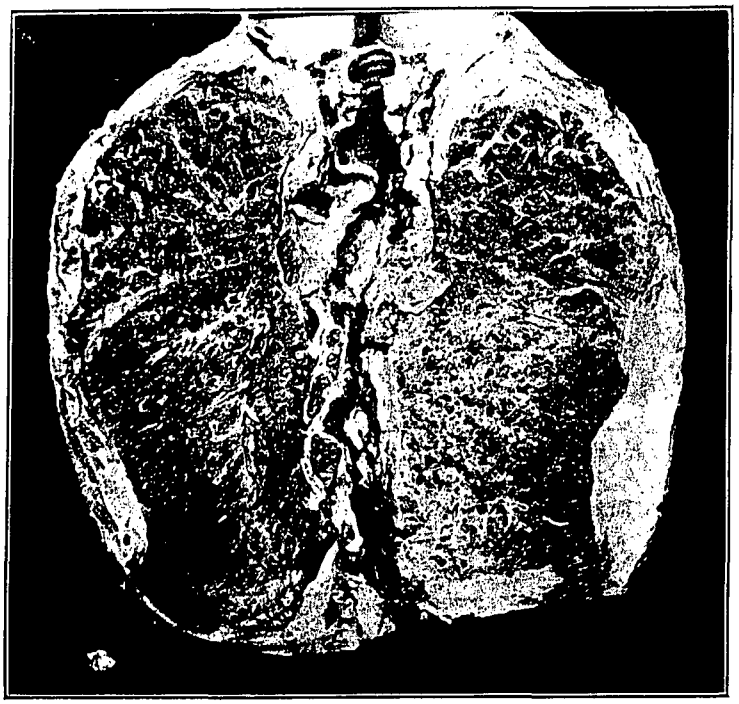


FIGURE 2

Silicotic lung showing extensive nodulation especially in lower lobes.

which may go on to a true red hepatization. These terminal conditions will be referred to again under the complications and sequelae of the silicotic lung.

In uncomplicated cases little of importance may be found in the other organs. Myocardial degeneration is not uncommon and is considered by some pathologists to be due to the obstruction in the pulmonary circulation as a result of the fibrosis, but it must be remembered that, as a rule, silicosis as seen in the autopsy room is a disease of the elderly industrial worker whose heart is likely to show signs of wear and tear in

any case. Although silicosis is believed to show a biochemical aspect there is as a rule no obvious toxic effect on distant organs in man, such as one finds in gout for instance, though lesions in the kidneys and other organs have been obtained by Gye and Purdy [8] (1922) and others in experimental animals. Chronic interstitial nephritis may be found in silicosis but there are generally other reasons for its presence, (e.g. atheroma) and one cannot with any confidence claim that such lesions are simply the result of a silicotic toxin.

Microscopical Appearances

The Silicotic Nodule. The silicotic nodule is best studied when small and discrete and for this purpose the earlier the nodule is obtained the better. The key to the situation is to be found in the minute aggregations of lymphoid tissue which are normally located, in the lungs of man, in relation to the bronchioles, saccular ducts, small branches of the blood vessels and pleura. These aggregations of lymphoid tissue have been carefully described by W.Snow Miller [11] (1937) and should be distinguished from the lymph nodes which are found in intimate relation with the cartilaginous bronchi. These small collections of lymphoid tissue are simply minute aggregates of lymph corpuscles without the organised structure of supporting framework and capsule of connective tissue which a true lymph node of the cartilaginous bronchus possesses. In relation to the bronchioles they are found lying between them and the accompanying branch of the pulmonary artery. They are also to be seen along the course of the pulmonary artery and the pulmonary vein, in the interlobular septa and beneath the pleura, probably in this case in association with minute radicles of the pulmonary veins but not necessarily so. Miller has examined the lungs of elderly persons in order to determine the distribution of this lymphoid tissue in relation to the usual carbon pigment which

accumulates in the lung with age. The masses of lymphoid tissue around the bronchioles and the branches of the pulmonary artery were found to be hypertrophied into a common mass, whilst those associated with the pulmonary veins and the venous trunks in the interlobular septa were also hypertrophied. He further noted that these masses were usually found where alveoli were in the direct line of the air current against the septa. The lymphoid masses beneath the pleura are similarly hypertrophied.

These small masses of lymphoid tissue, therefore, are the sites of the formation of the silicotic nodule, a point which has been well stressed by Strachan and Simson [10] (1930). Pathologists get very few opportunities of examining the nodule in its very early stages in man and the picture tends to be obscured by subsequent events, especially by the presence of pigment, but probably the sequence of events is as follows.

Amorphous particles of silica together with carbon pigment reach the saccular ducts and alveoli. Here they are taken up by large macrophages, generally designated by the unscientific but expressive term 'dust cells,' and reach the masses of lymphoid tissue described above. At this site these 'dust cells' come to rest and are surrounded by fibroblasts. In the course of time they degenerate and disappear leaving behind them the insoluble particles of silica and carbon pigment. Meanwhile the fibroblasts have given place to collagenous fibres and one or two minute capillaries may be formed from neighbouring vessels. The early nodule is now complete. When suitably stained it resembles a small round wicker basket in which the interwoven wicker work may be taken to represent strands of collagenous fibres. (Figure 3) In the centre are a few small capillaries, and lying between the fibres are particles of silica and carbon pigment. Surrounding the whole, new fibroblasts are formed producing in their turn more collagenous fibres. By this process the older fibres in the

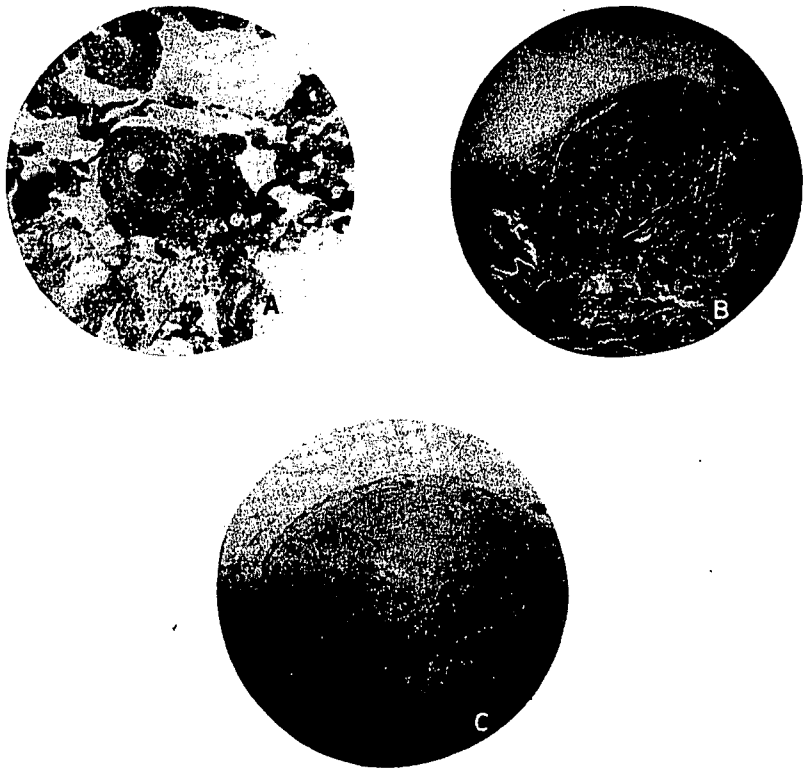


FIGURE 3

Discrete silicotic nodules. *a.* early stage (x 80); *b.* intermediate stage (x 40); *c.* late stage (x 40).

inner parts of the nodule are compressed and come to lie closer together and have a hyaline appearance when stained. The capillaries too are compressed and may ultimately disappear. The picture is now less like a wicker basket and more like a large onion with tightly packed concentric layers. A circle of capillaries can often be found around the nodule in this stage, especially when the nodules are subpleural. In this

late stage the South African writers have also described both a fatty degeneration and a pre-calcification phase. In the course of time discrete nodules come into juxta-position by reason of the collapse of intervening alveoli. They then form an irregular composite nodule. As a rule the individual nodules forming this composite mass can still be recognised and around it there may be small areas of emphysema. (Figure 4)

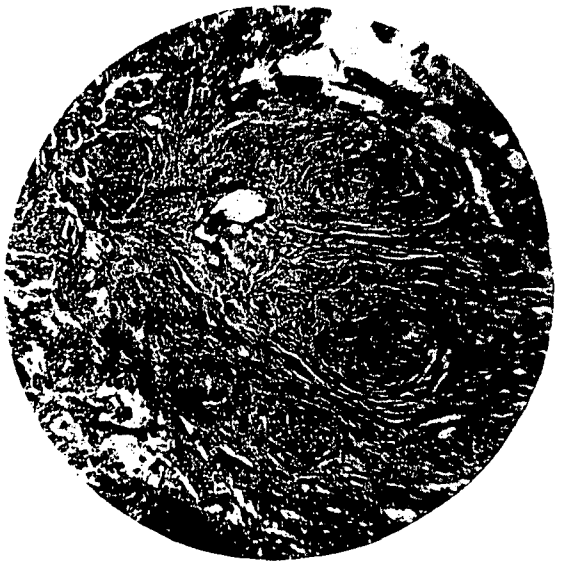


FIGURE 4

Composite nodules of silicosis (x 60).

The remaining portions of the lung show collections of macrophages in the alveoli, whilst desquamation of lining epithelium of the small bronchi is common. Hypertrophy of the aggregations of lymphoid tissue is the rule.

The Complications and Sequelae of Silicosis

The following are the chief complications and sequelae noted in the post-mortem room.

Emphysema. This has already been referred to as an almost constant finding. Occasionally an emphysematous area may become sufficiently large to form a cavity filled with muco-pus and inhaled pigment from neighbouring bronchi. Such a cavity may be mistaken for a tuberculous or bronchiectatic cavity but as a rule it does not possess the well defined wall of either of these.

The Heart. The patient often dies with all the symptoms of cardiac failure. But this is a notoriously unsatisfactory term, and at the autopsy there is usually nothing to be found except a pale, soft, flabby heart muscle with some dilatation of the chambers. There are no valvular lesions, and histologically there is little to be made out.

Septic Bronchitis and Pneumonia. Secondary infection with one or other of the organisms associated with bronchitis and pneumonia is common, the streptococcus being the organism most frequently found. On section of the lung muco-pus exudes from the bronchi around which are areas of broncho-pneumonia.

The combination of a septic broncho-pneumonia and the nodular fibrosis of silicosis may be confused with chronic fibro-caseous tuberculosis ; it is never safe to pass over the condition without microscopical investigation.

Histologically the lesions resemble those of septic bronchitis and pneumonia in general, but mention must be made of the hypertrophy of the minute aggregations of lymphoid tissue in the bronchial wall which may be confused with early microscopic lesions of tuberculosis.

Bronchiectasis. Dilatation of bronchi may supervene as a secondary result of the silicotic fibrosis. It is not very com-

mon and as a rule does not reach the stage of a fully developed foul smelling purulent bronchiectasis.

Tuberculosis. Next to septic bronchitis and pneumonia, tuberculosis is the commonest sequela of silicosis. Indeed, so common and so intimate a part of the pathological picture is it that some writers have declined to admit that such a disease as simple silicosis exists, and have attributed the whole condition to a chronic form of tuberculosis. Silicosis accompanied by tuberculosis is often referred to as 'infective silicosis.' This form of the disease will therefore be treated separately.

Carcinoma. Bridge and Henry [12] [1928] have proposed that cancer, in order to be classified as of industrial origin, should fulfil the following conditions: (1) The rate of incidence in the occupation under review should exceed that in the general population to a significant extent, and (2) in the occupation concerned there should be sufficient association of the worker with a substance proved experimentally to have carcinogenic properties. Suggestions have been made from time to time, notably in the case of the Schneeberg miners in Germany (Rostoski [13] [1928] Schmorl [14] [1928]) and again recently by Dible [15] [1934] in England that carcinoma of the lung is likely to supervene upon pneumoconiosis of long standing. The data we possess at the moment however are not sufficient to satisfy Bridge and Henry's postulates and we cannot do more than regard the association of silicosis and carcinoma (and of asbestosis and carcinoma) with suspicion. The varieties of cancer found in association with silicosis are those found in intrathoracic malignant neoplasms in general (Gloyne [16] [1930]). They are: (1) growths involving the mediastinal glands (2) growths involving the bronchi.

The primary growth involving the mediastinal glands is a rapidly growing white tumour situated in the neighbour-

hood of the bifurcation of the trachea and along the course of the two main bronchi. The glands become adherent to each other as they enlarge and coalesce into an irregular collar of growth surrounding the lower end of the trachea, the two main bronchi, and the great vessels. For many years this type of growth was described histologically as a lymphosarcoma, but it is now generally regarded as a carcinoma, an oat cell carcinoma, by reason of its characteristic oval cells, though it is not clear from what part of the respiratory tract it arises. The deep layer of cells of the epithelial lining of the main bronchi has been suggested.

The other type of malignant growth is more clearly bronchial in origin. It may arise in large or small bronchi and may be single or multiple. It needs to be carefully distinguished from the type of growth described above which has a predilection for the mediastinal glands. These primary bronchial growths extend along the bronchial wall pushing their way through the silicotic tissue as they grow. Ulceration and necrosis with cavity formation is common. This last point is important because the growth cavity may easily be confused with a pyogenic abscess or bronchiectatic cavity. Microscopic sections, therefore, should never be neglected. Histologically they are, for the most part, squamous cell carcinomata possessing the usual characteristics with prickle cell arrangement, keratinisation, and cell nest formation.

The precise relationship of these tumours to the silicosis in the matter of causation is of course impossible to define. This subject will be referred to again under the heading of asbestosis and carcinoma where the pathological relationship has been worked out further.

Silicosis and Tuberculosis

If, when silicosis is accompanied by tuberculosis, the two pathological processes were always found side by side as they

are in silicosis and pneumonia, there would be little difficulty in differentiating between the two diseases. Unfortunately, this is not always so. The two are often intimately associated in such a way as to produce what is tantamount to a separate disease. In theory, there are three possibilities in the modes of onset and of progress : (1) the tubercle bacillus is already implanted in the lung before the silica exposure begins, the tuberculous focus being either (a) quiescent or (b) active ; (2) the tubercle bacillus reaches the site as a secondary infection in a lung which has already become silicotic, the silica exposure (a) having ceased or (b) being still in operation ; (3) the tubercle bacillus and the silica particles reach the healthy lung more or less simultaneously. This third possibility must be rare. The legal mind is very much alive to these possibilities and questions relative to them are frequently asked of medical witnesses in court. Unfortunately Nature does not always paint her pathological pictures with an eye to these fine distinctions and we must be careful to distinguish between the post-mortem appearances actually found and the inferences drawn from them as to the mode of onset. For descriptive purposes we may classify the various findings of the post-mortem room into three main groups, though it will be realized that in actual practice the picture is not always so well defined : (1) silicosis with obsolescent tuberculosis ; (2) silicosis with open tuberculosis ; (3) tuberculo-silicosis.

Silicosis with Obsolescent Tuberculosis. Three cardinal signs of obsolescent tuberculosis are generally recognisable : (1) dense pleural adhesions, especially in the upper part of the pleural sac tethering the apex of the lung to the apical pleura ; (2) fibrotic contraction and scarring of the visceral pleura and subjacent lung tissue without adhesions, again especially well marked at the apices ; (3) calcified nodules. In the presence of a chronic fibrotic disease of the lung such

as silicosis these signs are often difficult to interpret. Calcification is probably the most reliable, but the South African pathologists have described small deposits of calcium in the silicotic nodule, and of course, calcification of non-tuberculous origin may take place in a vessel wall or around a foreign body. A calcified nodule beneath the pleura, or attached to the wall of a small bronchus, or in a mediastinal gland generally means obsolescent tuberculosis. If calcified deposits are found in the mesenteric glands, this fact may be taken as corroborative evidence. Generally speaking, however, silicosis found in the presence of obsolescent tuberculosis does not present any unusual features.

Silicosis with Manifest Tuberculosis. Frank active tuberculosis when it occurs is most commonly a terminal event. The following varieties are to be found: (1) pleural effusion, tuberculous empyema, or pyopneumothorax; (2) old cavities, often multilocular, with fibrotic walls having silicotic nodules adjacent thereto or actually forming part of the wall; (3) rounded foci of caseous tuberculosis with a sharply defined black rim of compressed pigmented lung tissue surrounding the foci; (4) miliary spread from an older tuberculous focus; (5) tuberculous broncho-pneumonia (which cannot always be distinguished from that due to other organisms without microscopic examination). We have here a composite picture resulting from the invasion of the pulmonary tissues by two very different types of invaders. The one is an insoluble particle which reaches the distal parts of the bronchial tree but does not reproduce, the other is a parasite with a capacity for multiplication and for being lysed by the tissues. The former remains strictly bronchogenic in its spread, the latter soon reaches the blood stream and is disseminated widely. This applies also to the next group, but to a less marked degree.

Tuberculo-Silicosis. It is convenient if not essential to have

a special term by means of which to designate a form of silicosis accompanied by tuberculosis in which the one disease is modified in association with the other. The term tuberculo-silicosis is used in this chapter in this sense only.

The modification is generally in favour of the tuberculosis, which is frequently of such a chronic fibroid type that it cannot be recognised until histological examination is made.

The first and essential feature is the excessive fibrosis. The second characteristic is the closely interwoven texture of the two disease processes, tuberculosis and silicosis, in contradistinction to the picture seen in silicosis with manifest tuberculosis. The third is the tendency to calcification.

Tuberculo-silicotic nodules vary a good deal in appearance as a result of the distribution of pigment, the silicotic portion being as a rule darkly pigmented and the tuberculous portions free from pigment. The result is that at least four different types of nodules may be recognized : (1) a nodule with a soft gray tuberculous centre and a pigmented silicotic periphery (2) a nodule with a darkly pigmented silicotic centre and a gray tuberculous periphery (3) a pigmented silicotic centre surrounded by a ring of small gray satellite tubercles (4) tuberculous and silicotic nodules side by side, the one compressing the other. These various types of nodules may result in the affected portions of the lung having a mottled or marbled gray-black appearance. (Figure 5)

Histologically the tuberculo-silicotic nodule has the following characteristics : (1) a tendency to be more irregular in shape than the simple silicotic discrete nodule, the foci running together more readily into composite nodules (2) collagenous fibres form only part of the nodule, the rest being caseous material (3) minute deposits of calcium are not uncommon (4) giant cells are few and generally lie just outside the nodule (5) tubercle bacilli are variable, the more caseous looking the nodule the greater is the likelihood of



FIGURE 5

Tuberculo-silicotic nodulation, the upper half of the photomicrograph showing caseous nodules, the lower a tuberculo-silicotic nodule with a caseating centre (x 80).

finding them ; they should be searched for in the neighbourhood of the giant cell (6) lastly, small branches of the pulmonary artery may exhibit an obliterative thromboarteritis which may be mistaken for silicotic nodules.

Of course, not all nodules found in a case of tuberculosilicosis exhibit these characteristics ; simple silicotic nodules and uncomplicated fibro-caseous tuberculous nodules occur.

Kettle and Archer [17] [1933] have investigated some of the problems encountered in this disease, which are particularly difficult in Great Britain, where a low-grade tuberculosis with excessive fibrosis is common apart from dust hazards. They believe that the differential histological diagnosis depends upon the amount and distribution of collagen

in the tissue reaction. This is a matter of degree and they cannot define any delimiting standards. They suggest, however, that a chemical analysis for silica will often help when taken in conjunction with the other findings. In their experience an 'ordinary dried lung' (amongst which may be included uncomplicated tuberculosis) does not contain more than about 0.2 per cent of silica whereas in the tuberculo-silicotic lungs they investigated, analyses up to 2.7 per cent were obtained. It goes without saying that a number of different portions of lung should be selected for analysis. (Figure 6)

Some Unusual Pathological Varieties of Silicosis

The range of industries whose workers encounter free silica is a wide one and it is not surprising that certain forms of the disease should, from the pathologist's point of view, appear unusual. At least three of these call for special descriptions.

Silicosis in Coal Miners. This condition is occasionally referred to as anthraco-silicosis and is met with in a variety of forms in those parts of the coal field which show a silica risk. A great deal has been written on this subject and a distinction is not always made between pure anthracosis and anthracosis with silicosis. One of the best recent accounts is that of Cummins and Sladden[18] (1930) dealing with miners in the Welsh coal field in the 'hard headings' of which silica dust is present. The outstanding feature of their cases was a lung showing deep black to dark gray consolidated areas on a background of chocolate coloured or gray aerated lung substance. The consolidated areas usually varied from small 'maculae' in the lower lobes to large confluent areas in the upper. Histologically the characteristic appearances were a diffuse fibrous hyperplasia of the lung with massive dust accumulation in the alveoli and lymphatics together with

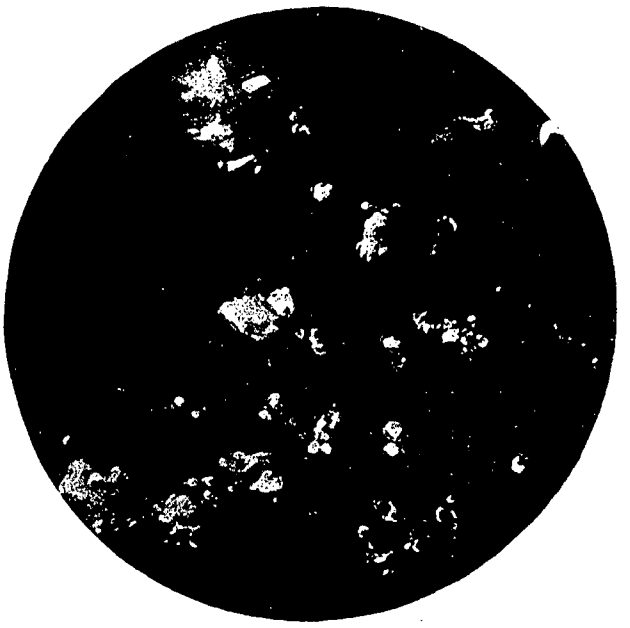


FIGURE 6

Quartz particles from stone-mason's lung. Photographed with dark ground illumination (x 1200). (Courtesy of *Tubercle*)

the occurrence of fibrous nodules, some of which showed little or no lamination while others were markedly laminated or whorled.

Silicosis in Hematite Miners. Stewart and Faulds [19] (1934) have described a series of cases in which both hematite and silica dust were present in large amounts in the lungs (sidero-silicosis). The hematite found was a ferric oxide producing a fine dry dust and gave to the affected parts of the lungs a bright red colour. The condition appeared to resemble closely the coal miner's lung in its histology, a diffuse or massive fibrosis with much dust accumulation and a 'few discrete rounded nodules of the usual silicotic type.'

In the one case the fibrotic areas appeared as bright red areas on a dark brown aerated lung background, in the other, the fibrotic areas were almost coal black on a background of chocolate coloured or gray aerated lung. Both these types of disease are of much interest to the pathologist and doubtless other similar conditions will in the course of time be described.

Broadly speaking there are two large groups of cases (1) one in which the free silica dust is preceded or accompanied by massive accumulation of a fine non-siliceous, non-toxic dust which acts chiefly in choking up the alveoli and the whole lymphatic drainage system of the lung, (2) a group in which the free silica is accompanied by a silicate which may have toxic and lethal properties of its own. (In this connection the writer is indebted to Dr.E.R.A.Merewether for a lung which shows the effects of exposure to free silica and asbestos.)

Acute Silicosis. Of recent years a number of cases of so-called 'acute silicosis' have been described, characterized generally by a short exposure to a siliceous dust and a rapidly fatal issue. The records have been collected and reviewed by Middleton [20] (1936) who gives the employment history of the largest group so far reported. In most instances there has been something unusual in the kind of dust to which the worker has been exposed. Often it has been a highly siliceous one mixed with an alkali or powdered soap for the manufacture of abrasive soap powders, and the suggestion has been made that a silica sol may be quickly formed in the presence of alkalis which is responsible for the rapid course of the disease.

Descriptions of the pathology are still very meagre. The first autopsy record was that of MacDonald [21] (1930) and colleagues. They described the lower lobes of the lung as being solid, greenish-gray in colour somewhat resembling

marble, with innumerable tiny pinhead nodules of a lighter gray colour, whilst the upper lobes were still aerated but contained similar nodules. Microscopically there were large fibrotic patches which were practically acellular and were surrounded by small-celled infiltration. They found no histological evidence of tuberculosis and no tubercle bacilli.

Kettle [22] (1932) after examination of material from cases of acute silicosis considered that the sections showed 'acute tuberculosis enhanced, no doubt, by the silica present.'

Stuart McDonald [23] (1932) recorded an example in which post-mortem examination revealed minute silicotic nodules in the lung not unlike miliary tubercles and strongly resembling the lesions of experimental silicosis in animals.

Chapman [24] (1932) described a case in which the lungs, at autopsy, were about half the normal size, the middle two-thirds being shrunken, fibrotic, and almost as hard as stone. Histologically the fibrotic nodules were found to have hyaline centres.

Obviously the scanty nature of the pathological material available at present prevents our constructing a satisfactory picture of the pathology of the disease, and there is a danger of confusing the issue, as Kettle pointed out, with cases which are primarily acute tuberculosis. A distinction should be drawn between (1) workers who have had a very short but intensive exposure to siliceous dust but whose lungs, at autopsy, contain large and even massive patches of fibrosis resembling those of the ordinary chronic form of silicosis and (2) those with an equally short exposure but with miliary silicotic nodules resembling those of the experimental animal, as in Stuart McDonald's case. (The natural life of the laboratory animal is short and therefore more strictly comparable with an acute than a chronic silicosis in man.) It might be best, at the moment, to confine the term acute silicosis to this

second group. In any case it is inadvisable to include in the definition cases showing any evidence of tuberculosis, which can play such queer tricks in association with silicosis.

ASBESTOSIS

The word asbestosis was introduced into medical literature by Cooke [25] (1927) and Stuart McDonald [26] (1927) to designate that type of pneumoconiosis induced by the inhalation of asbestos dust. Several varieties of the mineral are used in industry, including chrysotile (white) crocidolite (blue) and amosite, the first named being the commonest. A good account of the geology of the mineral is given in a monograph by Hall [27] (1930).

Naked-Eye Appearances

In an uncomplicated case the pathological signs seen at autopsy are chiefly confined to the thoracic cavity, as follows.

Pleurae. Old dense adhesions are common, and are nearly always sessile, producing in the end complete emphysis of the pleura; the long pedunculated bands so frequently seen in tuberculosis are rare. Apical adhesions, however, occur and the interlobar fissures may be completely closed.

In the least affected portions the pleura retains its transparency but as the disease advances it assumes a typical ground-glass appearance and eventually exhibiting stiff, yellow and horn-like plaques some 2 to 3 inches in diameter. (The older writers on non-industrial fibroid lung were conversant with this type of thickening and likened it to cartilage.) At the apices there may be definite scarring, but whether this is to be regarded as an invariable sign of healed tuberculosis is doubtful. Here and there, generally near the apices also, the pleura may be thin over emphysematous bullae protruding from the lung. Lastly, covering and obscuring these signs of

old pleurisy are seen evidences of recent inflammation in the shape of reddish patches of lymph arranged in thin flat gauze-like layers or as small shaggy processes. Occasionally little patches of recent haemorrhage may be seen, such patches becoming browner and darker with age.

Displacement of the mediastinum is unusual.

The changes seen in the parietal pleura are similar to those found in the visceral pleura, but less marked and the horn-like plaques are absent.

Pleural effusion is rare, owing to the obliterative character of the disease.

Lungs. The asbestos worker is generally a town dweller and therefore the pathological picture, like that of silicosis, is superimposed upon a certain amount of anthracosis which forms a background. This confusion between the two is however greater than in silicosis.

The asbestosis lung is generally large and of increased weight, though as the disease progresses it may become contracted from excessive formation of fibrous tissue. It does not collapse when the thorax is opened, and like fibroid lungs in general, is firm, tough, and dense. (Figure 7)

On section blue-black patches of irregularly polygonal shape about 1/8 to 1/4 inches across are seen ; they are hard and rough to the touch and are surrounded by a gray framework of thickened interlobular septa. This forms the typical unit of the asbestotic lung, corresponding to the silicotic nodule. In the upper lobe these polygonal areas are usually discrete with unaffected lobules lying between them. In the lower lobe they tend to run together and become confluent. Engorgement of the lung or an actual bronchopneumonia frequently provides a deep red background for the blue-black polygonal areas. Much pigment and the blue-black polygonal patches of asbestosis are found in excess in the peripheral portions of the lung immediately beneath the visceral pleura,



FIGURE 7
The asbestosis lung.

exemplifying the 'pleural drift' of the dust so common in pneumoconiosis. The interbronchial and other lymph glands of the middle mediastinal group which drain the lung, are intensely pigmented and fibrotic.

The pigmented polygonal areas of asbestosis are roughly coincident in size and shape with pulmonary lobules, and if examined with a hand lens the cut ends of small bronchi, sometimes dilated, can be seen. The fibrotic nature of the lung is better appreciated when the specimen has been fixed. The pulmonary vessels and bronchi now show thickened walls and the cut ends stand widely open, but the writer has not seen a typical extensive purulent bronchiectasis such as is sometimes described in chronic fibroid lung of non-industrial origin.

Occasionally the cut surface of the lung shows a honeycomb appearance due to the breaking down of alveolar structure analogous to the spongy emphysema described by Cummins and Sladden [18] in anthracosis. This is commoner in asbestosis than in silicosis. Cavitation without tuberculosis, such as is described by Cummins and Sladden in anthracosis, has not been found in these areas.

Mediastinal Glands. The absorption of asbestos fibres into the mediastinal glands, is a much more difficult task for the lung than is the absorption of carbon or even small particles of free silica ; but in the course of time the glands become small and hard with a thick fibrous envelope, and trabeculae are seen as white lines traversing them. When bronchopneumonia supervenes, they become secondarily enlarged and reddened.

Other Organs. Apart from the complications and sequelae to be dealt with later, there are few naked-eye changes in other organs. As in silicosis the heart muscle may be thin, pale, and flabby, and the spleen hard and fibrotic, but there is little else of importance.

Microscopical Appearances

The Asbestos Fibre and the Asbestosis Body. The asbestotic lung is essentially a dusty lung. It contains in addition to the asbestos fibres and the bodies formed from them, carbon pigment common to all town dwellers, jagged carbonaceous particles such as are present in coal miners' lungs, blood pigment, and metallic impurities of the asbestos dust which tend to accumulate in the neighbourhood of the asbestosis bodies. These various structures are apt to obscure the real causative factor, the asbestos fibre. If asbestos dust be examined with dark-ground illumination, it will be seen to

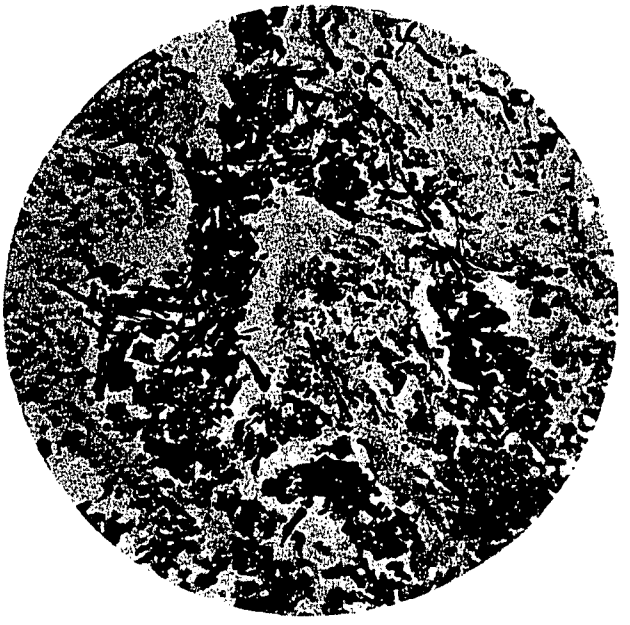


FIGURE 8

Asbestotic lung showing the respiratory bronchioles packed with asbestosis bodies, fibres and pigment (x 360).



FIGURE 9
Asbestotic lung showing reticular fibrosis of the alveolar walls (x 150).

consist of fibres of varying length mixed up with particulate matter of irregular shape and often of reddish colour, representing various mineral impurities. The fibres themselves are straight and highly refractive, and of course, vary in length according to the thoroughness with which the crude material has been broken up. They may extend across the whole field of the microscope or they may be no longer than an anthrax bacillus. They can even be broken up sufficiently small to make a suspension which can be inoculated intravenously into the blood stream of the rabbit without producing emboli. In the raw material the fibres are held together in thick, straight bundles which, when crushed, are split up into

smaller and smaller bundles, until eventually very fine fibres are obtained. No branching has been observed. Under dark-ground illumination the fibres appear to be brightly illuminated like the filament in an electric bulb, and when very thin have a bluish luminosity. They are readily distinguishable from the irregularly shaped silica particles of the silicosis lung. (Figures 8, 9 and 10)

The asbestos fibre is of a highly resistant nature, hence its use in industry. It will withstand heat, and is not destroyed by mineral acids. It has been stated that with high-power magnification minute pores can be perceived in the fibre which render it capable of absorbing, to a certain extent, dyes

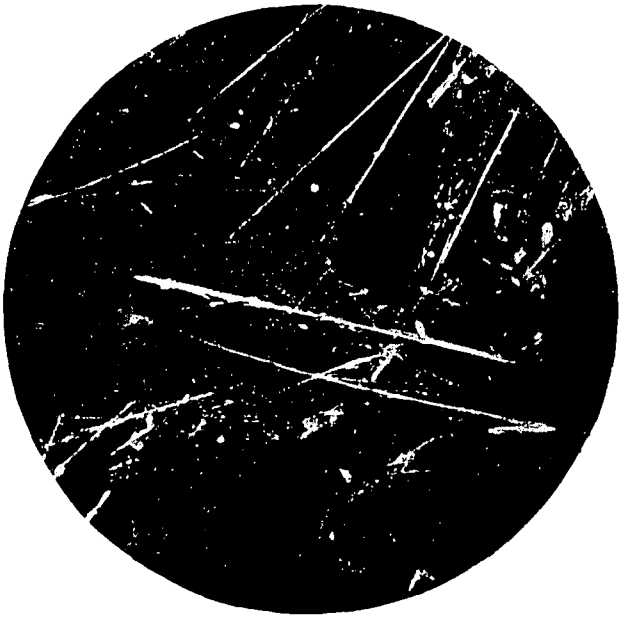


FIGURE 10

Asbestos fibres (crocidolite). Photographed with dark ground illumination (x 1200).

such as methylene blue. The writer has not been able to confirm this.

The fibres are found in large numbers in the lungs. Their detection is readily achieved by destroying the surrounding lung tissue with concentrated sulphuric acid, but in ordinary stained sections the presence of these fibres is often obscured by other foreign particles.

In the lung of man two types of tissue reaction take place. The one is the thickening of the fibre, as a result of deposition of iron-containing material along its course, to form the asbestosis body. The other is the cellular reaction to the fibre.

The asbestosis body has had a curious history. In 1914 Fahr,[28] demonstrating to the Medical Society of Hamburg specimens and photomicrographs of the lungs from a case of pneumoconiosis occurring in an asbestos worker, observed the presence in the lung of a large number of crystals and stated that similar structures had been seen in 1906 by Marchand and Riesel, who had speculated as to whether they were due to the inhalation of asbestos dust or were a haemoglobin derivative. No pictures are given in Fahr's communication, but it is probable that what Marchand and Riesel and Fahr saw was the asbestosis body. However, these descriptions attracted little attention at the time and seem to have been forgotten.

The next step came in 1927 when Cooke and Hill [29] and Stuart McDonald [26] described certain 'curious bodies' found in the lungs of an asbestos worker who had died in 1924. Cooke had previously recorded the case in 1924 but had not at that time mentioned these bodies. Cooke and Hill wrote of them as follows: 'They are yellowish-brown in colour and do not stain with the usual stains, but give the Prussian blue reaction. There is a uniformity in appearance, group distribution, and fructating heads that make one think of a fungus. The hyphae are verruciform and discoid, and definite spores

are seen.' Pathologists to whom the material was submitted suggested various possibilities, viz., diatomaceae, casts of small cavities, particles of asbestos fibre that had become coated with colloidal matter, fungoid bodies.

Later in the same year, Stuart McDonald, to whom post-mortem material had been submitted from the same case by Cooke, gave a fuller description of the bodies. He regarded them as foreign bodies associated with the asbestos and was satisfied that they were neither vegetable nor animal in origin, and quoted another case of an asbestos worker in whose lungs he had found similar bodies.

The first illustrations were given by Cooke and Hill in their original description. Subsequently they were depicted by Simson [30] (1928) in autopsies of South African cases. His paper also included a microphotograph of the bodies produced experimentally in the lung of the guinea-pig by Mavrogordato, the first occasion on which the bodies had been so produced.

At this stage Stewart and Haddow [31] (1929) made the suggestion that the bodies should be called 'asbestosis bodies,' a term which has now been generally adopted.

Finally Gloyne [32] (1929) showed in the following experiment that the body consisted of an asbestos fibre coated with a substance soluble in a strong acid.

A wet preparation of the bodies was placed on the stage of the dark-ground illumination microscope, and the position of a readily recognisable group of these bodies noted. Concentrated sulphuric acid was then run very gently under the cover slip, the bodies being kept under observation all the time. They began slowly to dissolve, and in the middle of the structure of each appeared a typical asbestos fibre. At the end of half an hour practically all the material of which the bodies were composed had disappeared, leaving only faint ghost-like outlines with refractile asbestos fibres within them.

The diversity in shape of the bodies makes it difficult to write a satisfactory description of them. The following summary comprises the chief points: (1) Marked variation in length (24 to 60 microns) and breadth (12 to 24 microns). Gardner and Cummings [33] record them as long as 250 microns in experimental animals. The short forms are sometimes ingested by phagocytes. (2) Golden-yellow colour. Gardner and Cummings have pointed out the resemblance in colour to haemosiderin deposits in tissue in which haemorrhage has occurred. (3) Homogeneous structure. (4) The central fibre. This can be best seen by reducing the illumination. The fibre may sometimes be detected projecting beyond the body. (5) No differentiation with polarised light (Cooke and Stuart McDonald). (6) Tendency to be arranged in irregular clumps and clusters. (7) Very early forms unsegmented ('sausage shaped'). (8) Later forms crenated, segmented, or resembling a series of oval discs or beads strung together in necklace form. (9) Bulbous or pointed extremities (or one bulbous and one pointed suggesting 'heads and tails'). (10) Generally quite straight, occasionally curved, very rarely S-shaped. When examined in a wet preparation slight pressure on the cover slip will sometimes cause them to bend owing to the elasticity of the central fibre. (11) Forms seen 'end-on' look like knobs or door-handles. Small secondary knobs and bosses sometimes seen thereon.

The bodies do not stain with the ordinary aniline dyes; but according to Gardner and Cummings, Wright's blood stain gives them a 'greenish cast.' They will also retain neutral red slightly, but these partial reactions cannot be called true staining reactions. Cooke and Hill and Stuart McDonald, in their original descriptions, noted that the bodies could be stained by means of the Prussian blue reaction, and Gloyne has shown that they become black on treatment with ammonium sulphide. Haematoxylin, which also has an affinity

for iron, can be made to colour them to some extent, but it is more a deposit of stain than a true staining reaction.

With regard to their formation, no such bodies have been found in crude asbestos or in asbestos dust, and it seems practically certain that they are only produced when the fibres gain access to living tissues and remain there for a considerable period of time.

Gardner and Cummings showed that the golden-yellow material which covers the fibre contained iron, whilst Simson and Strachan put forward the view that the iron-containing substance was of cellular origin.

From a study of a large number of different forms of asbestosis bodies, the present writer has endeavoured to build up a complete picture of formation from the original inhaled fibre. Figures 11_1 to 11_3 show deposition of some material on the fibres to a varying extent. When completely covered the body thus built up has a sausage-like form without any differentiation from end to end (Figure 11_4). These thickened forms next begin to show definite fissures (Figures 11_5 to 11_7), giving the appearance of incomplete segmentation at irregular intervals. In Figure 11_8 this segmentation has advanced a stage further, the fissures now extending almost to the mid-line of the body and occurring at irregular intervals along its length. These fissures produce the appearance of a head or knobs at the ends (Figures 11_9 and 11_{10}). As they become deeper and more numerous a completely but irregularly segmented body results (Figure 11_{11}). When the fissures reach the straight central fibre they appear as (a) a small number of long segments as in Figures 11_{12} to 11_{18}, or (b) a large number of short segments as in Figures 11_{19} to 11_{34}. These short segments may be regular in size and shape resembling beads on a necklace (Figures 11_{32} to 11_{34}), or the segmentation may be complete at one end of the body and incomplete at the other.

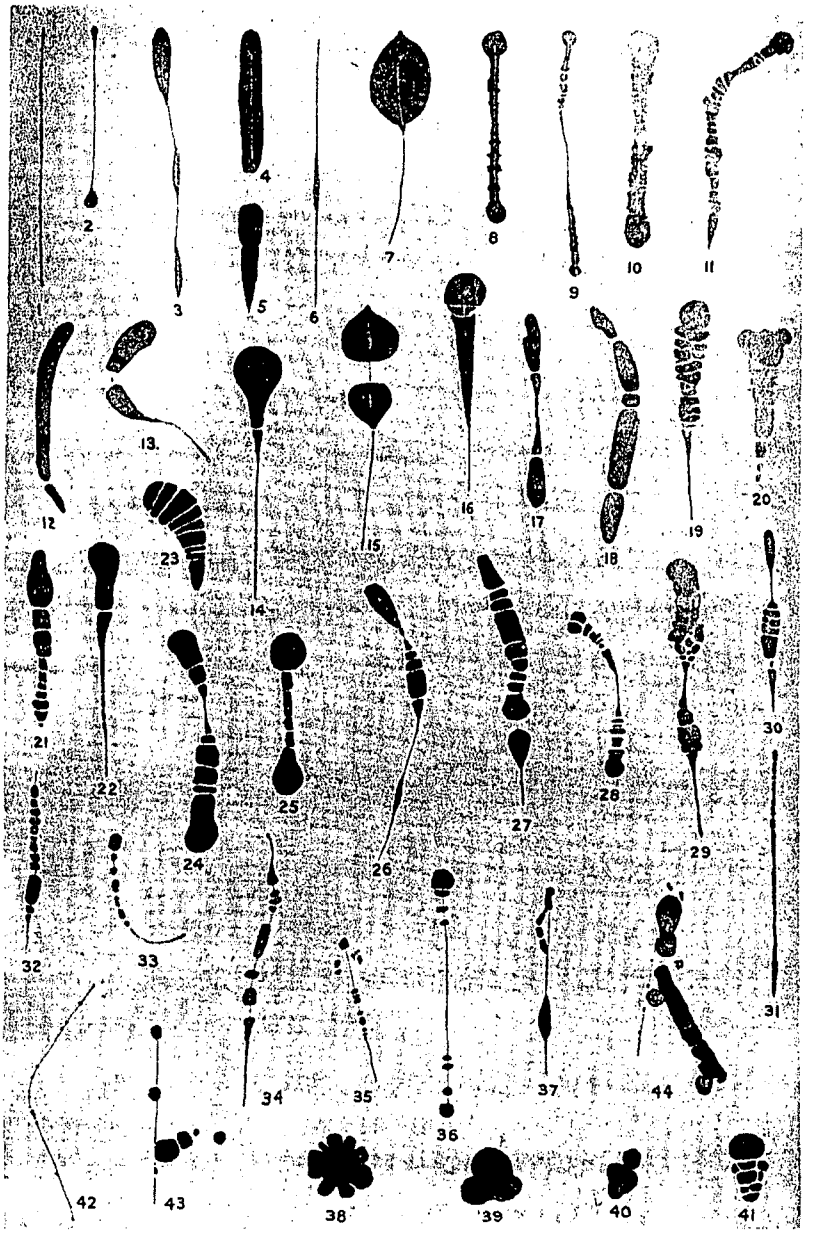


FIGURE 11

Stages of formation of the asbestosis bodies.
(Courtesy of *Tubercle*)

In this final stage some of the segments or even small portions break away leaving bare lengths of fibre between the remaining segments (Figures 11_{35} to 11_{37}). Very occasionally these broken fragments can be actually seen in the process of breaking away. This is well shown in Figures 11_{35} and 11_{37}. Bodies viewed end-on or obliquely are seen in Figures 11_{38} to 11_{41}.

The asbestosis body would appear, therefore, to be formed from the original fibre by a process of (1) deposition of some material around the fibre so as to thicken it; (2) fissure of this material giving rise to the appearance of segments; and (3) fragmentation or the separating off from the asbestosis body of fractured portions of the deposited material.

The central fibre having been satisfactorily demonstrated, the question arises, of what does the golden material consist which forms the segmented coating? Iron certainly enters into its composition, for, as far as we know, all asbestosis bodies give an iron reaction. This metal is present in crude asbestos in varying amounts, but it is also present in haemoglobin. On the whole, the evidence is in favour of the iron being derived from the tissues — probably from blood pigment — but the point has not been conclusively settled, and it seems probable that the golden yellow substance is the product of a colloidal reaction between the fibre and blood protein.

Asbestosis bodies have considerable power of resisting the ferments of the gastro-intestinal tract when swallowed with sputum and have been found in the faeces. They also resist heat. The effect of acids varies a good deal. In sections of lung tissue they resist decalcifying fluid, which contains 7½ per cent of nitric acid, for several weeks. Concentrated sulphuric acid leaves only a mere semblance of a ghost-like body with a central fibre. Hydrochloric acid has the effect of bleaching the bodies; the yellow colour is taken out almost immediately

leaving a colourless skeleton of the body, suggestive of a minute hyaline urinary cast. Nitric acid has less effect and glacial acetic acid none. The bodies will resist 20 per cent caustic soda for twenty-four hours without losing their golden-yellow colour. They become black when digested with trypsin.

The inference from these reactions would appear to be that the asbestosis body consists of at least two portions surrounding the fibre — viz., an acid resistant material which forms a framework and gives the body the characteristic shape, and a golden-yellow pigment contained therein which is not acid resistant.

The question now arises, what inference can be drawn from the presence of the asbestosis body? Since these structures have not been found in any other disease, their presence is an indication of exposure to asbestos dust, whilst the fact that the bodies have not been found in asbestos dust itself implies that their formation takes place in the body — probably as a result of a colloidal reaction with the blood proteins in which the iron plays an important part. They have been found in the guinea-pig by different observers from seventy days to six months after exposure to dust. Simson has recorded the bodies in a human lung with a history of only two months' exposure.

Lastly, can it be inferred from the presence of the asbestosis body that fibrosis necessarily exists in the lung? The presence of the asbestosis body implies a tissue reaction to the fibre, but it does not necessarily follow that this reaction runs parri passu with the production of pulmonary fibrosis. On one occasion the writer found fine collagenous fibres in the tissues of a guinea-pig as early as twelve days after subcutaneous inoculation of asbestos fibres, but the amount was very minute. A heavy dose of fibres may be needed — and even then years may elapse — before pulmonary fibrosis is sufficient to be detected by physical signs or roentgenogram during life. It

would be unsafe, therefore, to accept the presence of the asbestosis body as undeniable evidence of fibrosis ; all we can say at present is that the asbestosis body is an expression of a tissue reaction to a foreign body acting as a benign irritant, and that fibrosis of an appreciable extent may or may not be a part of that tissue reaction.

There is some evidence that asbestosis bodies are not formed with the same facility in all the tissues of the body. Thus they have not yet been described in the skin warts to which the workers are liable. The writer has examined without success sections of a wart which was known to have been present for many weeks, and has failed also to find them in the skin of a guinea-pig seven months after the asbestos fibres had been introduced into the cutaneous tissues. Dewirtz also does not make any mention of asbestosis bodies in sections of warts he examined from the skin of Moscow factory workers. Furthermore, even the lung varies in its reaction to asbestos in different animals. Gardner and Cummings [33] (1931) record that the lung of the rabbit apparently offers a much less favourable environment for the production of the bodies than does the lung of man and of the guinea-pig. The rat also does not produce bodies readily, whilst Schuster [34] (1931) failed to find them in the lungs of a dog which had been kept on factory premises for the purpose of ratting for several years, though fibrosis of the lungs was present.

The Lungs. The tissue reaction to the asbestos fibre in the lungs depends partly on the non-absorbable character of the particle and partly on its silicate content. There is little leucocytic reaction, the cells chiefly concerned being the lining epithelium of the respiratory bronchioles and the large macrophage which is the striking feature of the cellular reaction in many of the non-pyogenic pulmonary diseases. The bronchial epithelial cells do not phagocyte the asbestos fibre. They are simply desquamated into the lumen of the tube as

a mechanical result of the irritant action of the fibre. The macrophages are identical with the so-called 'dust cells' of the lung and surround the fibre in an attempt to engulf it. Frequently the fibre is too long to be completely engulfed and looks as though it was sticking into the cell with one end protruding, like a pin in a pincushion. Whether this is in all cases a true statement of facts is not quite certain owing to the liability of the long asbestos fibre to lie obliquely rather than in one plane. (Figure 12)

In addition to these tissue phagocytes, asbestosis giant cells and fibroblasts are seen. The former are foreign body giant cells probably resulting from partial degeneration of small collections of the above-described large phagocytes which have



FIGURE 12

Asbestosis bodies (low power) in unstained section of lung (x 520) (Reproduced from article by W.Burton Wood and D.S.Page) (Courtesy of *Tubercle*)

lost all their envelopes. In early lesions, e.g. in the guinea-pig, the cytoplasm has a pigmented stippled appearance in contradistinction to the almost structureless look of the tuberculosis giant cell. Sometimes the faint outlines of the degenerated individual cells which form this composite 'cell' can be seen. The cell also varies considerably in size according to the number of phagocytes composing it. Finally, the nuclei of the asbestosis giant cell are large and less numerous than those of the tuberculosis giant cell and have not the same tendency to be arranged round the periphery. The fibroblast does not appear to differ in appearance in asbestosis from the fibroblast of other varieties of pneumoconiosis. It is found around bronchioles and alveoli, in interlobular septa and in the subpleural connective tissue. (Figure 13)

The trachea and main bronchi are little affected in asbestosis. The bodies can be seen on the surface of the epithelium, having been carried there by bronchial secretion, and occasionally they may be seen in the mucous or fibrous layer of the wall of the main bronchus surrounded by phagocytes and free pigment. The writer has not yet seen them actually in the tissues of the trachea. In the intra-pulmonary bronchi, from the point where they enter the lung to the point where they terminate in respiratory bronchioles, the most marked change is the desquamation of the epithelium, and lying amongst the desquamated epithelial cells asbestosis bodies, fibres and loose pigment are frequently to be found, often plugging the lumen. The basement membrane is thickened and the fibromuscular coat surrounded by a ring of collagenous fibres. The elastic fibres generally remain intact, but in some places they are found to be contracted together as though definitely increased and when ulceration takes place they may be found in the sputum. Asbestosis bodies may be found in any part of the wall.

Gardner and Cummings [33] (1931) have shown by animal



FIGURE 13

The asbestosis giant cell showing phagocyted asbestosis bodies and fibres (x 1200) (Courtesy of *Tubercle*)

inoculation experiments, however, that the chief site of asbestos dust localisation is the respiratory bronchiole. In man, asbestos fibres, pigment, cellular debris, phagocytes and asbestosis bodies — the latter frequently in radially arranged clumps — are all to be found choking the lumina of these tracts, blocking the narrow alveolar ducts, and filling the air sacs and alveoli. Lastly, around these distal portions of the bronchial tree and of the alveoli a fine network of collagenous fibres is formed, ultimately producing a generalised reticular fibrosis which is a distinguishing feature of the disease. When this diffuse fibrosis has reached an advanced stage all alveoli in the area may be compressed out of recognition. Whorled nod-

ules such as those of silicosis are rare but are occasionally found in these areas of advanced fibrosis. Asbestosis bodies are occasionally present in the proximal parts of the plexus of lymphatics around the smaller bronchi, and become more numerous towards the respiratory bronchioles and are plentiful at the level of alveolar ducts. The lymphatics accompanying the branches of the pulmonary artery less frequently show asbestosis bodies or pigment. In the small lymph-nodes draining the lung tissue asbestosis bodies as a rule are not plentiful owing to the difficulty the fibres encounter in entering the afferent ducts. The aggregations of lymphoid tissue in the walls of the small bronchi are hypertrophied.

Changes in the vascular system are relatively unimportant, presumably because the toxicity of asbestos is not marked. There may be an increase of collagenous fibres around branches of the pulmonary artery and asbestosis bodies may also be seen lying in the connective tissue between the artery and the wall of the bronchiole. The capillaries are of course involved in the reticular fibrosis of the alveoli wall of which they are the chief constituents and may be compressed almost to the point of occlusion. The pulmonary venules are also involved in this fibrosis. Lynch and Smith (1930) have reported asbestosis bodies in the thrombi of veins.

The Pleura. The surface of the visceral pleura is generally extensively denuded of epithelium, with patches of fibrin containing a few desquamated endothelial cells lying on the denuded surface. The subendothelial portion is much thickened by the formation of collagenous fibres with many fibroblasts, and new formed blood-capillaries are numerous, especially in the deeper layers. Asbestosis bodies are scanty and tend to be short and stumpy — probably the larger fibres do not penetrate so far — and usually lie surrounded by connective tissue in small groups. Fibres are also occasionally seen, singly or in sheafs. The elastic layer is generally intact.

The interlobular septa may show asbestosis bodies, fibres and pigment with or without collagenous thickening. In two cases whorls of connective tissue resembling discrete silicotic nodules were found by the writer near the point where the septum joined the pleura — which is the point at which the pulmonary deep lymph-vessels in the septum anastomose with the subpleural lymph-vessels.

Histology of other Organs. H.L.Stewart, Butcher and Coleman [35] (1931) have found asbestosis bodies in the spleen. The present writer has failed to find them in any of the abdominal organs, even in the walls of the stomach and intestines where swallowed sputum might conceivably carry the bodies.

To summarise, the typical histological lesions of asbestosis are three : (1) The pouring out of large macrophages into the most distal portions of the bronchial tree and of the alveoli to surround and as far as possible phagocyte the invading fibres ; (2) the formation of the asbestosis giant cell ; (3) a generalised reticular fibrosis surrounding the bronchioles, alveolar ducts, alveoli and air sacs, capillaries and venules, the interlobular septa and pleura, ultimately resulting in a complete obliteration of all lung structure.

Complications and Sequelae

The chief complications and sequelae of pulmonary asbestosis are as follows : —

(a) Purulent bronchitis.

(b) Bronchopneumonia, which is best seen in the parts of the lung least affected with the asbestosis. This point is probably of importance because it explains why bronchopneumonia is such a fatal disease in asbestosis — it puts out of action the last remaining functioning portions of the lung.

(c) Pulmonary tuberculosis. In a series of 100 cases published by Burton Wood [36] (1934) and the writer, active

tuberculosis was found in 21. The cases coming to autopsy have been of the acute caseous type (in one case with a generalised miliary spread), with few Langhans giant cells and no obvious sign of healing. Asbestos fibres, asbestosis bodies and tubercle bacilli have been found lying side by side in the caseous centres, but there is evidence in animal experiments (Gardner and Cummings) that asbestosis bodies disappear in caseous areas. In the series quoted above there was one case of pyopneumothorax with lardaceous disease following treatment by collapse therapy. A number of cases of healed and obsolescent tuberculosis have also been found at autopsy. These are probably not more numerous than in post-mortem work in general, but they indicate that the supervention of asbestosis does not necessarily mean the lighting up of quiescent tuberculosis.

(d) Empyema. In the series mentioned above there was one case of tuberculous empyema.

(e) Bronchiectasis. Dilated bronchi are occasionally met with, but bronchiectasis with fusiform or cavity-like dilatations containing foul pus have not been encountered by the writer.

(f) Carcinoma has been found in 6 cases examined by the writer at autopsy. Until more statistics are available it is impossible to draw any very definite conclusions as to the relation of the two diseases. The points noted up to the present are (1) 5 out of the 6 cases of carcinoma have been of the squamous variety, the sixth being an oat cell carcinoma; (2) 4 out of the 6 have been associated with asbestosis which was fairly advanced and of long standing; (3) in 4 cases the carcinoma consisted of an isolated nodule only; (4) the nodules were in the portions of the lung most affected by the asbestosis — i.e. basal or peripheral; (5) 2 only showed secondary growths, one in pericardium, thoracic wall, liver and kidney, the other in thoracic vertebrae; (6) histologi-

cally the squamous carcinoma appeared to advance along the mucosa of the small bronchi, pushing aside the pigment, asbestos fibres and asbestosis bodies as it grew, whilst in the 1 case of oat cell carcinoma observed, the tumour grew outwards from a centre in a compact mass without any tendency to confine itself to prolongation along and infiltration of the bronchial mucosa; (7) death occurred in 4 of the cases whilst the growth was still small (i.e. not more than 1 to 1½ inches in diameter.)

Clinical Pathology and Post-Mortem Examination

Laboratory investigations of silicosis and asbestosis have been few. The following is a brief summary, and the reader is referred to text books on biochemistry for the more detailed account of silica estimations.

Sputum. Patients usually produce very little sputum until the late stages of the disease when the material expectorated is often the product of intercurrent bronchitis and, owing to the excess of mucus, may be of small value. The sputum of asbestosis is more likely to yield helpful information than that of silicosis by reason of the asbestosis body.

Asbestosis. The sputum is generally thick and mucoid — frequently so thick that it resembles semi-coagulated egg albumin. In the latest stages small nummules of pus appear. The asbestosis body may occasionally be found by examination of a direct smear, but as a rule a concentration method is necessary for its detection. The one in general use is that originally devised by Stewart and Haddow[31] (1929). It is carried out as follows: — About half an ounce of sputum is dissolved in an equal quantity of undiluted formalin, centrifuged, the deposit washed and examined microscopically. This may be done by drying and fixing a direct smear of the deposit (which need not be stained) or by mounting a small

quantity under a cover glass as in examining a urine deposit. Stewart [37] (1934) suggests that the occurrence of asbestosis bodies in clusters is indicative of tissue destruction in the lung sufficient to liberate the clusters from the small bronchioles in which they have been formed.

Stewart and Haddow (1929) [31] have also obtained the bodies by direct lung puncture, whilst the present writer has obtained them in faeces in a case in which it was believed that the sputum was being swallowed.

Silicosis. Here there is no distinctive structure to fall back upon and the detection of particles of free silica by direct illumination and the ordinary microscopic technique is impossible. Burke [38] (1935) has described a technique which unfortunately requires a larger amount of sputum for its performance than is available in the great majority of instances. A large quantity of sputum (100 cc or more) is gently heated in a beaker until most of the water is evaporated; 50 cc of agua regia is then added and the mixture left at room temperature for twenty-four hours with occasional shaking. It is then centrifuged at high speed for one hour, the deposit smeared on chemically clean slides and incinerated to a white or pale grey ash. This deposit is examined by polarised light for doubly refractile particles which are generally angular or needle-shaped and vary in size from 1 to 5 microns. These particles can be further identified as quartz by mounting in immersion fluids of known refractive indices.

Blood. Schlomka and Nolte [39] (1935) have made blood counts and sedimentation rate estimations on a series of patients with silicosis. Their results indicate that this method of approach may be helpful in marking the onset of tuberculosis. They found that in various types of silicosis accompanied by tuberculosis the blood sedimentation rate was increased, the total leucocyte count was higher and the propor-

tion of polymorphonuclear cells in the differential count was increased, when compared with the figures obtained in simple silicosis.

King and Stantial [40] (1933) record methods of estimating the silica in the blood by a micro-determination technique of analysis developed from those used by chemists in estimating small quantities of silicic acid in various natural waters. Briefly this consists in the reduction of a yellow silicomolybdic acid complex to give a blue colour with aminonaphthol-sulphonic acid, a blue colour being more accurately matched with a colorimeter than a yellow. The methods are too long and complicated for abstraction here and the original papers should be consulted. Both soluble and total silica are examined, the former with 2 cc and the latter with 5 cc blood. The authors state that there appears to be an absence of any insoluble particulate silica and of any organic compound of silicon in the blood so that the figures for soluble and total silica are approximately equal.

Urine. King and Stantial [40] have also applied the above methods to the estimation of silica in small quantities of urine, viz., 5 cc.

Silica is a natural constituent of animal tissues and the deductions which can safely be made in cases of silicosis call for a good deal of experience. King and Dolan [41] (1934) found that silica which was absorbed from the lung or other parts of the body into the blood stream was rapidly excreted in the urine. The elimination of silica in the sputum was higher than in persons having no silica exposure. Attempts made to influence the absorption of silica from the lungs by the administration of alkali were inconclusive. Comparing the blood silica content of non-silicotic and silicotic persons (all of whom had tuberculosis) King and Dolan were unable to detect any significant differences. The results obtained by these workers accord with those obtained with numerous

other biochemical substances in disease. There appears to be a 'tissue threshold' which enables the body to maintain the constituents of the blood on a fairly even keel until the last stages of disease. Biochemical investigations, therefore, in the present stage of our knowledge do not help the clinician very greatly in assessing the functional capacity of the silicotic or asbestotic lung, though they are invaluable in all research work on the subject.

Autopsies. The following details in the making of post-mortem examinations in these cases have, in the writer's practice, been found worth attention.

The lungs should be opened in such a manner that they can be compared with the roentgenogram and the chest diagram of physical signs made by the physician during life. It makes things unnecessarily difficult for those who have to assess compensation claims to find that the lungs have been cut to ribbons in all directions. The writer's practice is to place the lungs, with trachea and bronchi attached, on the operating table with their posterior surfaces uppermost and then to make serial horizontal slices, which can be replaced, from the mid-line of the mediastinum outwards to the pleura. If this operation can be postponed until the lungs have been hardened in formalin, so much the better.

The natural tendency of the pathologist is to reserve the most diseased portion of the lung for histological examination. Whilst this is in general sound practice, the portion thus selected is often so altered by fibrosis or tuberculosis that satisfactory study is difficult. The portion of lung which, naked eye, looks least affected by the disease is often the best for microscopy. Here the silicotic nodules may be found lying in the midst of relatively healthy tissue and can be examined readily. In cases of doubt a portion of interbronchial lymph gland should also be selected ; if there are silicotic nodules anywhere in the specimen they are most likely to be found

here. A minimum of three sections should therefore be chosen, (1) the most typical portion of advanced disease ; (2) the healthiest looking piece of lung, and (3) an inter-bronchial lymph gland. If possible (1) and (2) should include pleura, whilst portions of lung representing intermediate stages are also selected as required in difficult cases. The writer uses a chest diagram similar to those employed by physicians and marks thereon the position of all portions of lung selected for histological study.

Histology. In silicosis where the demonstration of the whorled nodule is all important, a good stain for the collagenous fibre is essential. No doubt pathologists will differ on this point, but the writer has a strong preference for Van Gieson's stain. Reliance on a simple routine haematoxylin stain is liable to lead one astray, especially in tuberculo-silicosis. The only safe rule is to stain three sections from each block of tissue with (1) haematoxylin and a good counter stain for cellular structure, (2) a collagenous fibre stain such as Van Gieson or Mallory and (3) Ziehl Neelson for tubercle bacilli.

These rules apply with less force to asbestosis. A warning may be given, however, with regard to the resistance properties of the asbestosis body.

In studying the microscopical picture of the different organs one error must be guarded against, viz., the assumption that the asbestosis body has been actually formed at the site in which it is found. Although these bodies probably do not easily migrate far, if at all, during life from the position in which they are formed, they are easily moved in dead tissues by various manipulations. Moreover, they are highly resistant to cleansing, and glassware should be carefully cleansed by concentrated sulphuric acid. In forming an opinion as to whether an asbestosis body has been actually formed where it is found lying in the tissues the following points are help-

ful: (a) the presence of amorphous brown pigment, phagocytes and fibrous tissue around; (b) its relation to surrounding structures (bronchiole, capillary, etc.) The cardinal point in the histology is the liability of the asbestos fibre to be held up at the distal end of the respiratory bronchiole.

Silica particles cannot be detected satisfactorily in ordinary sections mounted in Canada balsam and viewed with transmitted light. With a mounting fluid of high specific gravity such as bromobenzene, particles are more readily detected, but it is only by the use of crossed nicols in a petrological microscope that silica particles can be demonstrated with any certainty. Viewed in this way the quartz particles stand out clearly on a dark background. For the actual technique of the petrological microscope the reader is referred to special mineralogical textbooks.

Irwin[42] (1934) has devised a simple method of micro-incineration which enables the observer to detect silica particles in situ in silica nodules in sections. After removal of the paraffin from the section (which should not be more than 5 microns thick) in the usual way, the slide is heated to 550–600°C. After cooling the incinerated section is mounted in concentrated hydrochloric acid covered with a coverslip and examined by direct illumination for quartz particles. A small furnace is needed for the incineration. The microscope slides vary a good deal in their resistance to the necessary heat and in the writer's experience there are a good many casualties in the way of cracked slides. The ash pattern obtained with silicotic nodules by this method is characteristic. A somewhat similar technique has been used by Schutz and Braun (quoted by Siegmund and Köppenhofer 1935).

Dark ground illumination also has its uses. It may be employed with advantage to detect the characteristic asbestos fibres in wet or dried fibres of sputum and in sections. Siegmund and Köppenhofer have also used it in silicosis (quoting

it as the method of Timms) but the present writer has found so many pitfalls that he has discontinued it.

Extraction of Asbestos and Silica Particles from Lung. The extraction of asbestos fibres and asbestosis bodies from the cut surface of the lung is usually a simple matter. A direct smear on to a slide or a film from the centrifuged deposit of a little tissue fluid which has been expressed from the organ is usually sufficient. In the old stone-mason's lung, also, quartz particles can sometimes be obtained readily by removing a subpleural nodule with the point of a scalpel and compressing it between two slides.

But in most instances silica particles are much more difficult to isolate. The best technique is that devised by Jones [3] (1933) for the isolation of sericite fibres and termed by him 'sliming.' Portions of the lung freed from preservative by preliminary washing are treated by gradual disintegration with strong nitric acid over a period of several days. The slime thus obtained is diluted about ten times with hot water and the mixture filtered through No. 54 Whatman's filter paper (this is important, since from the ash of many filter papers minute fibres of cellulose which may cause confusion under the petrological microscope are detached). After repeated washings the residue is carefully dried (the fumes given off are inflammable) and the material ignited in the usual way. The light ash is removed with a mixture of bromoform and benzene in a Sollas separator and the minerals which sink to the bottom are washed with benzene, then alcohol, dried, and mounted on slides in Canada balsam. A petrological microscope is necessary for the identification of particles.

Chemical Analysis of the Lung. This is done by chemical assay of the wet or dry ash of the lung. In both cases it is necessary to remove the organic matter first. Jones recommends the method of wet ashing (as described above for the examination of silica particles) on the ground that a dry ash

may contain a high percentage of constituents such as phosphoric oxide, sodium oxide, etc., which are removed by his wet ash technique. The majority of analyses have, however, been carried out by dry ashing. This consists in mincing up large slices of lung, drying at 100° until friable, powdering in a coffee mill, mixing and drying again at 110°, ashing a small portion (e.g. 10 gms) in crucible, weighing ash and assaying it (e.g. by gravimetric method of fusion with alkaline carbonates, precipitation of silica by repeated treatment with hydrochloric acid, ashing the precipitate and determining the true silica content of this ash by treatment with hydrofluoric acid) (Kettle and Archer [17] [1933]).

King and Stantial [40] (1933) have devised a micro-determination technique by means of which small quantities of tissue can be analysed (about 1 gram fresh tissue or 0.25 grams pulverised dried tissue) as in the case of blood and urine mentioned above. The advantages of using such a method are obvious in that they afford an opportunity of comparing areas of diseased lung by chemical and histological methods which the ashing of a large slice of lung does not.

No hard and fast line can be drawn between the percentage of silica in the dried lung in silicotic and non-silicotic persons, but probably it would be safe to put the upper limit for the non-silicotic lung at about 0.2 per cent. Silicotic lungs often yield analyses of 1 to 2 per cent.

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from acting on the tissues for a considerable period of years. The effect of these substances is apparently exerted within the tissues in a manner not yet understood. Infection tends to neutralize the inhibition so that the quartz can act more rapidly. Ultimately the protective action is overcome and a more or less modified silicosis develops. It would be profitable to determine quantitatively the amount of feldspar or mica or other unnamed substance which is necessary to neutralize the action of a unit quantity of quartz.

From the experimental and clinical observations that have been cited it no longer seems reasonable to assume that the silica hazard is only proportional to the amount of free silica in an atmospheric dust. The other substances present in combination with the silica modify its action. Some of them, like iron and gypsum, prevent its inhalation; others, like the components of granite seem to prevent it from affecting the tissues. The mode of action of metallic aluminum has not yet been determined although the preliminary reports suggest that it may fall in the latter category. Much more must be done to evaluate the quantitative relationships which govern the action of inhibitor substances on silica but that they exist and exert a very definite influence is the burden of this argument.

Asbestos

The only form of asbestos tested by inhalation in animals has been chrysotile but there is every reason to believe that the other fibrous silicates would produce similar effects for in human beings the reactions to all members of this group are apparently indistinguishable. When guinea pigs, albino rats and rabbits were exposed simultaneously to chrysotile dust of an average concentration of 43,000,000 particles per cubic foot of air only the guinea pigs developed significant reactions. (Gardner and Cummings 1931) [30] Apparently the

upper respiratory protective mechanisms of the rabbits and rats were adequate to exclude fibrous foreign bodies. After about seventy days exposure, asbestosis bodies had developed about fibres of chrysotile within the air spaces of the guinea pigs lungs. These asbestosis bodies closely simulated those found in human beings but were considerably smaller. They presented the same golden-yellow color, the club-shaped terminal swellings and the transverse segmentation of the iron-containing deposit upon the mineral fibres. They were located very largely in the alveoli opening along the sides of the respiratory bronchioles. Many of them were free ; some were surrounded by giant cells of the foreign body type. As the inhalation continued more of them formed in the same location where they either remained or penetrated the wall of the bronchiole. None could be found in the intra-pulmonary or tracheobronchial lymphoid tissues. Only the non-fibrous particulate elements of the dust were transported to the lymphatic system.

With further exposure the walls of these air spaces, penetrated by the fibrous foreign bodies, gradually became thicker due to an infiltration with mononuclear leucocytes. After fifteen months a moderate amount of fibrous tissue had formed, which surrounded the bronchiole like a sleeve. At the time it was believed that contraction of this fibrous sleeve would close the tube and produce an atelectasis of the peripheral air spaces. Study of the further stages of the disease has failed to confirm this prophecy. Instead, the reaction seems to extend peripherally along the bronchiole apparently because the foreign bodies are deposited further along its course. This, in turn, seems to be due to the smoothing of the inner surface and perhaps the loss of elasticity of the walls of the tube. As more fibres are inhaled they apparently pass through the smooth, inelastic bronchiole and are deposited in the alveolar ducts given off at its terminal end. These tubes in

their turn respond to the local irritation and their walls become fibrous. In time the still more distal atria are similarly involved. Although appreciable reaction in the alveolar sacs and alveoli has not been observed within two years and nine months of exposure, it now seems more probable that these structures are eventually involved by the process of peripheral extension.

In another group of guinea pigs it was demonstrated that the reaction to inhaled asbestos dust is not a progressive one. The animals were exposed to the same concentrations of chrysotile as used in the first test for a period of eight months and twenty days. They were then removed to a normal atmosphere and members of the group were killed at intervals during a subsequent period of three years. At the end of the exposure, chronic inflammatory reaction had developed in the walls of the respiratory bronchioles. During the next three months this process gradually progressed to form a slight amount of fibrous tissue. Thereafter the reaction was, if anything, retrogressive in character. At the end of the three year period there were very thin sleeves of fibrosis about the bronchioles but no extension into more peripheral air spaces had occurred.

In 1937 a third experiment with chrysotile was begun. This time the asbestos fibres were ground in a steel ball mill until practically none more than 3 microns in length were left. It was anticipated that, if the irritation were of a chemical nature, the reaction should proceed more rapidly. Average concentrations of 175,000,000 particles per cubic foot (light field count) are being maintained in the dusting room. Thus far *the response to 4 times the concentration of a much finer dust has been less pronounced than that obtained in the first experiment.* Instead of being held on the irregular surfaces of the respiratory bronchioles, the particles have passed immediately into the terminal air spaces. The very short fragments

of asbestos are surrounded by a typical golden-yellow coating and have the appearance of minute asbestosis bodies. Practically all of them are found within the bodies of mononuclear phagocytes. Only a few can be detected in the interstitial tissue. Most of these seem to be intracellular. Some have been transported to lymphoid tissues. Evidence of chronic inflammatory reaction in the terminal bronchioles and other parts of the pulmonary stroma are practically absent. Compared with animals exposed for similar periods to the longer fibres of chrysotile in the first experiment, the picture is very different and more nearly resembles that produced by inert dusts.

These experimental observations seem to be indicating that asbestos fibres cause fibrosis in the lungs because they act as mechanical irritants in an organ whose function involves considerable movement. The long fibres cannot effectively be removed by phagocytes and the lymphatic system ; they penetrate the walls of the bronchioles and with each respiratory effort they continue to injure adjacent cells. The development of a protective coating about them must materially reduce the friction but as long as the inhalation continues new uncoated fibres are constantly accumulating to keep up the irritation. This is inferred from the lack of progression after the exposure has ceased. If the irritation were chemical in nature one would expect that it would develop more rapidly with finer than with large fibres, that it should be produced by injection into organs other than the lungs, as is the case with free silica, and that serpentine, whose composition is identical with that of chrysotile, should also cause fibrosis. But none of these effects have been observed. Final proof of the mechanical action of asbestos upon the lung is still lacking ; it remains to demonstrate that the inhalation of pure, very long fibres of chrysotile will produce results even more quickly than the dust used in the first experiment, which contained only a limited amount of such

material. However, the findings in the various experiments already finished all point to mechanical irritation. Since summarizing these observations a paper by Sundius and Bygdén [31] (1937) has come to hand. As a result of elaborate chemical and petrographic analyses of asbestos dust and asbestosis bodies these writers have reached the same conclusion.

The experimental exposures have thus far failed to reproduce the widespread fibrous lesions which have been observed in a few of the human cases of asbestosis. Where the latter are uniformly distributed throughout the lungs but retain their characteristic fine, interstitial arrangement they may be considered as the result of prolonged and intensive exposure. But where a human case reveals localized areas of massive fibrosis, it seems most probable that infection has complicated the picture. The majority of tuberculous infections in guinea pigs with asbestosis tend to spread for a time but subsequently heal with the formation of considerable areas of fibrosis. Likewise, accidental non-tuberculous infections of the lungs may heal and organize to produce fibrosis. The similarity between these foci of healed tuberculous and non-tuberculous infections in experimental animals and certain massive areas of fibrosis in the human cases strongly suggests a common origin. Furthermore, in guinea pigs the interjection of the factor of infection causes the fibrosis to become progressive and it is felt that the same may be true of human cases. This may explain the belief, entertained by certain authorities, that asbestosis is itself a progressive disease.

Infection and Pneumoconiosis

One of the important contributions to the study of pneumonoconiosis in recent years is the demonstration that silicosis specifically predisposes to tuberculosis and that the combined action of silica dust and the tubercle bacilli produces a chronic progressive disease which is responsible for

which there are large numbers of several types in use, by means of which the dust produced in drilling is drawn by suction into a collecting bag made of dust-proof material: (3) respirators, which can be worn where means for suppression or removal of dust are not practicable: (4) efficient ventilation of all parts of the mine workings especially those at which dust is produced by drilling, and fumes and dust are produced by blasting: (5) protection from currents of cold air of workmen while being brought out of hot pits on tramways.

Compensation. The Various Industries (Silicosis) Scheme, 1931 [4] as amended by the Order of 22nd October, 1934 [52] provides for the payment of compensation in respect of disablement or death from silicosis for all workmen employed in *"any operation underground in any coal mine."*

Asbestosis

Asbestosis is described in the Official Memorandum on Silicosis and Asbestosis [53] as a disease of the lungs produced as a result of the inhalation of asbestos dust, usually over a period of several years, and occurs in workers in occupations associated with production of asbestos dust in a fine state of division. It is characterised by a special type of fibrosis of the lung tissue which resembles silicosis in its general features of causation and development, but differs from it in some of its clinical and pathological manifestations.

Asbestos, which causes the disease, is defined in the Asbestos Industry Regulations 1931 [54] as follows: — *"Asbestos* means any fibrous silicate mineral, and any admixture containing any such mineral, whether crude, crushed or opened."

This somewhat wide definition is limited in practice, by commercial and industrial usage, to a group of silicates having the common properties of finely fibrous structure and flexibility. There are two principal varieties, serpentine asbes-

tos or chrysotile, the 'white' asbestos, which is a hydrated silicate of magnesium with a small proportion of iron ; and the hornblende minerals, the most important of which are crocidolite, or 'blue' asbestos, and amosite, a brown coloured variety ; blue asbestos and amosite consist largely of iron silicates, with a small proportion of magnesium.

Asbestos is imported into Great Britain in increasing quantities, year by year, owing to a remarkably rapid expansion of its uses in industry. The chief sources of supply are Canada, Cape Province, Italy, Rhodesia and Russia. The valuable properties of the fibrous minerals include the facility with which they can be disintegrated into flexible fibres and spun into yarn and woven into cloth ; their resistance to heat and electricity confers valuable properties for insulation, and the resistance to acids, of some varieties, favours their use for chemical processes.

The numbers of persons employed in the asbestos manufacturing industries is difficult to compute, but it is probably between 3,000 and 5,000, and they include both sexes. The largest section of the industry is engaged in the manufacture of asbestos textiles and the products into which these enter : the processes include the preliminary disintegration of the crude mineral, carding the fibre, spinning, plaiting and weaving the yarn ; the making of insulating mattresses of asbestos cloth filled with loose fibre ; making brake-linings, fire-proof curtains, clothing, etc. These branches of the industry are largely centred in Lancashire and Yorkshire, and in and near London. Other branches of the industry are connected with building, engineering and shipping, and are spread over the centres where these are carried on. The non-textile products of manufacture include asbestos mill-board ; asbestos-cement sheets, tiles and pipes ; brake and clutch-linings ; packings and jointings ; electrodes and switch-board panels. Mixtures of asbestos with magnesia, kieselguhr and other materials are

used as cements or fillings for insulating boilers, engines, pipes and other parts. Many persons are employed in handling and applying these mixtures in buildings and ships in course of construction or repair.

The dust given off from asbestos in the processes of manufacture consists of fragments of fibres and small rounded or angular particles. The concentration of the dust-cloud varies within wide limits, the most important factors being the nature of the process and the variety of asbestos in use. In a series of samples of atmospheric dust taken by H.H. Watson [55] with the thermal precipitator the following concentrations were noted :

TABLE 4

Process.	Asbestos.	Position.	Particles. (in 1 c.c.)	Fibres. (in 1 c.c.)	Total.* (in 1 c.c.)
1. Carding	White.	B.L.	235	180	415 (12)
2. do.	do.	G.A.	120	95	215 (6)
3. Doubling	Blue.	G.A.	105	95	200 (6)
4. Spinning	do.	G.A.	115	120	235 (7)
5. Weaving (cloth)	White.	B.L.	240	85	325 (9)
6. do.	do.	G.A.	55	40	95 (3)
7. Weaving (brake-lining)	do.	B.L.	140	70	210 (6)
8. do. do.	do.	G.A.	130	55	185 (5)
9. Winding	Blue.	B.L.	200	240	440 (13)
10. Plaiting	White.	B.L.	120	50	170 (5)
11. do.	do.	G.A.	120	85	205 (6)
12. Mattress making	Blue.	B.L.	405	820	1,225 (35)
13. do.	Various.	G.A.	35	25	60 (2)
14. Bagging	Amosite.	G.A.	210	210	420 (12)
15. Disintegrating	do.	G.A.	190	260	450 (13)
16. Making sections	do.	B.L.	690	1,630	2,320 (66)
17. do.	do.	G.A.	150	170	320 (9)
18. Pipe making	Blue.	B.L.	90	190	280 (8)
19. do.	Amosite.	B.L.	110	215	325 (9)
20. do.	do.	G.A.	80	135	215 (6)

B.L. = breathing level of worker ; G.A. = general air of the room.
* The figures in parenthesis represent the number of millions of particles in a cubic foot of air.

The median length of the *fibres* in 5 counts was between 2.65 μ and 3.5 μ ; from 8 per cent to 16 per cent less than 1 μ and 8 per cent to 13 per cent over 10 μ. In one sample the thickness of the fibres had a median diameter 0.56 μ, 5 per cent were less than 0.2 μ and 98 per cent less than 2 μ. The median sizes of the rounded or angular particles in 5 counts

varied between 0.46 μ and 0.8 μ diameter; 32 to 54 per cent. were less than 0.5 μ and 96.6 to 99.9 per cent. were less than 5 μ.

The history of the recognition of asbestosis as a serious industrial disease, and the increase of knowledge regarding it, are described elsewhere, but it may be stated here that the first case recorded in England was that of a card room worker, who died in 1899, the last survivor of 10 men employed in the same room. The case was described by Dr. Montague Murray in 1906.[56] During the same period a similar state of affairs existed in France, when in 1890 a textile asbestos factory was established at Condé-sur-Noireau (Calvados) and 16 workers died of pneumoconiosis between 1890 and 1895.[57]

Merewether [58] examined 363 workers in various processes in the asbestos industry and found in 95, or 26.2 per cent, definite fibrosis due to asbestos dust. Roentgenological signs of a diffuse fibrosis were found in 52 out of 133 persons examined. These figures do not represent the average incidence in the industry as a whole, because, for the clinical examinations, workers with longer terms of service were selected, and for the radiological examinations those with some clinical or other suggestive sign were given preference. There was, however, no undue proportion of workers from the more dusty processes. Age, considered apart from duration of exposure, and sex, appeared to have no influence on the onset of fibrosis.

During the seven years 1930 to 1936, 78 deaths were certified due to asbestosis in England and Wales. 40 of these were women and 38 were men. The numbers vary between eight and sixteen in the individual years, but they show no tendency towards diminution in the later years of the period. This is due to the delayed development of the disease, which in 42 fatal cases showed an average of twelve years exposure, and the fact that the gravity of the danger from asbestos dust

was not realised until 1924, although, as already mentioned, Montague Murray had recorded a fatal case and referred to others in 1906.

Prevention. The Asbestos Industry Regulations 1931 [54] were made following a medical and engineering inquiry into conditions in the industry by Merewether and Price.[58] The Regulations apply to all factories and workshops in which any of the following processes are carried on : —

(i) breaking, crushing, disintegrating, opening and grinding of *asbestos,* and the mixing or sieving of *asbestos,* and all processes involving manipulation of *asbestos* incidental thereto ;

(ii) all processes in the manufacture of *asbestos textiles,* including preparatory and finishing processes ;

(iii) the making of insulation slabs or sections, composed wholly or partly of *asbestos,* and processes incidental thereto ;

(iv) the making or repairing of insulating mattresses, composed wholly or partly of *asbestos,* and processes incidental thereto ;

(v) sawing, grinding, turning, abrading and polishing, in the dry state, of articles composed wholly or partly of *asbestos* in the manufacture of such articles ;

(vi) the cleaning of any chambers, fixtures and appliances for the collection of *asbestos* dust produced in any of the foregoing processes.

The Regulations require that machinery, used for processes in the manufacture of asbestos giving rise to dust, be so constructed and maintained that dust cannot escape otherwise than by mechanical exhaust draught which prevents the escape of dust into the air of a workroom. Mechanical exhaust draught is also required at processes in which asbestos is manipulated.

Special requirements apply to the making and repair of

insulating mattresses, one of the most dangerous processes in the industry owing to the difficulty of controlling the finest dust. Ventilation of the room must be approved in writing by the Chief Inspector ; whilst filling, beating and levelling are being done, persons not actually employed on such work are excluded from the room ; damping of floors, benches and material is prescribed.

Cleaning of the cylinders of a carding machine is no longer allowed to be done by hand tools, and persons not engaged in the work are excluded from the room. In practice, suction apparatus is now used and this is one of the most important preventive measures.

A breathing apparatus or approved respirator must be provided for and worn by persons employed in chambers containing loose asbestos, in cleaning dust collecting apparatus, and in filling insulating mattresses. Persons under eighteen years of age must not be employed in any of these processes, or in sack-cleaning or blending asbestos by hand.

In all rooms in which the processes are carried on, floors, benches and plant must be kept clean and free from obstructing material or articles not immediately required, and every room must be adequately lighted.

Initial and periodical medical examinations may be of great value as a means of preventing or arresting the development of asbestosis. Such examinations are prescribed under the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931 [26], as amended in 1934 [27], for workmen employed in the processes of breaking, crushing, disintegrating, opening or grinding asbestos, and in mixing or sieving of asbestos or any admixture of asbestos, and in making asbestos textiles and insulating mattresses. The periodic examinations are carried out by the Silicosis Medical Board at the prescribed intervals of twelve months.

Compensation. The Asbestos Industry (Asbestosis)

Scheme, 1931 [59] provides for the payment of compensation by employers of workmen who contract asbestosis due to employment in those processes in the industry in which the disease is liable to occur. The processes are as follows : — breaking, crushing, disintegrating, opening and grinding of asbestos, mixing or sieving of asbestos or any admixture of asbestos ; the manufacture of asbestos textiles ; the making of insulation slabs or sections or mattresses composed wholly or partly of asbestos ; other manufacturing processes carried on in the same room as any of the foregoing processes ; the cleaning of machinery and plant used in the processes, and of chambers, fixtures and appliances for the collection of asbestos dust ; the sawing, grinding and turning in the dry state of articles composed wholly or partly of asbestos, except articles composed wholly or partly of woven asbestos impregnated with bitumen or other bond of an adhesive nature.

The Prevention of Silicosis

Silicosis, like every occupational disease, is preventable. The long latent period between first exposure and the appearance of symptoms, and the chronic course of the disease tend to obscure the connection between the fatal result and the conditions which brought it about. This leads to the argument that the conditions which existed in this or that industry thirty or forty years ago were responsible for most of the silicosis mortality of today. That may be true of some industries but conditions do still exist which are capable of producing silicosis and new processes have been introduced without due regard to this danger.

The most important measures for preventing silicosis are (1) abandonment of the process ; (2) finding a safe substitute for a dangerous material ; (3) suppression of dust immediately on its production ; (4) removal of dust near its point of origin so that it does not reach breathing level ; (5)

conditions was limited to the small number of those whose investigations of pulmonary dust diseases had made them conversant with the subject.

Arising out of the period of economic depression, the situation with respect to silicosis and asbestosis became manifest as a medico-legal phenomenon of a scope and intensity that was at once preposterous and almost unbelievable. Damage suits, under the common law, were instituted against employers by employees, alleging pulmonary dust diseases, in industrial centers all over the United States, to an amount in excess of 100,000,000 dollars. To understand this situation, both in its economic and public health aspects, it is necessary to review not only the history of silicosis investigations in the United States but also the status of compensation for occupational diseases in the various States.

The reader is referred to the first chapter of this book. Briefly, governmental agencies began studies of silicosis in the mining industry in 1914 and these were carried on until they were interrupted by the entry of the United States into the World War. These studies had for their purpose the detection of silicosis, the extent and severity of the disease in the areas studied and the establishment of methods of control. The question of compensation did not arise, which must seem strange to the non-American student of the subject.

In almost every State of the Union, there is a State Department of Labor and Industry. These State Departments are charged with the administration of the laws pertaining to factories, mines, and workshops. Previous to 1930 only 3 or 4 Labor Departments included in their activities a division of industrial hygiene and these few were not established on a basis that made possible original studies or research. They were mostly concerned with routine inspections and also endeavored to enforce laws and regulations pertaining to

health, whose vague wording often made them almost non-enforceable.

The State Departments of Health, although their functions might be thought to embrace public health in industry, uniformly ignored the subject altogether, with the exception of Ohio and Connecticut, where occupational diseases were made reportable. Following the widespread national attention focussed upon occupational diseases by the silicosis furor, this situation has entirely changed and at present (1937) State Divisions of Industrial Hygiene are, with a few exceptions, located in the State Departments of Health.

The Federal Government embraces several agencies that concern themselves with occupational diseases and industrial hygiene. In the United States Department of Labor, the Bureau of Vital Statistics has been actively engaged in the field of investigation. In the United States Public Health Service, the Office of Industrial Hygiene and Sanitation has likewise been engaged in industrial studies and at the request of and in co-operation with the United States Bureau of Mines investigated dust diseases. These governmental departments have jurisdiction in industrial establishments of a governmental nature only, except that by virtue of recent legislation, the United States Department of Labor may supervise conditions under which work is done under a contract with the Federal Government. The Government officers enter a mine or a factory only with the permission of the owners and usually at their request. The Federal officials have no power to enforce existing State laws nor to initiate new laws or regulations. Reports of silicosis investigations, when published by the department responsible, might and did contain suggested methods for the prevention and control of this disease but they were addressed not to the State authorities nor to the industrialist but to the general public as contributions to the

existing knowledge. Both State officials and industrialists might profit by this new knowledge if they so wished. Usually they did so and a great many improvements were instituted but these were largely confined to those industries that had been studied at first hand. In fact, during the period 1914–1929, silicosis was considered a serious hazard only in certain phases of the mineral industry, with the exception of a few specific occupations, such as sandblasting. Asbestosis was practically unknown.

To summarize, the investigations of silicosis during the period 1914–1930, and of both silicosis and asbestosis for some time subsequently, were scientific (embracing both medical and engineering aspects), informative and non-legislative, with the exception of the study of silicosis among rock drillers made in New York City in 1928 under the auspices of the New York Tuberculosis and Health Association.[1] In this instance, definite recommendations regarding workmen's compensation were made.

Since 1912, in Massachusetts, occupational diseases have been compensated by court interpretation of the wording of the accident compensation law. In 1914, no other State had made provision for compensation for occupational diseases. By 1930, however, 10 States and 3 Territories had enacted laws making occupational diseases compensable.[2] In addition, there were 3 Federal laws, affecting employees of or under the jurisdiction of the United States Government. At present (November, 1937), 21 States have some form of occupational disease compensation and of these, 16 include silicosis and other dust diseases. When the time came to enact legislation bringing occupational diseases within the scope of the compensation acts, some of the States followed British precedent and enacted a schedule of the diseases which were thus made compensable. In the other States, occupational diseases were covered under a general description such as 'diseases arising

out of and in the course of occupation,' without further designation — the so-called 'all inclusive' or 'blanket law.' The Federal Government followed the latter method in providing compensation for its employees.

Between 1914 and 1930, the total amount of compensation paid for occupational diseases was very small, averaging not more than a minor fraction of the accident compensation cost in the various States. Interestingly enough, up to 1935, not one State operating under a schedule included silicosis, asbestosis, or pneumoconiosis in the list of compensable diseases, a situation difficult to understand, considering that silicosis is the most truly occupational of diseases, that reports of studies of silicosis in the United States had been forthcoming for twenty years and that there was available a vast amount of literature embodying the experience of England, South Africa, Australia, and Canada. In the States having 'all inclusive' coverage, the number of awards for silicosis was negligible.

The explanation of this curious state of affairs is, probably, that silicosis was considered as distinctly a hazard of mining. In those mining States, where hard rock mining was a major industry, there was no compensation for occupational diseases nor was there any pronounced demand for such compensation. 'Eating dust' was looked upon by the hard rock miner as a natural danger of his trade. Mining companies usually pensioned older employees disabled with silicosis or saw that they were cared for in one way or another. Also, following the governmental reports, the mining companies concerned started to put their mines in order, spending large sums of money on ventilation and dust control.

Then, with dramatic suddenness, came the era of the silicosis damage suits. Lawyers who specialized in this type of law practice instituted suits for damages under the common law on behalf of employees of various corporations for injury to the lungs due to the inhalation of dust. They were aided not

only by the general and increasing unemployment but by the almost total ignorance of industry — aside from mining — concerning pulmonary dust diseases and by the absence of silicosis in the compensation schedules which enabled them to bring suits for amounts far in excess of the limits of compensation. An interesting exception occurred in one or two States — Ohio, for instance — where the absence of silicosis from the schedule was held to bar suits or claims for this disease either under compensation law or common law. In those States where there were no occupational disease laws, claimants were at liberty to sue under the common law.

Among the first claims for damages were those for asbestosis, a hitherto unheard of disease in the United States.[3] Damage suits multiplied by hundreds, starting on the Atlantic seaboard and extending to the industrial States of the middle West and far West. The terms 'pneumoconiosis,' 'silicosis,' 'asbestosis' were strange and unfamiliar to judges, juries, and industrialists and were enveloped in medical and pathological technicalities. Consequently, the claimant's witnesses and medical experts, as far as their prestige with juries was concerned, carried as much weight as did those of the defendants. So great was the confusion and lack of knowledge on the part of courts, juries, and physicians that awards were made for silicosis to men who had never been exposed to the dust of free silica. Many of those seeking damages undoubtedly had silicosis — many did not. Damage suits were filed against cement companies and against mining companies whose operations were in limestone formations where there could be no possibility of a silicosis hazard.

All of this might be dismissed as a historical and medicolegal curiosity except that it greatly modified existing compensation laws in the United States and stimulated the passage of new laws. Commissions were appointed by several of the States to inquire into and recommend legislation for compen-

sation of occupational diseases and especially silicosis. It was generally felt that it was not sufficient merely to add silicosis to an existing schedule or include it when a new schedule was planned but it was essential to make special provision for regulating and limiting the benefits to be paid for silicosis. Many industries had acquired through the years considerable numbers of employees who had, or might be deemed to have, silicosis; industrialists, insurance companies, and the State funds feared that simply to make silicosis compensable was to invite a deluge of claims that would exhaust existing funds and make insurance rates prohibitively high. In most of the enacted laws, a fixed period of residence within the State was stipulated to prevent silicotics from migrating from a State where there was no compensation to one where there was.

The problem of compensation for a chronic illness as contrasted with an acute one presented difficulties, inasmuch as it did not seem that the insurance principle, on which all compensation in the United States is based, could be successfully applied to disability or to diseases already existent and whose extent was problematical. With business conditions as they were, the industries did not feel that they should be compelled to establish special silicosis benefit funds to provide for accumulated cases which had occurred under conditions where there was no statutory requirement imposing an obligation upon the employer which he had failed to meet. Moreover, it was often evident that silicotics had not contracted their disease in the place of present employment but in a previous one and, perhaps, in another State. The attitude of the average industrial firm was that, being shown that they had a silicosis hazard, they were willing and anxious to clean up and endeavor to control dust and to pay compensation to those who might subsequently contract silicosis but that they should not be required to pay for disability from a disease of whose existence they were previously unaware. The various commissions

and legislative committees endeavored to iron out these complexities as best they could, having in mind their own local conditions.

Some years previously, the Province of Ontario had assumed the attitude that, tuberculosis being a disease common to the human race generally, when silicotics contracted tuberculosis, their compensation benefits for disability should be diminished and such a provision was enacted into law.[4] A similar economic point of view, regardless of its logic or justice, influenced those engaged in drafting legislation in the various States, so that when silicosis legislation was finally enacted, in some of the States the individual disabled by silicosis received less award than one suffering from any other compensable occupational disease. Furthermore, several of the State Committees took the position that uncomplicated silicosis, as observed in American industries, was not disabling and that disability did not occur until infection (tuberculous) had supervened and then the disability was considered total. Hence, in some States, partial disability due to silicosis is not recognized.

The New York law,[5] as in some other States, provides that compensation shall not be paid for partial disability due to silicosis or other dust diseases. For permanent disability or death, the awards are fixed according to a sliding scale as follows: For a total disability or a death claim, presented within the first month in which the Act becomes effective, not exceeding $500; for every subsequent month thereafter, $50 is added, but in no event shall the compensation exceed the aggregate of $3000. The maximum for total disability from other occupational diseases or from accidents may be considerably more, depending upon the age of the deceased and upon the number of dependents.

It was hoped that the sliding scale would have a twofold effect. It would safeguard the continuity of employment by

spreading the economic burden which was expected to result from these laws and it would also tend to induce the workman with an early silicosis to keep on working. In addition, it would stimulate the employer to vigorously attack his dust problem with the hope of averting total disability claims.

In Pennsylvania, the occupational disease law [6] provides that for diseases which develop to a point of disablement only after an exposure of five years (to be determined by the Workmen's Compensation Board), compensation costs for disability or death, arising during the period of ten years succeeding the effective date of the Act, shall be paid jointly by the Commonwealth and the employer as follows: In the first year in which the Act becomes effective, one tenth by the employer and the remainder out of funds controlled by the Commonwealth. In each succeeding year, the employer pays one tenth more, the Commonwealth one tenth less, to the expiration of the ten year period. Partial disability for silicosis, anthraco-silicosis, and asbestosis is not recognized and the total liability of the employer for these diseases is limited to $3600.

In North Carolina, the recently enacted compensation law [7] states that in silicosis or asbestosis, compensation shall be paid as provided in the Act but if either is complicated by tuberculosis, the rate of payment may be reduced one-sixth.

While some of the foregoing might seem to lie in that realm of reasoning so difficult of comprehension to the non-legal mind, the underlying motive was not a desire to deprive working men of their just dues but a feeling that it was necessary to place a drastic check on the expected avalanche of silicotic claims. Undoubtedly, the common law damage suits confused and terrified industrialists and insurance officials. As a matter of fact, many of these suits were never brought to trial, many were compromised by settlements out of court, but those that did come to trial were won by claimants in sufficient number

to inspire dread among all exposed to this type of legal action. If these apprehensions prove to have been unwarranted it is probable that in the next few years, Silicosis Acts may be modified to make their awards more in line with the general trend of compensation.

The North Carolina Act also stated that if an employee is affected by silicosis or asbestosis, he shall be taken out of employment and paid partial or total disability until he can obtain other occupation. The State will train him for other occupation at State expense. If later he returns to a job with a silicosis hazard, he forfeits compensation benefits.

The experience of Wisconsin is very instructive and for a description of the complexities of silicosis compensation in one State, the reader is referred to the informative article by Kossons and Fried,[8] from which the following figures were obtained. Wisconsin is an important industrial State with mines, quarries, foundries, and enamel ware industries, besides many others. During the seventeen year period — 1920–1936 inclusive — 893 claims for disability from silicosis were brought before the Industrial Commission. Of these, 469 claims were allowed (one third of them were death claims) and 55 per cent of these were filed during 1933–1934. A comparison of costs for the fifteen years — 1921–1935 — shows that for the entire period, the cost of silicosis compensation was 2.23 per cent of the total cost, including and in spite of the peak years of 1933–1934, when it was 9.40 per cent and 8.80 per cent respectively.

Extent and Nature of Hazard

It is not possible to estimate accurately the number of persons exposed to the harmful effects of silica dust. Comprehensive statistical information, based upon accurate death records, is lacking. Silicosis is not included in the list of reportable diseases. Many of the deaths due to silicosis with

tuberculosis have been recorded simply as tuberculosis. Neither hospital records nor death records customarily give a workman's industrial history — indeed, it is unusual for even the present or final occupation to be stated with sufficient clarity to be of value to the investigator. Morbidity records are of less avail than mortality records, and physical examination of new employees in industrial establishments, while fairly common, have rarely been of such a nature as to reveal early pulmonary disease. Even when industrial conditions are good, there is considerable movement of labor from place to place and from State to State. When times are bad, this migration is increased. Unemployment then drives men not only from place to place but into new occupations.

Lanza and Vane [9] estimated that there were about 500,000 industrial workers employed in occupations exposed to silica dust. It is more than likely that this estimate errs considerably on the conservative side. This estimate does not take into consideration the thousands of men who have been employed where there was a silica hazard and who are now scattered throughout industry.

The menace in silicosis has always been tubercle infection. The evidence [10] here is overwhelming and has been stressed by investigators throughout the world. In 1927, Watkins-Pitchford [11] stated : "In ascertaining, from our records, what the liability of the simply silicotic miner to become tuberculous has been in the past, account has of course been taken of all instances in which the change has been detected during life ; in addition to this, however, it has been assumed that, in those persons suffering from simple silicosis who have died of their disease without having been again examined, death has been the result of this change. This is an assumption which is warranted in practically every instance. Relying on this assumption, and also bearing in mind that, as already stated, the liability to tuberculous change has appeared less marked

in each succeeding annual batch of cases, one finds that the combined results of seven years' observations show that, by the end of the seventh year, a little more than half of the cases of simple silicosis have either developed overt tuberculous changes or have died as a result of those changes."

Collis [12] also bears witness : "Silica dust alone possesses the further characteristic that it increases the liability to succumb also to pulmonary tuberculosis."

Kettle [13] states the case very clearly : "The silicotic lung appears to be a more favorable medium for the growth of the tubercle bacillus than the normal lung, not because it is fibrotic, nor because its lymphatic drainage is interfered with, but because it contains silica."

Much research and study have been applied to the question of infection in silicosis but in spite of the fact that the nature of the relationship between silica and the tubercle bacillus is still to be determined, the reality of this relationship is affirmed by recent investigators. Irvine [14] and his associates analyzed 543 deaths in 1923, cases originally certified as having simple silicosis. Approximately 75 per cent of these deaths were due to tuberculosis. They note that this inherent predisposition to tuberculosis is as marked in very early (anteprimary) as in later stages (primary).

Simson and Strachan [15] state : 'The importance of the infective factor in many and ultimately in the late stages of most cases of silicosis has always been recognized in South Africa, as elsewhere.' They comment further that many cases are infective from the outset, in others there may be a reactivation and local extension of pre-existing dormant foci of infection or there may be a new infection from outside.

The experience in the United States is similar. Meriwether [16] states for the Picher, Oklahoma District 'The production rate of tuberculosis (exclusive of old cases that progressed) was 0.75 per cent or 750 per 100,000 population,

which, for 1929, is certainly higher than that of the general population of the country.'

The following is quoted from the United States Public Health Service report on Anthraco-Silicosis among Hard Coal Miners : [17] "Several surveys have shown that tuberculosis of the lungs occurs among 1 to 2½ per cent of the general adult white male population of the country. In a study of tuberculosis in Framingham, Mass., it was found that about 1 per cent were suffering from the disease in an active form, and another 1 per cent were classified as having arrested tuberculosis.[18] Physical examination of 100,924 adult white males made by the Life Extension Institute indicated a prevalence rate of about 1½ per cent when suspected cases were included.[19] A somewhat higher percentage, namely, 2½ per cent, was found by the Public Health Service from the examination of 10,000 male industrial workers.[20]

"Among the anthracite workers examined, the clinical tuberculosis rate was below normal in the younger adult ages, but at ages 35 to 44 clinical pulmonary tuberculosis was diagnosed in about 5 per cent of the hard-coal mining employees ; at ages 45 to 54 in 10 per cent ; and at ages 55 to 64 in 20 per cent. No such rise with age occurred in any general population group for which comparable data are available.

"The prevalence of tuberculosis was greatest among the rock workers. The next to the highest rate occurred among anthracite workers who had changed more than five years previously from very dusty to relatively nondusty occupations in the industry. The third highest rate was exhibited among persons who had had appreciable exposure to harmful dusts in other industries. Among the regular miners working at the face the rate was definitely higher than in the control group which showed a prevalence rate of less than 1 per cent.

"When the term of service exceeded twenty years, more than two or three of which involved exposure to heavy concen-

trations of rock dust, about 37 per cent of such employees (classified as rock workers) showed evidence of pulmonary tuberculosis. Service of twenty-five to thirty-four years was associated with a tuberculosis rate of 8 per cent among non-rock workers employed in the haulageways, of 14 per cent among the regular miners, but with a rate under 2 per cent among men exposed to less than 5,000,000 dust particles per cubic foot of air."

As has been pointed out on numerous occasions, the severity of an individual case of silicosis depends upon the extent of dosage of silica; that is to say, upon the duration of exposure together with the intensity of silica dust concentrations in the air. The early investigators of silicosis in the mining industry in the United States were confronted by working conditions of the utmost hazard — that is, a maximum amount of silica dust in the air with no provisions for its control nor for the protection of the men exposed. Hazards of this severity have happily disappeared except, perhaps, in isolated instances; certainly such conditions are no longer industry-wide.

Some recent investigations reveal a degree of exposure which is manifested as a disabling illness after middle age has set in — characteristic of certain phases of the foundry industry.[21] Again other studies have shown the existence of silica hazards sufficient to produce characteristic roentgen-ray appearance but without disability that can be directly charged against pulmonary fibrosis. It seems fairly certain that the industrial hygienist of the future will not see the typical cases of far advanced silicosis, presenting roentgenograms with "snow storm" appearance and areas of massive fibrosis described by early investigators both in this country and abroad.

On the other hand, the tendency to minimize the silica dust hazard which has been manifested on recent occasions is not justifiable. If there is one thing that stands out with con-

spicuous regularity in the vital statistics of this and other countries, it is the high incidence of tuberculosis and other respiratory diseases in the silica dust trades.[9] [22] We know that where silica has been inhaled in sufficient quantity to produce its characteristic effect, a definite condition of susceptibility to tuberculosis has been established. We should hesitate to infer, therefore, that exposure to silica dust to an extent insufficient to produce characteristic nodulation may not carry with it implications in terms of tuberculosis and other respiratory diseases. From a public health standpoint and for the promotion of healthful conditions of work, there is demanded constant attention to dust hazards in industry with the utilization of every practical method for the prevention of atmospheric pollution.

Asbestosis

Asbestosis was first described in the United States by Pancoast, Miller, Smyth and Landis in 1918, but its importance was not realized until it was introduced into the damage suit situation which led to the first comprehensive study of this disease in the United States.[3] Recent years have seen an immense increase in the use of asbestos, both for brake linings of automobiles and for heat resisting and insulating materials. With relation to public health, asbestosis is not nearly as serious a problem as silicosis. The number of persons in the industries using asbestos is very much less than for silica and in the United States amounts to about 12,000 factory workers, of whom approximately 25 per cent are women. Not all of these are exposed to dusty processes.

Most of the asbestos used in the United States is imported from Canada. It has been suggested that the various types of asbestos differ in their ability to cause pulmonary disease, but so far no proof of this is forthcoming.

Much of the industrial exposure arises from textile proc-

esses in which dust control measures can be readily instituted. As soon as the hazard was realized, industrial firms fabricating asbestos took energetic steps to control the dust so that it is probable that cases of asbestosis will become uncommon. A recent report from the United States Public Health Service details dust control methods instituted in an asbestos textile plant which illustrates in a striking manner what can be done to alleviate dust.[23]

Asbestosis has not appeared to be as serious a disease in the United States as it has in England, judging from the various reports from the latter country. Asbestos "mattress making," mentioned in the British reports as a serious hazard, is a process not found in this country. The role of infection in asbestosis is not as clearly defined as in silicosis and the clinical evidence available indicates that tuberculosis does not have the intimate relationship with asbestosis that is so conspicuous in silicosis.[24] [25]

Effects of Silicosis upon the Families of Silicotics

From the earliest times, when silicosis was first described as a disease of miners, the fact that the wife did not contract tuberculosis from the husband was mentioned and the many recent investigations have not upset this general conclusion. Hoffman [26] stated in 1922, "The frequency of pulmonary tuberculosis among immediate relatives of granite cutters, but particularly wives, daughters, and mothers, is much less than, as a mere matter of probability, would be assumed to be the case." This was borne out by the later studies of McFarland [27] and Russell.[28]

Cummings of the Saranac Laboratory, who investigated this subject in connection with the Picher Clinic, commented on the fact that, despite the wretched living conditions, and the lack of cleanliness and sanitation in many homes of advanced silicotics whose sputum was loaded with tubercle bacilli, no

adult type tuberculosis was found among exposed children up to the age of sixteen.

Nevertheless, these various testimonies do not exclude the necessity of treating tuberculo-silicosis as a communicable disease and of making follow-up investigations of members of families of advanced or disabled silicotics, to determine the presence of tubercle infection or the desirability of segregating contacts from the diseased member of the family. Sound public health procedure should be the rule and reliance should not be placed upon the supposed immunity of the family because the tuberculosis is complicated by or consequent upon silicosis.

Control and Prevention of Silicosis

The control of silicosis is both an engineering and a medical problem, as are so many other situations that concern the public health. Both the engineer and the physician, with their technicians, have their parts to play. In some instances, the medical phase may predominate ; in others, the engineering, but it is seldom that they can be separated or be independent of each other if a thorough job of prevention is to be done.

Engineering Control

The development of silicosis is dependent upon the dosage of silica and the matter of avoiding a dangerous quantity might be thought simple. The lay mind, familiar with the definiteness of the hazard of carbon monoxide, and other toxic substances producing acute effects, usually fails to appreciate the inherent difficulties of establishing a threshold limit for a substance producing a chronic disease whose development is measured in terms of years rather than hours.

From time to time, critical comment is directed toward the industrial hygienist by the engineer because the former has failed to inform the latter of the safe or threshold limit of

silica dust in the air of working places. That is not an unnatural criticism from those whose profession involves dealing with definite and measurable factors. Unfortunately, neither physiology nor pathology in their relationship to the incidence and progress of chronic disease has yet advanced to the point where they are susceptible of expression in terms as definite as those used by the engineer.

The circumstances under which silica dust is introduced into the air of working places varies to an extreme degree. Furthermore, these circumstances and others that, in turn, affect them, vary from day to day and from hour to hour; they are affected by wind and weather, by dryness and humidity. All this would be complex enough if we were dealing only with the dust of pure silica, but in actual practice, the silica dust is frequently combined with other substances which may modify its action and about which little is known. Some industrial processes lend themselves to dust control measures; others do not, and in these latter, an entire change of the process itself may be necessary to overcome the hazard.

Dust Sampling

To deal with any health hazard successfully, its nature and severity must be understood. For silica dust, the two factors involved are the proportion of silica in the particular rock or mineral substance from which the dust originates and the amount of dust present in the air breathed by the workmen exposed. A great deal of study and research has been concentrated on this subject. It is customary to define the extent of exposure by stating the number of dust particles, under ten microns in greatest diameter, in millions per cubic foot of air.

Various methods of counting the dust particles in the air have been devised, later to be discarded, until at the present time in the United States reliance is chiefly placed on the impinger method as defined and practiced by the United

States Public Health Service and the United States Bureau of Mines. The merit of the impinger is that it takes large samples, over a considerable period of time (one-half to one or two hours). The objection to the impinger is that it is cumbersome, time consuming, and demands expert technical skill if the results are to be trustworthy. Simpler devices consisting of smaller portable instruments, taking a small quantity of air — "grab samples" — and necessitating less skill are also used. The objection to these devices is that due to the smallness of the sample, the margin of error is considerable. Both methods have their place, but when serious studies of dust hazards are involved, it is probable that reliance will continue to be placed on the impinger method until a more satisfactory one is evolved. Small electric precipitators, modeled on the Cottrell precipitator, are in use but are mostly restricted to laboratory practice and to standardize the impinger. Other devices are still in the laboratory or experimental stage.

To summarize: While no one method of sampling air for the purpose of making dust counts has proved entirely satisfactory, up to now the impinger is accepted as most reliable in field work. No two methods are quite comparable; even samples taken by the same method in the same location and at the same time frequently show marked differences.

State Departments of Labor are naturally desirous of establishing standards of air dustiness for the control of the dust hazard and for the guidance of factory managers and factory inspectors. To a very limited extent, such standards have been suggested as a result of various government investigations.[28] However, much caution is needed here and too much reliance should not be placed upon the formulation of "standards" of air dustiness and the enactment of factory codes setting forth permissable limits of dust counts. Such threshold limits as have been worked out have very definite value as a guide in endeavoring to secure better working conditions but their

limitations and possible transitory nature should be well understood. They should not as yet be enacted into law, otherwise a situation analogous to that of school ventilation may be repeated. Some of our State laws and regulations controlling ventilation in schools provide for an air change of 30 cubic feet per minute per occupant — a highly expensive and troublesome provision. This standard was based on what was thought at the time these laws were enacted to be a correct conception of "pure" air and "bad" air. Our present knowledge of ventilation and atmospheric control has proved the inadequacy of this carbon dioxide standard but it is the law in many places. If history repeats itself in formulating threshold limits of air dustiness, the result will be not only vexatious but costly.

The first and basic principle in dealing with a dust hazard is to remove the dust at its point of origin and thus prevent its dissemination into the air. This is the only procedure to which the term prevention may be properly applied. Once the dust is in the air, the protection of the individual becomes difficult and often unsatisfactory.

The removal of dust at the point of origin may be accomplished by suction devices working on the principle of the well known vacuum cleaner. In certain processes, such as drilling, water may be used to advantage. In some instances, water and air suction may be combined. So great is the variety of industrial processes from which silica dust may arise that a detailed description is not feasible. Each type of process usually presents its own problems and difficulties to the engineer and what will work satisfactorily under one set of circumstances will not always be successful in another.

Once the dust has been disseminated into the air, its removal may present serious engineering difficulties. Water may be employed, either as a spray to increase the humidity and facilitate the settling of dust particles, or water blasts may be

used to accomplish the same results more rapidly and thoroughly. It is obvious that this method is limited in its application. The dust may be diluted by increasing the quantity of air through forced ventilation — again a method not always applicable and apt to be expensive. The two methods may be combined. Both methods, either singly or in combination, have been widely applied in mining operations.

Some experimentation has been done with the use of hygroscopic substances applied to the surfaces of mine passages to prevent dust from being continually stirred into the air. It has also been proposed to add to silica dust other kinds of dust with the hope of inhibiting the action of silica in the pulmonary tissues. While this method of combatting silica dust may be both practical and valuable under certain conditions, it is not likely that it will be accepted as a final satisfactory solution. The effects of long continued breathing of such mixed dusts are unknown. Boiling or chlorinating infected drinking water may be a necessary procedure but satisfactory hygienic standards require that the community be supplied with water from reasonably pure sources and enormous sums of money are spent to secure such water. Likewise the air that industrial workers must breathe should be reasonably pure. Air containing harmful quantities of an injurious substance plus a counter-acting or antidotal substance would seem to leave much to be desired with respect to purity and proven harmlessness. The industrial age has seen too many miracles of achievement and too many evidences of inventive genius to make one believe that satisfactory dust control is beyond our resources.

It is essential that where dust control devices have been installed that check tests be made from time to time to determine their continued efficiency. Such equipment tends to lose its value unless continuously supervised and maintained in good working condition. It is the common experience of

investigators to find installations of ventilating and dust removal machinery that are of little use due to failure to keep them in proper working order while at the same time they continue to give a feeling of unwarranted security.

Face masks, helmets, and respirators have been developed to protect the individual in dust polluted atmospheres. Their use is justified when the exposure, or the dusty process itself, is intermittent and of short duration. Filter type respirators, which are approved by the United States Bureau of Mines as adequate for protection against silica dust, can be relied on. They may be uncomfortable, especially when the temperature is high, and their continued use calls for a degree of co-operation from the individual workers which experience has shown is difficult to maintain. Positive pressure helmets and masks are generally efficacious when used under conditions for which they are suited. The adequate supervision and maintenance of protective equipment is all important. A faulty device or one improperly used will give a false sense of security that will eventually betray its user.

Medical Control

Pre-employment physical examinations, where a definite health hazard is involved, are an essential public health procedure, definitely important in controlling the incidence of occupational disease. The benefit should accrue impartially to the individual, the industry, and the community. It is also important that the function of the pre-employment examinations be understood so that they may not be abused. It is desirable to place men in types of work for which they are adapted. It is equally necessary not to bar, needlessly, employment to those seeking it. Much of the dispute and ill feeling engendered during the last few years on the subject of physical examinations, both pre-employment and for those already employed, as related to the silicosis furor, might have been

avoided if the basic reasons for such examinations had been understood and if employers and others concerned had thought their problem through. Some misunderstanding was inevitable under the general circumstances.

It is not practicable to lay down definite rules or to assume a dogmatic attitude. Circumstances vary in different occupations and different parts of the country and economic considerations always stand well to the fore and must be recognized.

Obviously no one with active or recently active tuberculosis should be exposed to silica dust, nor anyone suffering from other pulmonary disease, especially if of an infective nature, which would tend to increase the hazard. On the other hand, there is often no good reason for rejecting an applicant who already has silicosis, if the silica hazard in the work place involved is under control and if the workman's physical condition is properly supervised thereafter. This is an important consideration where the industry is a dominant one in its locality and other opportunities for work are limited. Here is where the experienced industrial physician must bring his judgment to bear both upon the individual and upon the work he is to do.

In connection with the recent compensation laws passed in some of the States, the attitude toward pre-employment examinations is interesting. The New York [5] law states, "It is hereby declared to be the policy of the legislature of this State, in enacting this article, to prohibit, through every lawful means available, any requirement to employment which compels an applicant for employment in any occupation coming within the purview of this article to undergo a medical examination."

As contrasted to this, the North Carolina [7] law states, (where the Industrial Commission has found an occupation to have a hazard of asbestosis or silicosis) "it shall be the

duty of every employer . . . to provide prior to employment necessary examinations of all new employees for the purpose of ascertaining if any of them are in any degree affected by asbestosis and/or silicosis or peculiarly susceptible thereto; and every such employer shall from time to time, as ordered by the Industrial Commission, provide similar examinations for all of his employees whose employment exposed them to the hazards of asbestosis and/or silicosis." At least one member of the Advisory Medical Committee or other physician designated by the Industrial Commission shall make such examination or be present when such examination is made. Further on, the law continues: "The refusal of an employee to submit to any such examination shall bar such employee from compensation or other benefits provided by this Act in the event of disablement and/or death resulting from an exposure to the hazards of asbestosis and/or silicosis subsequent to such refusal."

Particularly to be commended are the control methods set up in the Province of Ontario. A co-operative agreement is in force between the Workmen's Compensation Board and the Provincial Health Department. Examination centers are established where necessary and pre-employment and periodic physical examinations, with the use of the roentgen ray are made by a physician of the Health Department for the Workmen's Compensation Board. Examination certificates are issued to acceptable workers. Claimants for compensation come before a Referee Board of Physicians of the Health Department. This Board works in co-operation with the industries concerned and the Compensation Board.

Where workmen are exposed to a silica hazard, medical supervision goes hand in hand with engineering control of the dust. Indeed, it is the industrial physician who must finally determine whether the dust control measures in his establishment are efficacious. Annual, or perhaps bi-annual,

medical examinations with the roentgen-ray are usually sufficient with more frequent examinations of those individuals who, the industrial physician feels, need more constant supervision. As in any other circumstance where an occupational health hazard is involved, no mechanical method of prevention can be taken for granted.

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