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THE ROENTGENOLOGICAL ASPECTS OF PNEUMOCONIOSIS AND ITS MEDICO-LEGAL IMPORTANCE*

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WE HAVE been intensely interested in the roentgenological aspect of pneumoconiosis since 1916. This interest originated in a desire to learn something of the changes produced in the roentgenograms of presumably healthy adult chests by certain factors which were not definitely pathological but more or less generally operative in modern life among city dwellers and workers in certain occupations in which individuals were exposed to varying quantities of various kinds of dusts. This naturally led to investigations of workers exposed to harmful quantities and qualities of dusts, as a result of which definite and striking pathological pulmonary changes were induced. At the time of our preliminary communication (1), we were further stimulated by the reports on silicosis among the Rand miners in South Africa (2). It became a most fascinating study as added experience brought out new and interesting features. It became quite evident to us later that the physical, histological, and pathological processes involved in pneumoconiosis made the teaching of the roentgenological aspect of pulmonary tuberculosis to students a much easier problem.

Naturally a deep interest in one aspect of the subject of pneumoconiosis became conducive to a desire to delve into all of its various phases. It should be borne in mind that roentgenological appearances are always demonstrations of pathological processes of disease in the living subject and as they may be modified by the physiological functions of life.

It has seemed quite evident that the two most important ways of study-

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since roentgenology is such a powerful means of attack and defense in medico-legal questions. We stress these facts for the reason that we have heard so much inexpert testimony of the inexperienced offered as expert testimony and even accepted as such.

Our interest in all of these phases of the subject does not belittle the importance of the clinical examination, which has indispensable values, especially in relation to the various aspects or degrees of incapacity, particularly in the late stages of the disease, and in the detection of complicating tuberculosis and in differential diagnoses. But when roentgen examinations are made with the proper care and by the correct technic, and interpretations are based upon the factors mentioned, what information is there to be determined clinically which can compare in importance with the roentgen findings? Nevertheless, the clinical study is essential in every case in rounding out its complete status as a pathological problem. There are but few clinicians in this country as yet who have had sufficient experience to qualify them in the expression of authoritative opinions upon cases of silicosis, or pneumoconiosis of origins other than silica, although their opinion may be of very definite value in differentiating other conditions if present in the individuals in question. Moreover, even clinicians experienced in examining cases from one industry may not be qualified to express opinions upon cases in other industry occupations. Even the clinical detection of pulmonary fibrosis in its various degrees does not give a complete knowledge of the pathological process which may be present.

The technic of roentgenological examinations is of the utmost importance in all cases in which the exact status of the pathological condition is of unusual importance, should any exist. First a word in regard to routine fluoroscopic studies alone: These may be ample in the examination of a large group of individuals under certain circumstances and in follow-up studies after roentgenographic studies have been made primarily. They must be made with care, however, and with the eyes fully accommodated. Fluoroscopic observations alone have no value whatever as medico-legal records, nor are they likely to give much information in certain industries, especially when the silica intake is unusually rapid, as in high percentage silica dusts. When an exact knowledge of the lung condition, and as to whether any exists, is highly desirable, the technic of examination must be of the most exacting precision possible, and at least equal to that employed in the detection of early tuberculosis. Unfortunately, it is carried out in this manner with comparative infrequency. Stereoscopic roentgenograms, made in the postero-anterior direction if possible, are of the utmost importance, especially when unusual questions are to be settled. Aside from their general diagnostic superiority over flat films, the latter are apt grossly to exaggerate minor apparent abnormalities which may become actually insignificant when viewed stereoscopically. In advanced silicosis the lungs are practically always emphysematous, and the thicker lower portions contain much more than the normal amount of air and sometimes comparatively less interstitial pulmonary tissue, hence these portions are likely to be over-

ing pneumoconiosis are, first, through pathology,—a natural process of investigation in the dead or an experimental process in living animals, and, secondly, by roentgenological examinations on living subjects. A correlation of investigative observations along these lines together with a study of the physical and chemical factors involved in the inhalation and subsequent disposition of the dust has been the means of a thorough understanding of the condition of pneumoconiosis. By the physical and chemical factors is meant the physical quality of the dust, its chemical composition, determined especially by petrographic estimations in the case of silica, the dust count (by light or dark field whichever may be recognized as standard in the future) at the individual locality during general working conditions, the time at work and duration of occupation, the protective devices employed, and their approximate protective value. To these should always be added an investigation into previous occupations and dust exposure.

It has been most fortunate for general information of all of us interested in the subject that ample opportunities have been afforded to various investigators for autopsies on individuals presenting all the various degrees of progress of silicosis in many different industries. In the autopsy check-up of roentgen appearances in early and all stages of pulmonary tuberculosis it has been necessary, with very few exceptions, to await death from the terminal stages of the disease in the human subject, and few opportunities have been afforded for comparison of early roentgen appearances with the pathological process as it existed at that particular period. Since the workers in many dusty industries have been exposed frequently to other hazards such as industrial accidents which have resulted in death, it has been possible to compare the roentgenological appearance with the pathological picture at the time in practically all stages of the condition.

It has been our endeavor to learn the interpretation of roentgenographic and fluoroscopic appearances as nearly as possible on the basis of pathological studies and we believe that anyone qualified to interpret roentgenological findings must be familiar with the appearances of the healthy chest and lungs, with those produced by pneumoconiosis in all stages of progress in all dusty occupations, with the pathology of the disease under all of these conditions, and with the physical factors involved in its production. Otherwise one is not qualified to render accurate interpretations, especially when they are of vital or medico-legal importance. A knowledge of pathology is just as important as differential diagnosis; moreover, anyone familiar with the roentgenographic appearances of the various stages of the condition characteristic of but one industry, such as mining, pottery, or granite cutting, is not necessarily qualified by that experience to make accurate interpretations in connection with the disease acquired in certain other industries such as sand pulverizing or asbestos working. Appearances which might be interpreted as of comparatively trivial importance in workers in one industry may be of very great importance in another. Such knowledge is of the utmost importance in both diagnosis and prognosis, particularly

exposed especially as to penetration, if the routine technic adapted to the normal individual of the same physical development is employed. It is often advisable, therefore, to make a set of roentgenograms by the usual technic for the build of the individual which will show the upper chest adequately but which may blot out much of the pathological change in the lower portion by over-penetration, and then to supplement these roentgenograms by another set of lesser penetration to show to better advantage changes in the lower lung fields. (Figs. 1 and 2). Often in advanced cases there are dense fibrotic consolidations, in the mid or subapical lung fields which may be tuberculous in addition to being silicotic. Another single roentgenogram or stereoscopic pair of very much over-penetrated exposures, and especially if made with the use of the Potter-Bucky diaphragm, may throw additional light on these areas. Sometimes we have in this way shown cavities in them which could not ordinarily be found. Lateral views are often very important in the location of lesions, in showing the posterior costophrenic spaces, in the determination of the exact degree of emphysema, in the estimation of cardiac size, and the condition of the heart and aorta, and sometimes in differential diagnosis. It is a well recognized fact that the sagittal view does not give the correct information as to heart size and configuration of its shadow in the advanced silicotic individual, and the lateral view is imperative for this determination. The marked emphysema of the advanced silicotic is likely to cause rotation of the heart and to diminish the width of the cardiac shadow in the sagittal view. The localization of large, dense areas of consolidation by the lateral view is often very important for differential diagnosis. If these areas are found to be in the apex of the lower lobe they are not apt to be tuberculous, as lower lobe apical tuberculous lesions are not likely to take on this appearance.

Supplementary fluoroscopic observations should be made of every case examined. The special advantage, in addition to those of every chest study, is the determination of diaphragmatic excursion and costal expansion, which is a most important factor in estimating incapacity. In the cases of rapidly progressive interstitial fibrosis, this observation may be the deciding factor in diagnosis or in the interpretation of roentgenographic appearances. Moreover, a preliminary fluoroscopic observation is likely to be the means of determining just what additional roentgenograms may be required in any given case.

Much has been written upon the roentgenological aspect of pneumoconiosis, and we are loathe to add to that which we have already presented (3, 4, 5, 6) except to emphasize what appear to us to be very important points in the present-day chaotic state into which the subject of this condition has fallen, or, perhaps, it would be better to say arisen. The most important of these would seem to be the necessity for exact correlation of roentgenographic findings with pathological processes. It becomes necessary, therefore, to review briefly the known pathological changes in order to show how we have attempted to make the roentgenographic appearances conform with them. Most dusts which

FIG. 1. Normal general roentgenographic exposure of the chest of a male, aged 39 years, who worked for a little over a year in a sand pulverizing plant. The only abnormal appearance is a slight general haze in each lower lung field. This is due to the beginning of an interstitial predominance of silicosis, which, in view of the short duration of occupation, gives an unfavorable prognosis. The condition was further indicated by the marked restriction of both domes of the diaphragm. There was no nodular appearance.

FIG. 2. Another roentgenogram of the same case as illustrated in Figure 1, with less penetration, showing the lower lung fields to better advantage. This exposure in itself should not be used for diagnostic purposes, but only with the normal one for the size of the individual.

produce pulmonary fibrosis must do so by gaining entrance into the lymphatic system of the lungs. This means that the particles must reach the ultimate portions of the air passage beyond the ciliated cells and where the alveoli are in open relation, because it is only there that they can reach the lymphatic system. They must be taken into the lymph channels and spaces by phagocytic cells. This means that the dust particles must be sufficiently small to be phagocyted, and it is known that they must be 10 microns or smaller in size for this to occur. Most of the particles phagocyted and capable of doing definite damage are apparently from 2 to 5 microns in size, but those down to 0.5 micron are known to be taken up. It is uncertain as yet just what is the relative effect of these smallest particles as compared to that of the preponderance of the ones of 2 to 5 microns. Particles down to 2 microns may be detected by light field illumination, whereas those between 0.5 and 2 microns require the dark field method. It is unnecessary to enter again into any discussion as to the identity of the dust cell or the lining cells of the alveoli.

The dust cells apparently lodge in the outposts of lymphoid deposits. Some may reach only the most distal ones, others may go further; at any rate, they evidently initiate the change in chemical composition of the contained silica particles to a hydrated colloid, which is a destructive agent to cells. This change is supposedly brought about through the agency of the alkaline reaction of cells and body fluids. The dust-laden cells die as a result, and discharge their contents, which are destructive to the surrounding cells in the lymphoid deposits. Other dust cells or phagocytes no doubt carry some of this discharged silica to some of the more central lymphoid deposits, until eventually the pulmonary lymph nodes are reached. Wherever the altered silica is deposited there occurs a cellular reaction, with both infiltration and proliferation, and eventual local necrosis, usually microscopic in extent, followed by healing by fibrous tissue. So far as we have described this involves the lymphoid deposits and pulmonary lymph nodes in a progressive process. So far, then, we have definite pathological effects,—these structures become enlarged and are the seats of subsequent fibrosis and both processes cause a lymph block in the perivascular and peribronchial lymph vessels. There are also proliferative thickenings of the lymphatic walls which add to the physical block or stasis. Now let us see how the process thus far developed may alter the normal roentgenographic appearances. The enlarged and more or less fibrosed pulmonary lymph nodes will appear as enlargement and increased density of the hilum shadows, while the lymph stasis and thickening of the vessels is likely to intensify the trunk shadows and linear markings. In addition, the general enlargement of lymphoid deposits may cause a slightly increased density or faint haze of the portions of the lung fields most affected,—the mid fields and first on the right side. (See figs. 3 and 4.) This appearance may be due to another cause described later. At any rate, the slight enlargement of the lymphoid deposits is a *microscopic* forerunner of later *macroscopic* nodular appearances, also discussed later.

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FIG. 4. Roentgenogram of the chest of naked rambler, aged 46 years, 15 years cumpation. The disease, homogeneous hilum shadows and increased prominence of the trunk shadows indicate the perivascular-peribronchial lymph node type of silicosis. The increased density of the right lower mid lung field indicates an interstitial type of fibrosis, while is further suggested by restriction and slight peaking of the right dome of the diaphragm. In the roentgenogram where is a fine nodular mottling in the mid lung fields indicative of an early nodular type of the condition. This is too fine to be reproduced in a reduction. Three types of silicosis are manifest in this case

FIG. 5. Roentgenogram of the chest of an anthracite coal miner aged 36 years, who spent 11 years in the mine doing various kinds of work, including the use of the jackhammer (drill). No symptoms. Chest examined because of occupation during a routine study, and can be regarded as negative except for the calcified pleurisy in the right lung. One might expect some slight evidences of pneumoconiosis in this case because of his work as a driller.

It is erroneous to refer to all of these changes in appearance as the result of fibrosis alone.

Now this appearance of prominent hilum shadows and increased prominence of trunk shadows and linear markings, with or without the faint haze, has in this country at least been designated as the *first stage* of silicosis or pneumoconiosis. There may be some excuse for continuing to call this the first stage, but continued experience with cases of pneumoconiosis developing in various industries has led us to question the wisdom of designating any appearance of pneumoconiosis by any term denoting numerical stages of progress. In this particular instance under discussion the individual may develop the appearance in a comparatively short period of from 1 to 5 years, or not pass beyond it, on the other hand, in fifty years. Moreover, it is not apt to be distinguishable as a stage of the past in more progressive periods of the condition, and in some industries it may be insignificant or indistinguishable as a stage at all. We prefer to designate the appearance by a term which implies its pathological nature and to call it not a stage but the *perivascular-peribronchial-lymph node* type or preponderance of the condition.

The appearance of this type or preponderance or stage is by no means characteristic of pneumoconiosis but is found as a result of many other conditions, such as acute bronchitis, chronic bronchial catarrh, bronchiectasis, passive congestion from cardiac decompensation, the infiltrating type of malignancy, and polycythemia. It should not be accepted as an evidence of pneumoconiosis, especially in medico-legal cases, unless all of these conditions are positively ruled out and the appearance has become present or more marked over a period during which serial examinations have been made. Moreover, even if due to pneumoconiosis, the appearance does not represent an incapacitating degree of the condition in any way, or most certainly not unless it has developed rather rapidly after starting in a dusty occupation capable of producing it. With very rapid development of pneumoconiosis one is more apt to find evidences of the interstitial type of fibrosis. We may seem to be unnecessarily verbose in connection with such a minor aspect of the condition, but our experiences with so many cases of what have seemed unwarranted claims for total disability based largely on no more roentgenological evidences than those just mentioned, have forced us into this detailed expression of facts.

In cases with silicosis which is moderately slow in its progression, the microscopic nodular lymphoid deposit proliferations tend to become conglomerate and to produce a macroscopic nodular process which appears on the roentgenograms as the typical rounded shadows from 1 to 5 millimeters in diameter, or even larger, and which are so characteristic of the condition in many industries. As this appearance was found as a more advanced stage of silicosis in the earlier cases examined, and especially among miners in this country, it was and is still called the *second stage* of the condition. (See Fig. 5.) It is conspicuous by its absence or insignificance, however, in many industries, notably the granite cutter, sand blaster, sand stone, and asbestos workers. (See Fig. 6.) It is especially likely to be absent or incon-

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FIG. 5. Roentgenogram of the chest of a male aged 55 years, who had worked as an anthracite-coal miner for 40 years. His chief complaint was shortness of breath and subacute attacks for 1 years. The case is used as an illustration of the nodular type or predominance of pneumonoconiosis because the nodules are sufficiently large to allow in a reduction. As a matter of fact the case represents the earlier stages of a diffuse fibrosis indicated by the nodules tending to become conglomerate and the beginning of consolidated areas in the subapical regions. The appearances of these areas suggest that the origin of the consolidations is to be that of an interstitial type of fibrosis, which is interesting. The fact that they are not at the periphery indicates that they are probably not tuberculous in origin.

FIG. 6. Roentgenogram of the chest of a female aged 37 years, a sandan sbestos worker for 6½ years. The appearance indicates a fairly well advanced stage of asbestosis. Note the increased prominence of hilum and trunk shadow and the linear and interstitial fibrosis of the lower lung fields. The costophrenic angles and left cardiac border are characteristically indistinct. Note the absence of nodular appearances. Reproduction furnished through the courtesy of Dr. J. V. Sparks, London.

spicuous in those who are developing silicosis rapidly, such as in those working with pulverized sand without adequate protection. (See Figs. 1 and 2.)

If this appearance is absent or nearly so as a stage of progress in so many industrial silicoses, why designate it as a numerical stage of progress at all? We have ceased to do so, and have called this appearance the *nodular type* or preponderance of silicotic fibrosis, or, more correctly, silicosis.

A coincident tuberculous process of the typical adult type usually can be distinguished readily in silicotic individuals with this preponderance or type, provided the nodules are not large or too widespread beyond the central lung fields. When the nodules are large and more widely disseminated the diagnosis of superimposed tuberculosis becomes far more difficult and may be impossible. Gardner (7) has called our attention to the fact that such nodules may become the seat of tuberculous infection in some instances and with some dusts, such as granite. We have seen cases in which the nodular type of silicosis has been closely simulated by an ordinary or even hemorrhagic bronchopneumonic spread from a primary apical tuberculosis to the mid lung fields, either ipsolateral or contralateral or both. The appearance of nodular silicosis is often closely simulated by miliary tuberculosis and we have experienced difficulty in differentiating it from actinomycosis, sporotrichosis, and leptothric invasion of the lungs.

This type of silicosis may not be definitely incapacitating unless it has developed rather rapidly. It may not be found except by chance in routine examinations until after 20, 30, or more years of occupation.

There is another type of silicotic appearance which was very puzzling to us at first because of its exact identity and our inability to find the proper place for it in a numerical classification of progressive stages. Our attention was first called to it by Drs. Riddell and Brink (8), and later by Dr. Smith (9). Further investigations and experiences with it in connection with some sand pulverizers seemed to place it as a sort of missing link in many of the cases in which there was an omission of the old second stage or nodular type of silicosis. Fortunately a definite pathological place seems to have been found for the condition and a roentgenological classification for the appearance.

One can imagine the hilumward progress of dust and its effects being rather rapidly checked by an unusually profuse intake of silica, resulting in a quick or abrupt blockage of lymph channels. Then only the pleuralward pathways would be open for the lymphatic travel of dust cells. Soon these could be closed in much the same manner as the hilum ones. Gardner (10) then came to the rescue of our theory by stating that under such circumstances dust cells could and did penetrate the lymph channel walls direct into the interalveolar connective tissue to set up changes productive of an interstitial cell proliferation and subsequent fibrosis. Gardner further suggested that the previous condition of the pulmonary lymph nodes induced by childhood tuberculosis and other agencies might have some influence on the rapidity of onset and progression of silicosis. We have had reason to sus-

pect this possibility in some cases examined and showing marked bilateral calcification of these nodes as a result of childhood tuberculosis.

The interstitial cell proliferation and fibrosis so produced presents a typical roentgenographic appearance and to which we have just alluded. (Figs. 1, 2, 4, 6.) It is a fine and rather homogeneous haze usually first seen in the right lung in the middle third and just below, and later in the left lung. It tends to spread rather evenly into larger areas. It may or may not have the perivascular-peribronchial-lymph node type of the condition associated with it, depending upon the rapidity of progress of the process. (Fig. 4.) It is likely to appear in very rapidly progressing cases, but is also found though less frequently in those of slower progress. We have designated this as the *interstitial type or predominance*, and have subdivided it into rapid and slow, which subclassification must depend largely upon the physical factors and time of exposure. The detection of this type requires roentgenograms of the best quality and a stereoscopic set in order to identify the appearance as that of an intrapulmonary condition. Fluoroscopic observations are necessary to determine whether or not the underlying cause of the hazy appearance is responsible for restricted diaphragmatic excursion.

This type of fibrosis will almost invariably progress to the terminal stage with little or no appreciable nodular appearance. (Fig. 6.) Care must be exercised in differentiating it from a chronic interstitial lung change resulting from pneumonia or continued upper respiratory infection as in the tuberculoses. A tuberculous spread from another lesion must also be ruled out. If the appearance is limited to an upper lobe it most certainly is not due to interstitial silicotic fibrosis but is quite likely to be due to a rapidly progressive tuberculous process. We have had occasion to make this differentiation. Thickened pleura may closely simulate the appearance in the flat roentgenogram.

The condition responsible for this appearance in advanced form and of rapid development has been termed "acute silicosis" by Chapman (11) and some others. We have seen several cases similar to those to which Chapman alludes. These individuals worked in sand pulverizing plants and most of them eventually died of pneumonia or tuberculosis. It seems to us questionable whether such a term as "acute silicosis" is advisable. Silicosis is a condition which is extremely variable in its progress and yet the actual onset of fibrosis to some extent is probably largely simultaneous no matter how rapid the ultimate progress. This unusually rapid fibrosis differs from the better known forms of the disease only because of variations in the physical factors involved and the greater and earlier predisposition to pneumonia, tuberculosis, and other respiratory infections. It has seemed more logical to speak of a rapidly developing than an acute silicosis. Moreover, one cannot be certain in these cases that the acute factor is not largely a rapid tuberculous process or a pneumonia.

There are few if any arguments to be recorded in connection with the terminal stages of silicotic fibrosis. Instead of the *third stage*, we have preferred to call this aspect of the disease the *terminal diffuse fibrosis*. It is more

or less incapacitating in practically all instances, and all too often completely so. From the roentgenological standpoint we may regard the terminal appearances as having been reached in three ways or as presenting three strikingly characteristic appearances. A nodular type may become more and more conglomerate, with more or less interstitial fibrosis. An interstitial type may progress to a terminal condition and present appearances somewhat similar to and sometimes almost or quite indistinguishable from those of chronic diffuse fibroid tuberculosis or the terminal fibrotic stage of a Friedlander pneumonia in a patient who has survived. In the third form large consolidations are found in the subapical regions. These may be symmetrical as to size and location, or much larger on one side. Many of them no doubt possess a certain tuberculous element. It has been our experience that tuberculosis may be strongly suspected when these consolidated areas are peripherally or subpleurally located and especially if there is a homolateral deviation of the trachea. Cavity, of course, implies tuberculosis in almost every instance, whether the sputum is positive or not. Sometimes the presence of cavities may be determined only by over penetration of these areas. Definite diagnoses of coincident tuberculosis or its absence as a rule are otherwise extremely difficult. A tuberculous consolidated area may exist anywhere in such lungs, even where its identity is least suspected.

To summarize in respect to the classification of silicotic appearances based upon known pathological changes we have suggested the following in place of the old numerical stages:

1. Peribronchial-perivascular-lymph node predominance or type. This may be rapid or slow, usually the latter.
2. Early interstitial predominance. This may be extremely or moderately rapid, depending on the silica intake. It may or may not have an associated slight nodular appearance.
3. Advanced interstitial predominance.
4. Nodular predominance. Rapidly or slowly progressing.
5. Advanced diffuse or terminal fibrosis
   - Conglomerate-nodular type.
   - Interstitial type.
   - Massive fibrotic type.

This classification does not differ materially from that previously suggested (6), and we have used it to good advantage and without finding any disadvantages in it for over two years. It has been found applicable to both clinical and roentgenological studies for each individual industry or occupation investigated, with the possible exception of pulmonary asbestosis, about which there is a great deal yet to be learned. We believe that it can usually be applied to this condition, however (Fig. 6.)

Indeed, the subject of correlation of roentgenological appearances and pathological changes cannot be closed without brief reference to the condition of pulmonary asbestosis. Roentgenologists and others have found that appearances differ quite materially from those of practically all phases of true silicosis, which is easy to understand when we realize the differences in physical as well as chemical characteristics of the inspired dust. The

roentgenological appearances have been somewhat variously described by different authors (12, 13, 14, 15), and perhaps easiest for roentgenologists to understand by Sparks (16), and according to our own observations as well, they may be briefly summarized on the basis of the probable pathology of asbestosis somewhat as follows:

There is an appearance suggesting increased prominence of hilum and trunk shadows, especially in early stages, but this is overshadowed later on by other features. It is somewhat different from the perivascular-peribronchial-lymph node predominance of silicosis. Sooner or later, there appears a rather homogeneous density in the lower lung fields, evidently due to an interstitial type of cell proliferation and fibrosis. Later on there is a further increase in this density in a wider field, with a fine network of fibrosis, which may be a transition of interstitial into a mild terminal fibrosis. There is very little nodular predominance and it is always inconspicuous. With this appearance diaphragmatic excursion is limited and the outline may be uneven. Then there are some appearances which differ very materially from those of silicosis. The diaphragmatic outline is indistinct, the costophrenic angles are clouded, there is the absence of the typical emphysematous appearance of advanced silicosis, with the small shadow of the rotated heart, and the left cardiac border is often very indistinct. Now the first two of these last appearances cannot be laid to thickened pleura alone, because this is associated with silicosis also. The indistinct left cardiac border cannot be due solely to lung changes, because there is as much or more pulmonary change in silicosis. It is a theory of ours that the absence of marked evidences of emphysema and all of these other differences might be explained on the basis of a lack of aeration due to the very peculiar characteristic pathological features of asbestosis to be explained later. Certainly there is much to learn about this condition, and especially by serial study.

Space will not permit our entering into all of the details of the subject of pulmonary asbestosis. These are many of the essential pathological changes found in silicosis, such as those suggested and consisting of a moderate cell proliferation and fibrosis. The predominant pathological feature is the presence of the asbestosis bodies which numerous authors have described and to whom we have referred in a previous presentation on the subject (6). These bodies are of various sizes and are found in various stages of development in many localities, but particularly imbedded in fibrous tissue, most of them in large clumps in the alveoli and very large ones in the terminal air passages. Whether any fibres are sufficiently small to be phagocyted is uncertain. Certainly they do not reach the pulmonary lymph nodes (16). The bodies increase in size from the original fibres probably by colloidal aggregates of blood protein and containing an iron salt (17). Possibly, as Sparks (16) suggests, this reaction to the inhaled asbestos fibre is, after all, a safeguard against even more serious pulmonary changes. Gardner (18) has admirably described the relations of the largest of these bodies to the terminal air passages in which they are found. He states that they are apt to be surrounded by a fibrous collar in

the air passage wall, and it has seemed to us as quite likely that this collar surrounding what is really a foreign body of increasing size, together with the presence of large clumps of the smaller bodies in the alveoli, must be conducive to a condition of multiple minute areas of atelectasis or drowned lung. Such a state of affairs might well explain the lack of roentgenological evidences of emphysema and the other peculiarities of asbestosis as compared with silicosis, and also the intense dyspnoea and cyanosis of the late clinical stages of pulmonary asbestosis.

This is a comparatively new aspect of pneumoconiosis. Intensive studies of the condition have been made in Great Britain, especially by Merewether (19), and Merewether and Price (20), and in South Africa by Simson (21). Comparatively little has been written on the subject in this country except by Lynch and Smith, (22), and Gardner. The conditions in the industry in this country should be judged with a fair degree of accuracy by comparison with those in Great Britain. The report by Merewether and Price states that approximately 2200 individuals were exposed to practically pure asbestos dust in factories in England. Now Great Britain and the United States both use the Canadian asbestos product almost exclusively and conditions in the two countries are probably very similar. Merewether (23), in a personal communication, has stated that there were 319 recorded deaths from silicosis in Great Britain in 1931 and 9 from asbestosis or asbestosis with tuberculosis. Full particulars were available to date of 35 deaths from asbestosis or asbestosis with tuberculosis. In 11 cases tuberculosis was either a complicating or terminal factor and in 24 no tuberculosis was present. "The average age at death in the cases of asbestosis with tuberculosis was 45.7 years, and the average length of employment in asbestos, 13.5 years; in the cases without tuberculosis, the average age at death was 40.6 years, and the average length of employment in asbestos was 15.1 years." Their asbestos risk has been controlled since March 1, 1932 by a very stringent Code of Regulations (24, 25, 26) applying to the industry.

We must all realize that pneumoconiosis, while a dangerous and incapacitating condition under certain circumstances, is a more or less necessary risk of commercial development in the progress of civilization. We cannot get along without coal, and yet coal mining entails certain hazards, one of which is pneumoconiosis. Some miners will work their entire lifetime at their mining occupations and not develop any incapacitating form of the condition; a few others may not be so fortunate. We cannot adjust our mode of living to be deprived of gold, iron, and other metals, of pottery, asbestos products, the foundry, abrasive work, and innumerable other industries and their products. We must all admit that certain hazards must be accepted by the worker after he has been made fully acquainted with them, provided they are known by those individuals who are in a position to give the information. This is one point of view of the subject.

Another side to the question is that it is incumbent upon the employer to protect his employees to the best of

his ability by all known and proven safety precautionary measures.

Thirdly, an extremely important aspect of the situation, and one to which too little attention has been paid in the past, is a means of acquainting employers of possible dangers to their workers and of the best precautionary measures to be used in order to minimize the hazards or entirely remove them. Industrial medicine has known the hazards and has developed the preventive measures, but employers are not students of industrial medicine. State Health Boards have been active in spreading propaganda in connection with preventive medicine and communicable diseases and in enforcing certain regulations for the control of the latter, but how much attention have they given in past years to regulation of the dust hazards of industry?

Morally, the worker disabled as a result of the dust hazard would seem to be entitled to compensation just as much as though his incapacity were the result of bodily injury. His realization of this fact has suddenly developed a medico-legal aspect of pneumoconiosis in this country which is at the present time more or less in a state of chaos. How simple it would be if there had been enacted in every State uniform and adequate compensation laws and regulations governing the adoption of safety measures to eradicate or greatly modify the essential dangers of dusty industries, such as has been done in Great Britain, Ontario, Australia, New Zealand, the South African Union, and Germany. Only a few of our States enforce any preventive regulations or have any compensation laws which make silicosis a compensable industrial disease.

One of the great misfortunes to the medical profession of the legal situation in this country is the fact that decisions by Compensation Boards and in Civil Courts must be based to a considerable extent upon conflicting medical testimony. As the roentgenological examination is of so much importance in establishing the status of the claimant as to disability from pneumoconiosis, the roentgenologist plays an important part in the conflicting expert testimony. It seems to be a difficult matter for Boards or juries to decide upon the relative merits of opposing medical testimony, whereas it would seem that the basis of these merits should be the qualifications of the opposing witnesses. We have observed instances in which this might be a very difficult decision even for medical men, because medical opinions must often differ. On the other hand, there have been very many more instances in which it would seem that it should have been a very easy matter to have decided upon the relative merits of the character of testimony and of qualifications. We have presented the foregoing facts with a view of attempting to prescribe what the qualifications and knowledge of a roentgenological expert should be in connection with silicosis.

The tardiness which we Americans have shown in the adoption of adequate measures to adjust all difficulties in connection with dust hazards probably is best explained by the wide scattering of the industries in which dangerous dusts are evolved over such a large country as ours. In England and on the Rand, for example, where hazardous dusty industries are far more concentrated than here, attention was

centred upon silicosis at an earlier period and received much more serious attention. In our own country pneumoconiosis, and especially its hazardous aspect, receives very scant attention in the curriculum in most of the medical schools, and particularly in those in which there is no course in industrial medicine. Then, too, our form of government is not conducive to an easy control of the situation. The Federal Public Health Service has performed yeoman service along investigative lines in most aspects of silicosis, but the Federal Government does not make regulations and prescribe compensation laws. These duties must be left to each of the States individually.

There can be but one satisfactory outcome to the situation. Compensation laws must be passed by each State as nearly uniform in essential details as possible in order to avoid any effects leading to unjust competition. Medical Boards must be created having, among their many other duties, full authority to decide upon the merits of all claims for disability from pneumoconiosis, without recourse to conflicting medical testimony. This would require that all members of such Boards should be thoroughly familiar with all aspects of the condition. The physical problem such as dust counts and analyses of dusts would have to be taken care of by experts in these lines. The Board could have the advantage of investigative work by such bodies as the United States Public Health Service. Experimental work would probably have to be carried out as it is at present by well recognized private individuals or institutions. This seems like a Utopian scheme for this country, but it is only because we are a nation of individual states, each one of which must adopt procedures which in other countries are carried out with satisfaction by central governments.

Such Medical Boards, in order to be fully efficient, should have authority and be able to pass reliable judgment upon the health and the condition of the lungs of all applicants for positions under new employers and to provide means for the periodic examinations of all employees in hazardous dusty occupations. In the first instance, they should give due consideration to all possible effects from previous occupations, bearing in mind the progressive nature of silicosis of comparatively rapid development, even after years of cessation from a hazardous dusty environment. The last employer is not always responsible for the development of an incapacitating silicosis, as is well demonstrated in the case illustrated in Figures 7, 8, and 9. The reader is first referred to the illustration legends, after reading which the following comment is in order:

This patient first came to us in 1924, with a history of previous pneumonia and left pleural effusion (Fig. 7). At that time his occupations, past and present, were unknown. A diagnosis of pulmonary tuberculosis was entirely justifiable. He was next seen and examined in 1929 because of gradually progressing dyspnoea. At that time the roentgenographic appearances at once demanded an insight into occupations. It was found that he had been a machinist for 15 years, but before that had been a miner for 18 months and a firebrick worker for 5 to 6 years. The inferences to be drawn from the two last examinations in 1929

FIG. 7. Male, aged 35 years, occupation not known at the time of this first examination, August 2, 1924. The right lung shows two areas of an exudative tuberculous process in the upper lobe, some intensification of trunk shadows below, and a diffuse fine mottling in the mid lung field appearing in this reduction as a faint haze. The left lung shows a tuberculous lesion with cavity in the upper part of the lower lobe and a mottling in the lower field shown in this reduction as a marked haze. There is thickened pleura at the left base. The diagnosis then was tuberculosis, but silicosis should have been added, as it was later.

FIG. 8. The same case as illustrated in Figure 7, November 8, 1929. Terminal diffuse silicotic fibrosis with large consolidated areas in each lung and conglomerate nodules at the right base. No doubt tuberculosis is an important and active factor in the appearance. Spontaneous pneumothorax at the left base.

were that the man had tuberculosis in 1924, with some evidences of silicosis, and that in the interval of the next 5 years he became the victim of a rapidly progressing silicosis despite his then practically safe occupation. No doubt the appearances in 1929 were the result of both silicosis and tuberculosis.

This case represents an excellent example of the necessity for the greatest care in obtaining histories of previous occupations and for periodic examinations during occupation. It also shows how great injustice might be inflicted upon an employer by inexperienced deductions and interpretations.

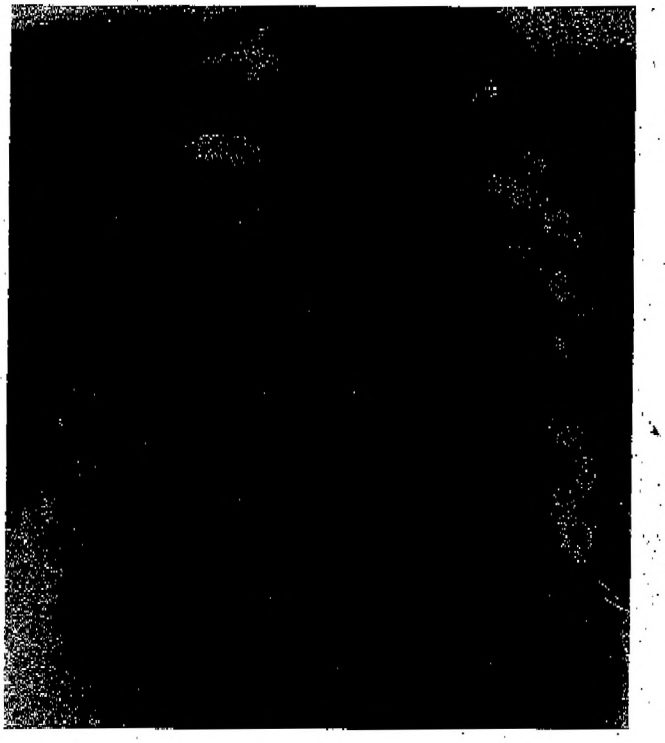

FIG. 9. Third examination of the case illustrated in Figures 7 and 8, made November 23, 1929, showing the same appearances as at the previous study except that the pneumothorax has almost disappeared. Pneumothoraces in silicosis are almost always higher up.

BIBLIOGRAPHY

1. PANCOAST, H. K., MILLER, T. G., AND LANDIS, H. R. M.: A Roentgenological Study of the Effects of Dust Inhalation on the Lungs. Trans. Assoc. Amer. Physicians, 1917, *and* Am. Jour. Roentgenol., 1918, *5*, 129.
2. WATT, A. H., IRVINE, L. G., JOHNSON, J. P., AND STEWART, W.: Silicosis ("Miners' Phthisis") on the Witwatersrand. Appendix no. 6, Miners' Phthisis Prevention Committee of South Africa, Pretoria, 1916.
3. PANCOAST, H. K.: Roentgenologic Studies of Pneumoconiosis and Other Fibrosing Conditions of the Lungs. Ann. Clin. Med., 1923, *2*, 8.

4. Pancoast, H. K., and Pendergrass, E. P.: A Review of our Present Knowledge of Pneumoconiosis based upon Roentgenological Studies, with Notes on the Pathology of the Condition. Am. Jour. Roentgenol., 1925, *14*, 381.
5. Pancoast, H. K., and Pendergrass, E. P.: Pneumoconiosis (Silicosis). A Roentgenological Study with Notes on Pathology. New York: P. B. Hoeber, 1926.
6. Pancoast, H. K., and Pendergrass, E. P.: Review of Pneumoconiosis: Further Roentgenological and Pathological Studies. Am. Jour. Roentgenol., 1931, *26*, 556.
7. Gardner, L. U.: Personal Communication.
8. Riddell, A. R., and Brink, G. C.: Personal Communication.
9. Smith, A. R.: Personal Communication.
10. Gardner, L. U.: Appendix on Pathological Studies. U. S. Pub. Health Bul. no. 187, 1929.
11. Chapman, E. M.: Acute Silicosis. Jour. Am. Med. Assoc., 1932, *98*, 1439.
12. Wood, W. B.: Pulmonary Asbestosis: Radiographic Appearances in the Skiagrams of Workers in Asbestos. Tubercle, 1929, *10*, 353.
13. Soper, W. B.: Pulmonary Asbestosis: A Report of a Case and a Review. Am. Rev. Tubercu., 1930, *22*, 571.
14. Oliver, T.: Pulmonary Asbestosis: A Socio-Medical Study. Arch. f. Gewerbepath. u. Gewerbehyg., 1930, *1*, 67.
15. Wood, W. B., and Gloyne, S. R.: Pulmonary Asbestosis. Lancet, 1930, *1*, 445.
16. Sparks, J. V.: Pulmonary Asbestosis. Radiology, 1931, *17*, 1249.
17. Cooke, W. E.: Asbestos Dust and the Curious Bodies found in Pulmonary Asbestosis. Brit. Med. Jour., 1929, *2*, 578.
18. Gardner, L. U.: Address before the Section on Industrial Medicine, College of Physicians of Philadelphia, December 9, 1932.
19. Merewether, E. R. A.: The Occurrence of Pulmonary Fibrosis and other Pulmonary Affections in Asbestos Workers. This Jour., 1930, *12*, 198, 239.
20. Merewether, E. R. A., and Price, C. W.: Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. London, H. M. Stationery Office, 1930.
21. Simson, F. W.: Pulmonary Asbestosis in South Africa. Brit. Med. Jour., 1928, *1*, 885.
22. Lynch, K. M., and Smith, W. A.: Asbestos Bodies in Sputum and Lung. Jour. Am. Med. Assoc., 1930, *95*, 659.
23. Merewether, E. R. A.: Personal Communication: An Extract from the Annual Report of the Chief Inspector of Factories and Workshops for the Year 1931.
24. Statutory Rules and Orders, 1931, no. 1140. Factory and Workshop. The Asbestos Industry Regulations, 1931. London, H. M. Stationery Office, 1932.
25. Statutory Rules and Orders, no. 341, Master and Servant. The Silicosis and Asbestosis (Medical Arrangement) Scheme, 1931. London, H. M. Stationery Office, 1931.
26. Statutory Rules and Orders, 1931, no. 344. Master and Servant. The Asbestos Industry (Asbestos) Scheme, 1931. London, H. M. Stationery Office, 1931.