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Pneumoconiosis

THE current use of a variety of classifications for the roentgenological appearances of silicotic lesions is making it difficult to correlate the results of a rapidly increasing number of observers. In South Africa, where silicosis is a product of a single industry, and where the problem is handled almost exclusively by a bureau of the government, the classification that has evolved is entirely adequate for their purposes; but in a highly industrialized country silicosis develops in a great variety of occupations. A diagnosis may be required of a physician in a plant, in a public health office, in a sanatorium, or in the physician's private office. Sometimes he may have had little experience in the interpretation of roentgenograms of the chest, and will be forced to lean heavily on the interpretation of a roentgenologist.

The legal aspects of the problem have placed grave responsibilities on the medical profession and have often been a source of embarrassment. Only the physician who has examined the subject has obtained an occupational history of an adequate exposure to silica dust, and has before him a suitable roentgenogram of the chest, should make the diagnosis of silicosis. The roentgenologist, not in possession of these facts, can merely state whether the shadows which he sees in a film are consistent with this diagnosis.

If objective terms descriptive of the type of pathological changes could be generally adopted, material progress would result. The clinician would not have to accept a diagnosis from the roentgenologist; general students of the disease would be able to correlate the findings of the various observers; and more accurate definition of roentgenograms would be available for medico-legal purposes. Roentgenograms of the chest, which are notoriously difficult to reproduce as illustrations, could be described in word pictures capable of interpretation in the light of the personal knowledge of the observer. An error in diagnosis need not necessarily be passed on to others not in possession of the original film.

With these obstacles in mind, a committee has been at work for the past 2 years preparing a tabulation of the various lesions of silicosis together with terms that attempt to depict the character of the shadows cast on an X-ray film by these lesions. This report is submitted to invite criticism, in the hope that the terms suggested, or others of a similar nature, will be generally adopted.

It should be distinctly understood that the tabulation which follows applies only to silicosis, that form of pneumoconiosis which results from the inhalation of dust with a high silica (SiO_2) content.* Other forms, like asbestosis, are excluded from this consideration because their pathology is essentially different from that of silicosis.

The tabulation contains two columns: On the left are the roentgenological appearances and on the right are the corresponding pathological lesions. There is further subdivision to

* Some of the non-siliceous components of certain industrial dusts seem to modify the pathological reactions, but the character of shadows cast by these modified lesions is not sufficiently defined at the present time to include them in the tabulation. Later, when more information has accumulated, certain other terms may have to be included.

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HEALTHY LUNGS AND ADNEXA

Roentgenological appearances	*Histological appearances*
1. *Healthy lungs*—As defined by the N.T.A. Committee report.	1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea.
2. Irregular exaggeration of the linear markings, with possibly some beading confined to the trunks.	2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi. Beading may be due to various causes, as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks.
3. Increased root shadow.	3. Cellular reaction in the tracheo-bronchial lymph nodes with extensions along afferent lymphatic trunks.

These changes come within normal variations when not accompanied by recognized organic disease.

SIMPLE SILICOSIS

4. *Nodulation*—Discrete shadows not exceeding 6 mm. in diameter, tending to uniformity in size, density, and bilateral distribution, with well defined borders surrounded by apparently normal lung shadows. The outer and lower lung fields characteristically show fewer nodules.	4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis.
5. Conglomerate shadows that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by— a. Localized increased transparency of the lung with loss of fine detail b. Intensification of the trunk shadows by contrast. c. Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm. d. Lateral view: Increase in the preaortic and retrocardiac space with exaggerated backward bowing of the spine. Widening of the spaces between the ribs may or may not be present.	5. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa.

describe the appearances of: (1) healthy lungs, (2) the uncomplicated silicotic lung, and (3) lung of silicosis with infection. The changes described under the first division are those compatible with a state of good health; and while they *may* be produced by the inhalation of relatively small amounts of silica dust, they are not sufficiently characteristic or advanced to substantiate a diagnosis of silicosis. Similar or identical appearances may also result from the inhalation of non-siliceous dusts, from certain infections, from cardio-vascular disease, and from certain other rare conditions. The changes involved are for the most part confined to the lymphatics and peri-lymphatic connective tissues and do not often affect the parenchyma of the lung. Since, by definition, silicosis is a disease characterized by nodular fibrosis in the parenchyma of the lung, these alterations, even when they may have been caused by inhaled silica, do not constitute a basis for a diagnosis

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SILICOSIS WITH INFECTION

The characteristic appearances described under simple silicosis are modified by infection as follows:

6. Localized discrete densities and/or string-like shadows accompanying those of simple silicosis described above.	6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of "healed" infection.
7. *Mottling*—Shadows varying in size with ill-defined borders and lacking uniformity in density and distribution, accompanying simple silicosis.	7. a. Areas of broncho-pneumonia with or without caseation, *i.e.*, acute infection. b. Lobular areas of proliferative reaction with or without caseation, *i.e.*, chronic infection.
8. *Soft nodulation*—The nodular shadows described under simple silicosis, 4, have now assumed fuzzy borders and/or irregularities in distribution. The change may or may not accompany the simple mottling of 7.	8. Perinodular cellular reaction either exudative or proliferative in character.
9. *Massive shadows* of homogeneous density not of pleural origin symmetrically or asymmetrically distributed.	9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or non-tuberculous origin superimposed upon a coexisting silicotic process. Outlines of normal structures may be partially destroyed.

of silicosis. The second group covers the discrete and conglomerate nodular fibrotic reactions of simple silicosis. The last group deals with silicosis complicated by infection. In the majority of instances the infecting organism is the tubercle bacillus, but the classification is sufficiently broad to include other types of infection. Certain criteria by which one attempts to differentiate various forms of infection will be discussed.

COMMENT

For the first group of appearances we have adopted the nomenclature of the National Tuberculosis Association Committee and described the lungs as *healthy* rather than as *normal*, as a perfectly normal adult human being is a great rarity. For a description of the roentgenological appearance of the healthy chest the reader should consult a paper by Pancoast, Baetjer, and Dunham in the *American Review of Tuberculosis* for 1927 (vol. 15, pp. 429–471).

As already mentioned under 2, *Irregular exaggeration of the linear markings with possibly some beading*, belongs in the healthy chest group even when found in persons with a history of considerable exposure to silica, for such changes are nonspecific in character and they do not involve the parenchyma of the lung. Silicosis as a clinical disease begins only when the lung proper is affected. Likewise, under 3, *Increased root shadow* may be of nonspecific origin and hence is not diagnostic. In the *early* stages of silicosis the mediastinal shadow may be widened, owing to the enlargement of the tracheo-bronchial lymph nodes from accumulated dust and cellular reaction to it; later, when specific fibrosis develops, the tissues generally contract and the nodes decrease in size. The changes described under 2 and 3 may be caused by many forms of irritation; if they are due to silica they are identifiable only by miscroscopic examination. They do not apparently interfere with respiratory function, and they are not of diagnostic significance.

The second group of changes is

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limited to *simple silicosis* uncomplicated by demonstrable signs of infection. This condition is characterized by the presence of small, discrete nodules of fibrous tissue disseminated throughout the functional parts of both lungs. The lesions and the shadows cast by them tend to be spherical, hard, sharply defined, and vary in size from 2 to 6 mm. While the distribution is usually uniform throughout both lungs, the extreme apices and the outer portion of the bases are frequently uninvolved. In less advanced cases the nodules remain discrete and separated by air-containing tissue. Irvine has aptly compared the shadows observed in the roentgenogram of the silicotic lung to those cast by a tree. In the earliest stages, when the reaction to silica is confined to the perilymphatic connective tissues, producing an accentuation of the linear markings, the shadow cast is that of a leafless tree. As small nodules begin to appear in the parenchyma of the lung, the tree begins to bud and the shadow of its branches is less clearly defined. When the tree is in full leaf, the stage of advanced nodulation, the shadow of the branches is completely obscured. Previous classifications have been concerned largely with the degree of nodulation as determined by the size and number of the nodules in the lung. We will not attempt to deal with this problem here, but choose to leave the "stage" of the silicosis to the internist, who has physical findings as well as a roentgenogram at his command.

Number 5 deals with the *conglomerate shadows* of simple silicosis, which appear to develop from a combination or consolidation of discrete nodules. The resultant lesion and the shadow that it casts are often difficult to distinguish from the *massive shadows* of silicosis with infection, 9. It is generally assumed that conglomeration results from accidental overlapping and fusion of discrete nodules when they become very numerous; but since conglomeration is usually a localized affair and does not occur in the same position of the lung of every individual, it is logical to inquire why the nodules happen to fuse in one portion of the lung and not in others. Microscopic examination of the tissues from such areas reveals no evidence of active infection. The nodules seem to be much closer together than in other portions of the lung, they are less uniform in size; and they are usually embedded in a matrix of diffuse fibrous tissue having the same characteristic hyaline appearance as that forming the nodules themselves. It seems probable that conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized inflammatory process occurring before or during the early period of dust exposure. Because the tissue was injured, more dust would tend to accumulate in the area, the nodules would develop irregularly and would frequently be very close together. The silica lodging in preëxisting granulation or scar tissue would exert its characteristic effect, and a diffuse hyalinization would result. This explanation for conglomerate reaction is at present hypothetical; proof will come from long continued serial roentgenographic studies of groups of persons exposed to silica dust and from the chance autopsy that may be obtainable. To differentiate *conglomerate shadows* from the *massive shadows* of infection, 9, reliance must be placed upon the absence of change in size and character of the shadows in serial films taken over an extended period, and upon the clinical findings in the case.

Emphysema is usually associated with far advanced silicosis and it is particularly liable to complicate conglomerate nodulation. It occurs in the

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immediate vicinity of the conglomeration as a result of the distortion produced by contracting scar tissue; there is also a generalized "compensatory" emphysema found along the borders of the lung, particularly at the bases. The latter type is also common in far advanced generalized nodulation.

In the last group, *silicosis with infection*, are included all cases with detectable evidence of infection whether active or inactive. In this respect we depart from the South African procedure, which includes here only active infection. The difficulty of determining activity, particularly in the silicotic subject, is our chief reason for this arrangement.

Number 6 covers foci of healed infection. Identification of such changes depends upon the same criteria that are generally employed in otherwise normal individuals. In the silicotic subject the shadows usually occur upon a background of generalized nodulation, although in some cases there may be a distinct tendency toward excessive nodulation in the immediate vicinity of the scars left by the infection. Where the exposure to dust has been limited, the major evidence of nodulation may occur about the foci of healed infection with much less reaction in the remainder of the lung. The string-like shadows of healed fibroid tuberculosis are not difficult to interpret if they occur in the classical location, *i.e.*, in the upper third of the lung. In the lower lung they present a problem whose solution depends largely upon the experience of the roentgenologist.

The term *mottling*, 7, we have reserved to describe the shadows of infectious lesions in contradistinction to *nodulation*, which is restricted to those of the silicotic dust nodule. It is essential that this distinction be appreciated and recorded in the terminology. In tuberculosis, mottling is due to bronchogenic or aspiration foci of disease which exhibit a characteristic clustered arrangement. The lesions may be exudative (acute) or productive (chronic) in type; the difference will be registered on the roentgenogram by a mottling which is fluffy and ill-defined, or hard and sharply defined as the case may be. The distribution of the mottled foci, together with the presence of large foci of older disease interpreted as tuberculosis, and clinical and laboratory findings establish the character of the infection. Mottling due to chronic infection that has developed previous to or simultaneously with the relatively early periods of dust exposure may exhibit little or no effect from the inhaled silica for many years. In non-tuberculous broncho-pneumonias the large chronic foci are absent, and the disseminate mottling may involve different parts of one lung or of both lungs. In many instances the nature of the infection must be established by serial examination over considerable periods of time and by careful correlation with clinical and bacteriological findings.

Soft nodulation, 8, is a term that has been coined to describe a rather uncommon combination of silicosis with infection, usually tuberculous. The ordinary hard, sharply defined nodular shadows of simple silicosis, under these circumstances, appear to have enlarged and lost definition. Their borders are now fuzzy and blend imperceptibly with the surrounding lung structure. Such lesions generally occur in association with localized conglomerate shadows in the apex or other portions of the lung. Histologically the infection appears to have localized in and about preëxisting silicotic nodules so that each is surrounded by a zone of exudative or productive cellular reaction.

Massive shadows of homogenous density, 9, are cast by the areas of combined silicosis and infection, usually

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chronic in nature. The two processes appear to have developed simultaneously, and unusual amounts of dust accumulate in the diseased areas. Generalized nodulation usually occurs throughout the remainder of the lungs. Pleural densities can be differentiated in stereoroentgenograms, and by overexposure it often becomes possible to penetrate the extremely dense intrapulmonary areas and analyze their internal structure. Not infrequently cavities may be visualized that were completely overlooked with the usual technic. When due to tuberculosis, such lesions are often bilaterally symmetrical. If the process extends to the pleural surface, a tuberculous etiology is postulated, while other infections are more often deep-seated.

Histological examination of such lesions shows conglomerations of simple nodules embedded in masses of more or less perfectly organized granulation tissue. Often the fibrous tissue has undergone the same peculiar hyalinization that characterizes the interior of the silicotic nodule. Usually the outlines of the original lung architecture are completely destroyed. Manifestations of infection depend upon the nature of the process—if tuberculous, there will be foci of caseation and possibly small cavities. Calcification is not infrequent. If the process is inactive, the presence of fibrous tubercles which do not exhibit the hyalinization of silicosis may be present. The occurrence of giant cells is helpful. A partially organized nontuberculous pneumonia usually contains foci of acute exudation of variable size. Clinically such unresolved pneumonias frequently exhibit periods of exacerbation followed by regression. They may be due to a great variety of organisms, including the Friedlander group and oral anaerobes. Where all manifestations of activity have disappeared, the lesion is probably best classified as a conglomerate shadow of simple silicosis, 5.

The differentiation between these *massive shadows* of infectious origin and the conglomerate shadows of far advanced silicosis is difficult and not always possible. Repeated reëxamination of the patient for evidence of change in the roentgenographic appearance of the lesion, penetration of the massive areas by over-exposure to analyze its internal structure, the clinical behavior of the patient, and repeated bacteriological examination of the sputum may all be necessary to determine whether an active infection is present.

It is hoped that the suggested terminology will receive a practical test. If others find it usable, the advantage of standard descriptive terms should outweigh the conservatism of those who are already using classifications of their own.

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