THE PATHOLOGY OF INTERNAL DISEASES

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identical with those of bronchial asthma. The entire picture is strongly suggestive of allergic inflammation; indeed there can be no other explanation. The allergic agent may not be the same in every instance. In some cases the larvæ of Ascaris lumbricoides which migrate through the lungs may be responsible, whilst in others the antigen responsible may be pollens or bacteria.

THE PNEUMOKONIOSES.

The inhalation of dust over a long period of time gives rise to changes in the lung of a proliferative and fibrotic character. These dust diseases or pneumokonioses differ in their course and prognosis, depending on the type of dust which is inhaled. The most important of these are silicosis, asbestosis and anthracosis, to which may be added hematite miner's lung.

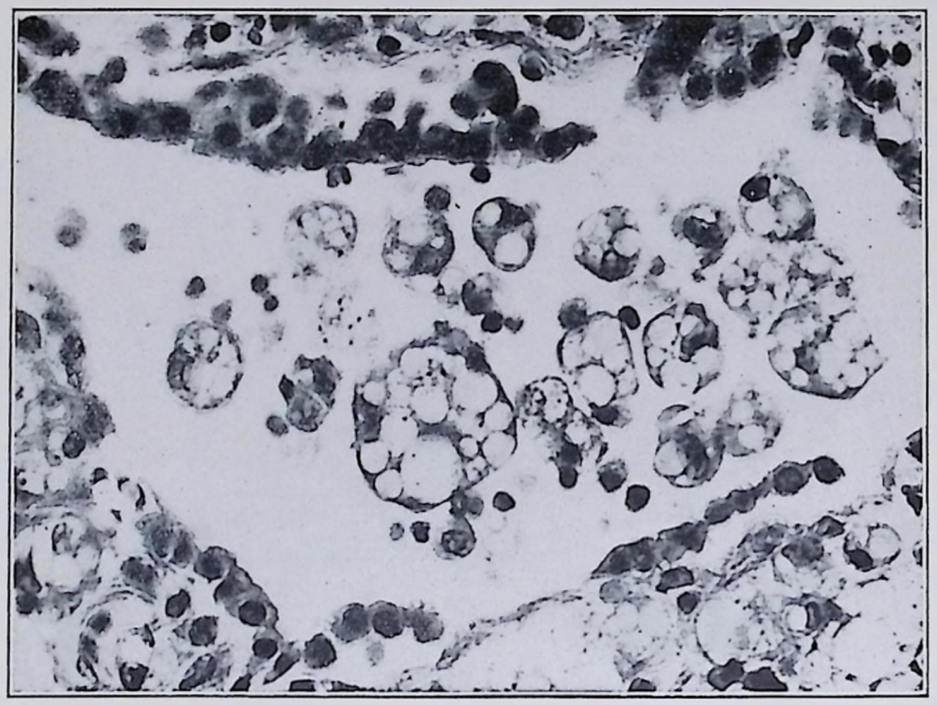


FIG. 76.—Phagocytic cells filled with oil droplets in a pulmonary alveolus. × 450.

Silicosis.—The cause of the disease is the inhalation of dust containing silica (silicon dioxide or quartz). This dust may be inhaled in the course of a variety of occupations. Indeed, as Kettle remarks, pneumokonioses or silicosis according to modern views are practically synonymous terms. It is among gold miners that it is most prevalent, but it may also occur in coal miners, stone workers, metal grinders, sand-blasters, etc. In the past many special names have been applied to the disease as it occurs in different occupations, *e. g.*, siderosis among iron workers, but in all cases the actual element in the dust which makes it dangerous is silica.

Those particles of silica which are not expectorated in the sputum are taken up by phagocytic cells in the smaller bronchioles and possibly the alveoli, and are conveyed by the lymphatics to the minute foci of

lymphoid tissue which are scattered through the framework of the lung. Here they set up a continuous chronic irritation as the result of which the local fibroblasts proliferate and lay down collagen so that the original focus becomes encased in layer upon layer of hyaline fibrous tissue. The result of this is to produce a large number of small, firm, gray nodules of concentric character, and these silicotic nodules in time coalesce so that large fibrous and even stony areas are formed throughout the lung. (Fig. 77.) The silica also reaches the bronchial lymph nodes, in which a similar series of changes takes place. Chemical analysis of the lung at this stage will reveal an enormously increased silica content. The silicotic lung is peculiarly dense in structure and of a gray color. In the earlier stages the silicotic nodules can be palpated

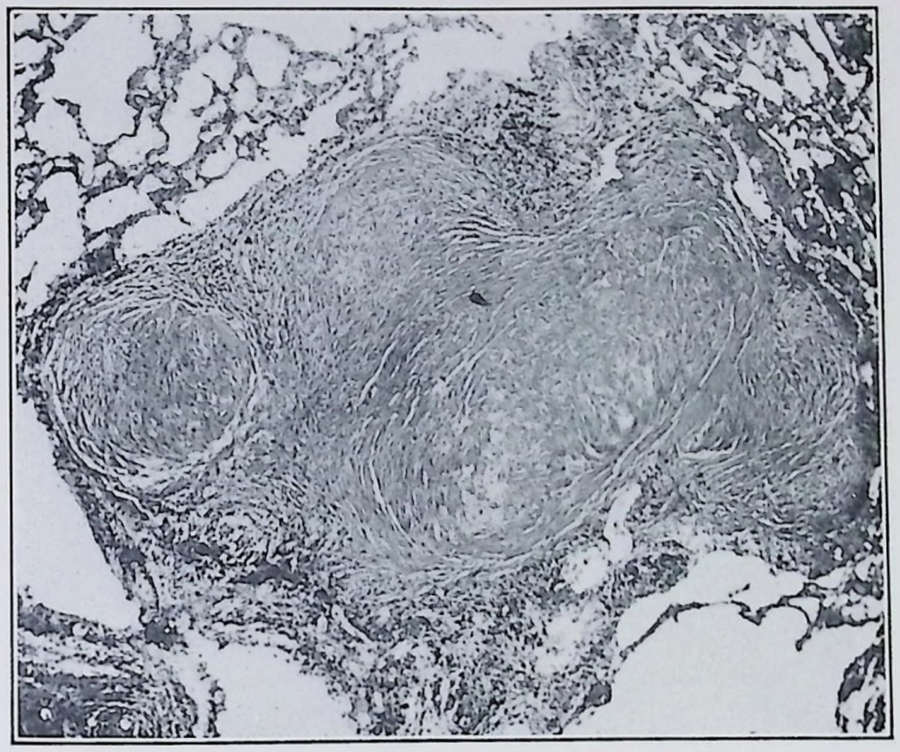


FIG. 77.—Silicotic nodules fused to form one mass. × 33.

and give a characteristic picture in the roentgen-ray film (Fig. 78). In the later stages larger areas may become stony hard, as in one case which I examined in which the lung had to be sawn open. The presence of silica in the lung leads to necrosis as well as to fibrosis, owing to the fact that it becomes slowly dissolved by the tissue juices and in solution exerts its necrotizing action. If it is injected in colloidal solution into the subcutaneous tissue of an experimental animal, necrosis rapidly ensues.

When the lesions are produced by pure silica dust, as in gold miners and sand blasters, the nodules have the clean-cut, laminated, onion-like character described above. When, however, as is frequently the case, the silica is mixed with other dusts, as in granite workers and anthracite coal miners, an interstitial fibrosis is added, and tongue-like

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projections extend from the nodules into the surrounding tissue. Moreover dust inclusions are commonly seen when the lesion is due to mixed dusts; these are seldom present in cases of pure silicosis.

In the human disease, however, although it must be admitted that silica by itself may lead to necrosis and breaking down of tissue, it is the association of tuberculosis with silicosis which gives to the condition its great importance as an industrial hazard. A number of years ago Gye and Kettle showed that when silica was injected into an animal at the same time as tubercle bacilli, the resistance of the tissues was lowered to such a degree that the bacilli multiplied with great



FIG. 78.—Silicosis. There is an unusually massive involvement, and at autopsy a saw had to be used to open the lungs.

rapidity. All the subsequent experimental work, particularly that of Kettle, has confirmed this observation. Thus, when silica is injected subcutaneously, and on the following day tubercle bacilli are injected intravenously, the bacilli become localized at the site of the silica injection and cause a rapidly destructive lesion. Kettle made the interesting observation that when the crystalline silica is coated with iron oxide and thus rendered insoluble and inert, it no longer exerted an activating action on the bacilli. Kaolin, which is a silicate, was also found to be a dangerous dust. The actual cause of much of the industrial silicosis appears to be cericite, a potassium aluminium silicate.

The deadly effect of silica when combined with tuberculosis is well exemplified by the experiments of Gardner with the "R1" strain of the tubercle bacillus, which is non-lethal for guinea-pigs and produces

regressive lesions. When guinea-pigs with pulmonary lesions caused by this strain are exposed to long-continued inhalation of quartz dust, the tuberculous lesions become progressive and kill the animals.

Although the combined effect of silica and tubercle bacilli is so important, it must be noted that the lesions of silicosis and tuberculosis are essentially similar. Every type of cellular reaction characteristic of tuberculosis can be found in pure experimental silicosis. An explanation of this remarkable similarity of effect may perhaps be found in the observation of Fallon that a phospholipid can be extracted from silicotic nodules which when injected into rabbits produces a fibrotic reaction identical with that caused by the phospholipid fraction of the tubercle bacillus. It is possible that this phospholipid comes from the mononuclear and epithelioid cells which collect in the early silicotic lesion.

Of practical importance in mining operations is the fact that in the experimental animal silica in combination with the irritating gases derived from explosives used in blasting causes silicosis very much sooner than when silica alone is used (Robson, Irwin and King).

Irwin and his associates have made the important observation that the presence of metallic aluminum inhibits the destruction and fibrosis caused by silica. This is probably due to a fine film of adsorbed aluminum hydroxide being formed on the surface of the quartz particles, so that these are shot off from the tissues with which they come in contact. By adding aluminum dust to air containing quartz dust it was possible to prevent the development of silicosis in experimental animals. The possibilities of industrial application are obvious.

The *radiological appearance* of the silicotic lung is of great importance, because it forms the basis for the clinical diagnosis. The earliest change is an increase in the normal reticulation. The specific appearance, however, is that of clean-cut nodules scattered widely throughout both lungs, but in cases due to mixed dusts the outline of the nodules is much more hazy. When calcium is deposited in the nodules they stand out still more sharply. When tuberculous infection is added the nodules develop a fluffy outline, increase in size, and eventually coalesce to form large shadows; this is in contrast to pure silicotic lesions which may remain discrete for years.

Asbestosis.—Asbestos is a fibrous mineral which consists essentially of magnesium silicate. Inhalation of asbestos dust may therefore give rise to a form of silicosis, but without the close relation to tuberculosis which exists in the dust disease produced by the inhalation of silica. Those liable to develop the lesion are not the miners who obtain the crude product, but the workers engaged in the process of separating the asbestos fiber from the crushed mineral. The lower lobes of the lungs are chiefly involved and the lesions take the form of a diffuse fibrosis in place of the whorled nodules characteristic of silicosis. The pleura is thickened and firmly adherent to the diaphragm. Emphysema and bronchiectasis are common, so that there is marked obstruction to the pulmonary circulation and right-sided heart failure develops. Bronchitis is common and terminal bronchopneumonia is of frequent

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occurrence. Death from pure asbestosis is much commoner than from pure silicosis.

The asbestos occurs in the lung in two forms: (1) small black particles of a material which is present in the crude asbestos and is liberated in the process of separation or carding; (2) "asbestos bodies." These latter structures are peculiar in form and highly characteristic. (Fig. 79.) They are long and tapering, up to 75 microns in length, and are of a golden yellow or brown color. They tend to be segmented and each end presents a bulbous expansion, giving the appearance of a greatly elongated dumb-bell. They appear to represent deposits around fragments of asbestos fiber. These bodies are found not only in the lung, often surrounded by giant cells, but also in the sputum. Their presence in the sputum enables a clinical diagnosis to be made.

There is a possible relationship between pulmonary asbestosis and carcinoma, for the two conditions are not infrequently associated. This is particularly striking in Homburger's series of 8 cases of asbestosis, in 4 of which carcinoma of the lung was present.



Fig. 79.—Asbestos bodies. × 510.

Anthraco-silicosis.—Much work has been done in recent years on anthracosis, but the subject is still in a state of confusion. One reason for this is the different composition of coal in different districts, and the varying type of work on which the men are engaged. This work may be (1) drilling hard rock for anthracite, (2) working at the soft coal face, and (3) trimming or bunkering coal on ships.

In drillers the lesions are identical with those of the classical silicosis of gold miners, *i. e.*, characteristic whorled fibrotic lesions. Workers at the coal face and trimmers in the holds of ships are exposed to an atmosphere saturated with coal dust in which there is a varying percentage of silica. In both of these types of worker the lesions are those of *anthraco-silicosis*. In microscopic sections the silica is masked by the coal, but it can be shown up by means of microincineration and examination with crossed Nicol's prisms. Gough, however, emphasizes the fact that the dust to which men working in ships' bunkers are exposed may have a very low silica content, and he doubts whether the presence of silica is a complete explanation of the lesions.

The gross appearance of the lungs in anthraco-silicosis is one of widespread extreme pigmentation. Microscopically there are areas of ill-defined fibrosis, which give a characteristic *reticulation* in the *x*-ray film, with fine net-like shadows. Belt has shown that the lesions responsible for the reticulation are innumerable dust-ridden cobwebs containing argentophil reticulum fibers; there is none of the dense collagen formation of true silicosis. In more advanced cases there may be definite nodulation. Finally massive fibrosis may be present,

particularly in those parts of the lung where tuberculosis is common. One of the most characteristic features is the presence of heavy pigment deposits surrounded by focal areas of emphysema, as may be seen in the illustrations of the papers of Gough and of Heppleston dealing with pneumokoniosis in Welsh coal workers. The foreign particles seem to be deposited at the bifurcation of the respiratory bronchioles, thus interfering mechanically with alveolar expansion and giving rise to focal emphysema (Heppleston). Belt remarks that the collier's lung is in a very real sense his occupational log book; it retains a qualitative and indelible record of the mineral particles breathed during life.

Hematite Miner's Lung.—A new form of pneumokoniosis was described in 1934 by Stewart and Foulds among the hematite miners in Cumberland. Many of these men died with grossly fibrotic lung lesions and both silica and hematite dust were present in the lungs in large amount. The gross appearance is highly characteristic, as may be seen in the colored plates in the original paper, for the fibrosed areas are of a bright brick-red owing to the hematite dust. Some of the areas are dark red or chocolate-brown. The fibrosis is diffuse rather than nodular, often massive, and most intense in the upper half of the lung. Tuberculosis was present in more than one-half the cases. The condition is a sidero-silicosis due to inhalation of silica-containing hematite dust. The Prussian blue reaction for iron is given by the brown but not by the bright red areas.

Bauxite Pulmonary Fibrosis (Shaver's Disease).—Another form of industrial pulmonary fibrosis which is now making a first appearance is that due to bauxite, an alumina abrasive. The irritating dust contains a mixture of silica and alumina. Workers with this material develop a diffuse reticulated fibrosis of the lungs accompanied by the formation of large emphysematous bullæ. The absence of the nodularity, so characteristic of silicosis, at once arrests attention. The microscopic picture is one of fibrotic thickening and hyalinization of alveolar walls, giving a "starched" appearance which is well seen in the illustrations of the paper by Wyatt and Riddel. (Fig. 80.) There is profound dyspnea and other features of a rapidly progressive and extreme emphysema complicated by pneumothorax due to rupture of the large bullæ. The radiological picture is so startling and individual that it first suggested to Shaver that this condition was quite distinct from ordinary silicosis, the principal features being diffuse, lace-like and granular shadows, with marked widening of the mediastinum and collapse of the lung. After a relatively short course the cases end fatally.



Fig. 80.—Bauxite fibrosis. × 210. (Kindness of Dr. J. P. Wyatt.)

Beryllium Pneumonitis.—Beryllium is used in the manufacture of fluorescent lamps, and in beryllium-copper alloy casting for precision instruments, airplane pipe

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lines, etc. Workers in these industries may suffer from an acute pneumonitis or from delayed beryllium poisoning. In the *acute form*, which is only seen in beryllium extraction plants, there is rapid onset marked by intense dyspnea, and unless fatal early, complete recovery takes place promptly when the worker is removed from the environment. In the fatal cases there is diffuse infiltration of the lungs with plasma cells, edema and hemorrhage, with a characteristic absence of polymorphonuclears. In *delayed poisoning* the symptoms may appear months or even one or two years after leaving the work. There is cough, severe dyspnea and cachexia, the condition being progressive and of grave prognosis. There is danger of right heart failure. The *x*-ray picture is usually diagnosed as miliary tuberculosis or sarcoidosis. The chronic granulomatous lesions also resemble these diseases, being characterized by giant cells of the foreign body type surrounded by epithelioid cells, with lymphocytes at the periphery. Conchoidal bodies are often present either in the giant cells or in the granuloma. There is gradual extension with organization and fibrosis. Similar lesions are found in the hilar lymph nodes.

SUPPURATIVE CONDITIONS OF THE LUNG.

An increased amount of attention has been paid of late to suppurative conditions in the lungs. We are coming to realize that many cases, which for years have been regarded as tuberculosis, are in reality not tuberculous at all, but are suppurative in character. Our large sanatoria are full of such cases, and the differentiation of these from true pulmonary tuberculosis has always been a difficult problem. The frequency of operations on the mouth and nose has added greatly to the number of septic infection of the lungs. Bronchoscopic examination has revealed the important rôle which foreign bodies of certain type may play in the production of these disturbances. And last, though not least, the introduction of lipiodol as a means of investigating the condition of the bronchial tree has provided a tremendous stimulus to our interest in these matters.

Of these conditions in which suppuration and destruction of tissue are the principal feature, the most important are abscess and gangrene of the lung and bronchiectasis. Abscess and gangrene are so closely related that they will be considered together. Bronchiectasis should logically be taken with diseases of the bronchi, but for reasons which will become apparent it will be more instructive to consider it in this connection.

Abscess and Gangrene of the Lung.—As has just been remarked, while these two conditions are by no means identical, they have so much in common that it will save needless repetition to consider them together. The principal difference is that abscess is a suppurative condition more or less circumscribed, while the latter is putrefactive and tends to be diffuse.

Etiology.—A lung abscess may develop in the following ways: (1) By aspiration of septic material. The passage of a foreign body into the bronchial tree may be included here. Bronchiectasis may also give rise to an abscess of similar type. All of these may be said to be intrabronchial. (2) A pneumonic or bronchopneumonic area may break down with the formation of an abscess. A primary carcinoma of the lung may behave in a similar manner. (3) Septic emboli carried to the

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