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PULMONARY ASBESTOSIS[1]

By J. V. SPARKS, M.R.C.S. (Eng.), D.M.R.E. (Cambs.), London, England

THE fact that the inhalation of asbestos dust produces an effect upon the lungs was observed many years ago, but the serious results which follow have only more recently been recognized. The pathologic lesions which it produces and its symptomatology have been studied in more detail just recently at the City of London Chest Hospital, by Dr. W. Burton Wood and Dr. S. R. Gloyne. As the result of the study of the radiologic appearances in a series of patients suffering from this disease we have been able to gain some idea of the changes it produces in the lung, which may, I hope, in future be of some value in the further study of this condition and its earlier recognition. This disease was described by H. K. Pancoast and E. P. Pendergrass (12), of Philadelphia, as long ago as 1925, since which time practically no literature on the subject has been published in America, so far as I am able to learn, apart from a recent editorial in the *Journal of the American Medical Association* (1) describing the death of a patient from pulmonary asbestosis. This patient apparently commenced to be exposed to the dust as long as 32 years before death. This would not suggest that the disease was a very serious one from the point of view of shortening the duration of life.

Asbestos, which was known to the ancient world, is a silicate occurring in minerals in combination with iron, magnesium, calcium, or aluminum and is quarried or mined in various parts of the world, including Italy, South Africa, and Canada. Of the asbestos imported into England, 80 per cent comes from Canada, so that it seems unlikely that you in the United States are dealing with a type of asbestos different from that employed in England. It seems reasonable to suppose, therefore, that an effect should be produced on the lungs similar to that produced in England, providing that the manufacturing processes are carried out in the same way. Unfortunately, I have no knowledge of the processes employed in America. It may be that we are more economical in our country in that we make use of the short asbestos fibers which would otherwise be wasted and they are used for dry cloth weaving. In its natural state asbestos differs from other minerals in that it is fibrous, occurring in long silky strands which are highly resistant to heat, strong acids, and alkalis.

During the manufacturing processes the workers may be exposed to dust containing varying amounts of asbestos. In some of these processes the asbestos content is very high and in others much lower, the higher asbestos content being present in the air of the rooms devoted to the textile branch of the industry. I have no knowledge of the effect on the workers mining the crude material as there are no mines in England.

It has been estimated by Dr. E. Merryweather (2), one of His Majesty's Inspectors of Factories, that at the present time something over two thousand workers in England are exposed to the inhalation of pure asbestos dust, as distinct from a very much larger number of workers who may be exposed to asbestos admixed with other products. In his report, however, it is very striking to notice the preponderance of workers who have been employed in the asbestos factories for a period of four years or less. It seems reasonable to suppose that after some years of such employment the

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workers may become affected by the inhalation of asbestos dust and seek other industries. The fact that they cease to be exposed to the dust does not, however, prevent the disease, when once established, from progressing.

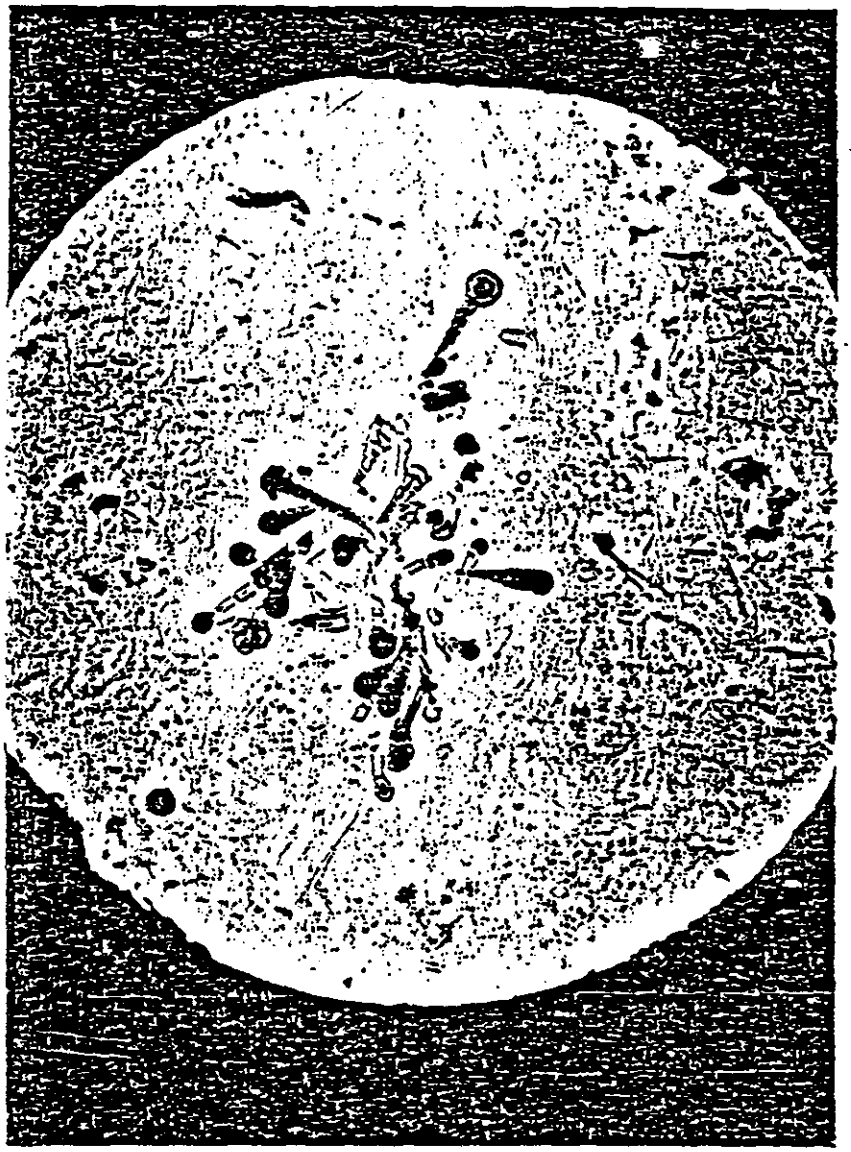

Fig. 1. Photomicrograph of tissue from lung showing asbestosis bodies (× 760).

Asbestos is usually broken up into short lengths before importation into England, where it is crushed and disintegrated and sometimes mixed with various substances for hardening into sheets, tiles, building and insulating materials, or brake linings. Sometimes it is carded, spun, or woven into various products like mats, mattresses, fire-curtains, or used as an inert filling of a heat-resisting character. That the asbestos industry is a growing one is shown by the fact that the importation of asbestos has trebled within the last five years, and it seems likely that the study of the dangers which accom-

pany it will become of increasing importance.

When examined microscopically by dark-ground illumination the fiber gives the impression of a sharp, brittle metallic wire broken off at various angles and in different lengths, and, being highly refractile, it has the appearance of the glowing filament of an electric bulb.

The fibers can be found in the nose and mouth and in the skin lesions of the workers. When inhaled into the lung the fibers set up a pneumonoconiosis of a characteristic variety.

ETIOLOGY AND PATHOLOGY

The first fatal case of pulmonary asbestosis recorded was that by H. Montague Murray (3), who made a postmortem examination of a case at Charing Cross Hospital in 1900. The next case recorded was by W. E. Cooke (4) and Stuart McDonald (5). In the lungs of this patient they noted the presence of certain curious golden yellow bodies having the appearance of minute crustacean forms.

In 1929 Stewart (6) and Haddow (7) showed that these curious bodies could be found in the juice expressed postmortem from the lungs in cases of pulmonary asbestosis, and that they also could be detected in the sputum. It was these writers who first suggested the name of "asbestosis bodies" for these structures (Fig. 1).

Later in 1929, S. R. Gloyne (8) showed by means of dark-ground illumination that when an asbestos body was dissolved in concentrated sulphuric acid it had a central core consisting of a minute asbestos fiber. He has also demonstrated the presence of foreign body giant cells in the lung sections from a guinea pig, and states that it is important to demonstrate tubercle bacilli in the human lung in the presence of asbestosis before assuming that tuberculosis is present as a complicating factor.

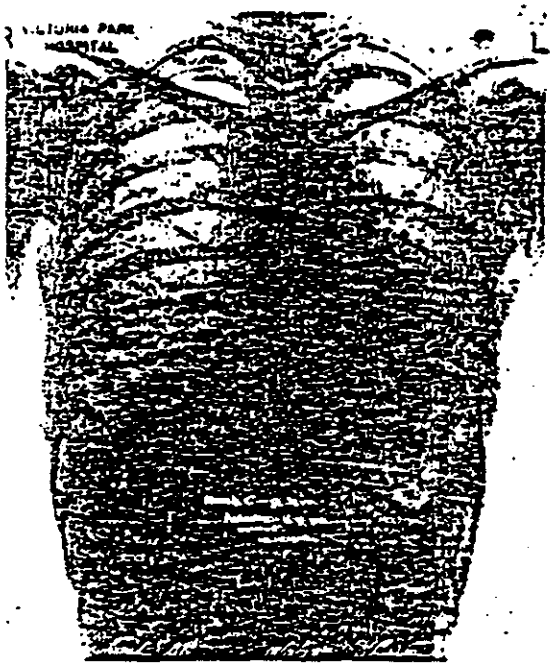

Fig. 2. Roentgenogram of chest of asbestos worker: exposed for nine years: duration of symptoms, three years.

Up to the present we have examined some fifty cases at the City of London Chest Hospital and have six records of postmortem findings.

The *Symptoms and Physical Signs* are described by W. Burton Wood (9) as follows:

The cardinal symptoms are dyspnea and cough; the former is in many cases the earliest symptom noted by the patient, who complains of slight breathlessness on hurrying or going upstairs, or that his chest "feels stuffy." He, therefore, discards the respirator worn hitherto under protest. In the late stage of the disease dyspnea may be extreme, and slight exertion may give rise to labored breathing. Cough is a variable symptom and, though usually present, it may be absent for long periods. It is either dry or accompanied by the expectoration of a little viscid phlegm. The sputum in this, as in other chronic pulmonary diseases, may on occasions be streaked with blood; this

is, however, exceptional. The only frank hemorrhage noted in our series of cases occurred in an asbestos worker who was also

PHYSICAL SIGNS

When the disease is established the skin has an unhealthy leaden hue. Cyanosis may

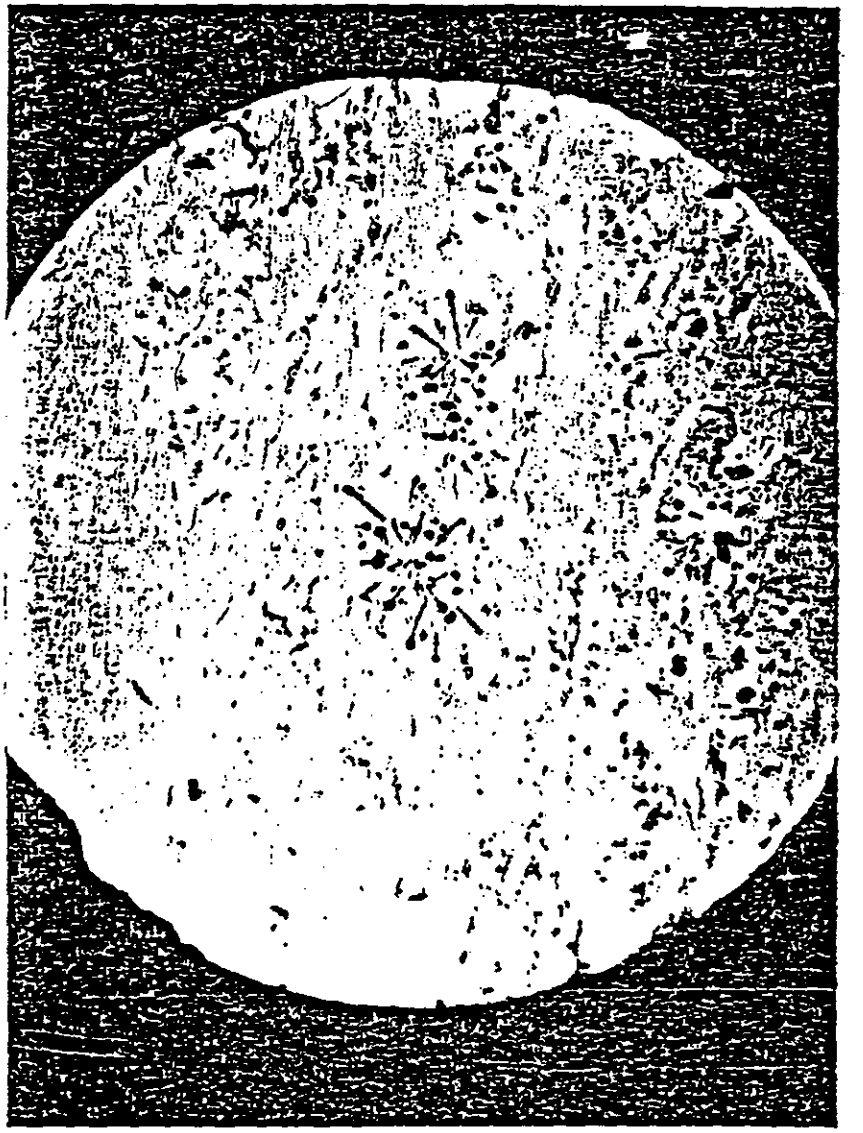

Fig. 3. Photomicrograph of section of lung of case shown in Figure 2.

suffering from adolescent phthisis, with cavitation.

Many patients complain of anorexia, lassitude, pains in the chest, and loss of weight; this latter is a noteworthy feature, for the wasting may be progressive and in the late stages is sometimes extreme.

be absent, slight, or sufficiently evident to cause a dusky complexion. The chest is poorly covered and expansion is defective—it may be reduced to one inch or less. The vital capacity of one of our male patients was only 1,600 cubic centimeters. Clubbing is seldom a marked feature, but a slight

swelling of the skin above the proximal ends of the nails is often apparent. Corns due to the irritation caused by the asbestos fibers in the superficial layers of the skin of the hands are occasionally seen. The skin in other exposed positions, *e.g.*, the legs of girl workers, may be similarly affected.

The physical signs in the lungs are those of pulmonary fibrosis, limited to, or predominant at, the lung bases. The coarse creakings and crepitations usually associated with fibroid change are, however, replaced by fine dry crackles. These may arise in the pulmonary parenchyma or be due to the movement of slightly roughened pleural surfaces. A definite friction rub may be heard over one or the other axillary base. As the fibrosis is bilateral there is seldom any appreciable cardiac displacement.

CLINICAL DIAGNOSIS

When the patient's occupation is known diagnosis should seldom be difficult except in the early stage of the disease. The history of cough and dyspnea, the signs of pulmonary fibrosis and the situation and quality of the adventitious sounds, the radiologic appearances, and the presence of asbestos bodies in the sputum combine to form a distinctive clinical picture. The presence of asbestos bodies in the sputum does not necessarily mean that the patient is suffering from pulmonary asbestosis, but merely that he has been subjected to the inhalation of asbestos dust. The previous occupation of the patient may be of importance in excluding the possibility of fibrosis from other causes.

PROGNOSIS

It is too early in the study of this disease to say very much on this subject, but once the asbestos bodies appear in the sputum the course of the disease would appear to be progressively downwards, nor does the cessation of exposure to the dust avail to check its spread. Symptomatic treatment is disappointing as we have no means of relieving the dyspnea which is the patient's chief complaint. Prophylaxis is all-important and the only hope for the asbestos worker lies in the adoption of the proper means of protection against the risk attendant on the inhalation of the fibers.

POSTMORTEM FINDINGS

The findings in one case (N. C., female, aged 34, Fig. 2) are described by Dr. Page (10). The body was emaciated. The pleurae were uniformly thickened to a slight extent on both sides; some recent plastic pleurisy was present. The lungs showed a diffuse fibrosis and were contracted—the left lung more than the right. On section, the trabeculae stood out rather prominently, forming a fibrous network, especially in the right upper lobe. The bronchi were only slightly dilated. The lungs were congested and in patches were bronchopneumonic. Microscopically the fibrosis became more readily apparent in less affected areas; it was more prominent around the blood vessels and less so in the alveolar walls, while in some sections little was seen but scar tissue, with blood vessels and numerous elastic fibers. Some lymphocytic nodules were present in the fibrous tissue, and giant cells of the type associated with foreign bodies. No typical tuberculous giant cells were found and no tubercle bacilli. In addition to a large quantity of amorphous dark brown pigment, numerous golden asbestosis bodies were seen, the majority of them embedded in fibrous tissue. If the fibrosis was less advanced, many were seen to be in clumps in the alveoli (Fig. 3). Varying in size and shape, the majority showed a large clubbed head, a segmented body, and a tapering tail. The hilum glands were pigmented, but were only slightly enlarged and no asbestosis bodies were found in sections

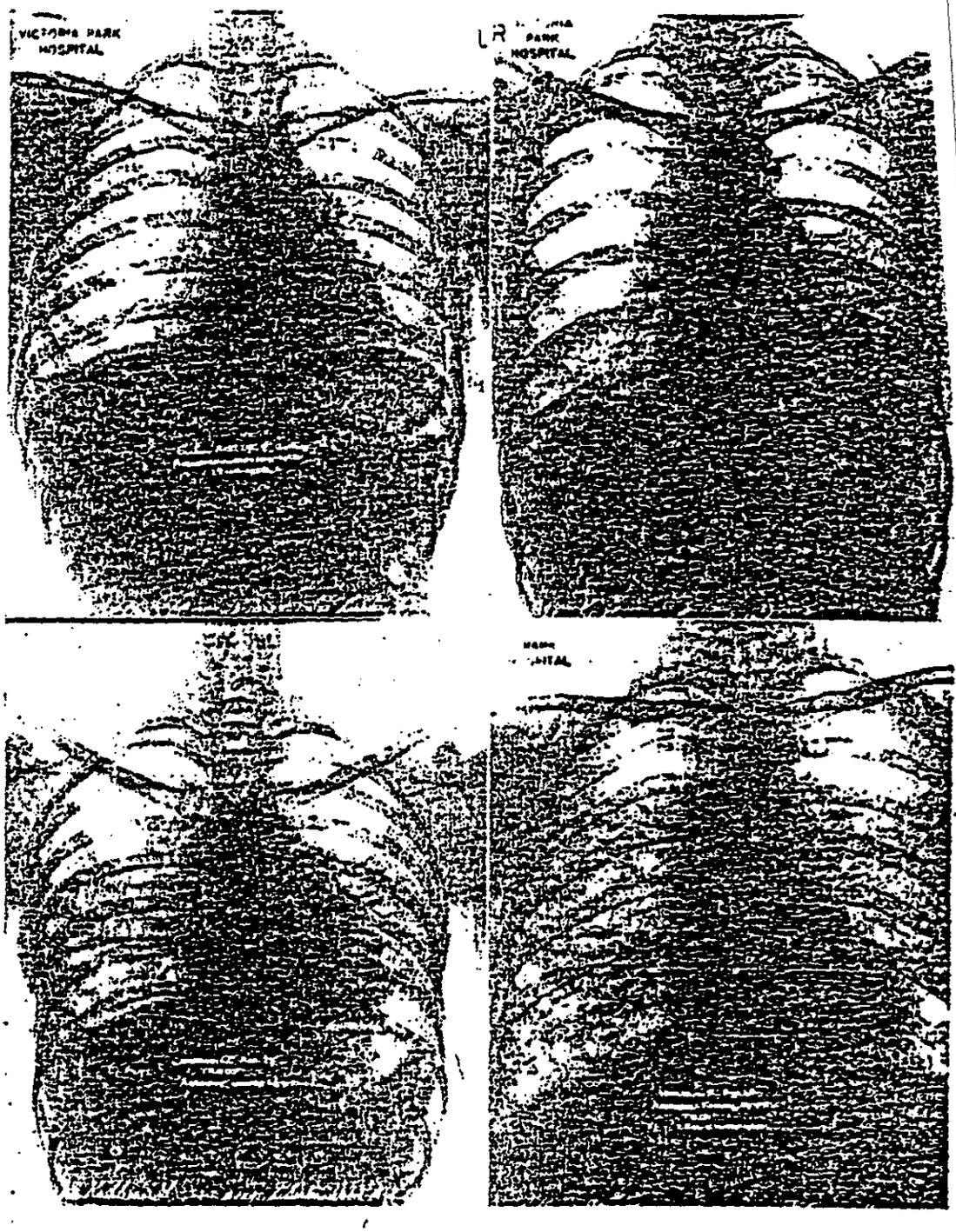

Fig. 4 *(upper left).* Roentgenogram of chest of asbestos worker, exposed for four years.

Fig. 5 *(upper right).* Roentgenogram of chest of patient who showed asbestos bodies in the sputum twelve years after leaving the factory.

Fig. 6 *(lower left).* Roentgenogram of chest of individual who has worked six and a half years in asbestos.

Fig. 7 *(lower right).* Roentgenogram of chest of individual who has been an asbestos worker for fourteen years.

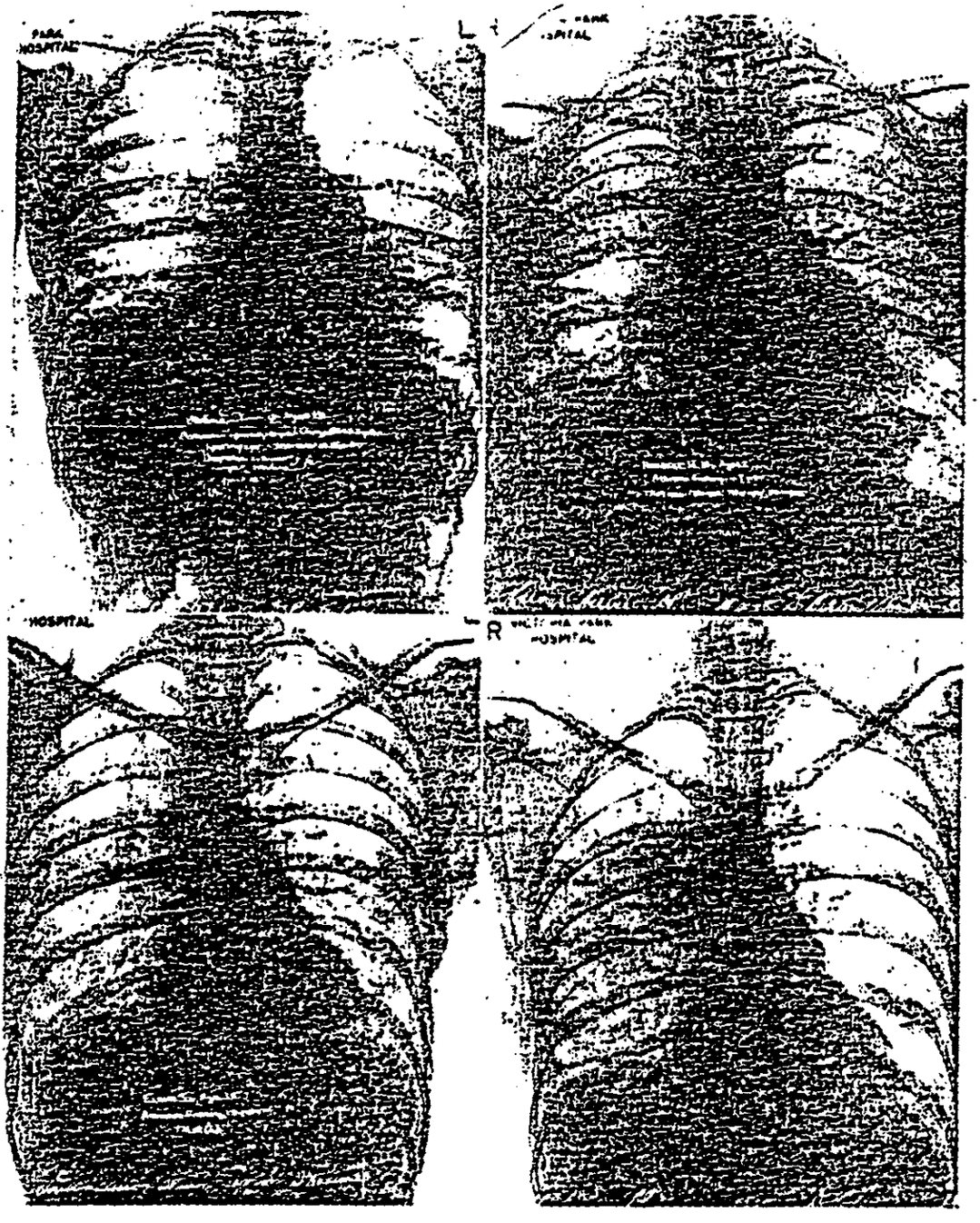

Fig. 8 *(upper left).* Roentgenogram of chest of patient who was an asbestos spinner for twelve years and had symptoms for three years.

Fig. 9 *(upper right).* Roentgenogram of chest of patient who was an asbestos worker for seven years. He was under observation for two years, when he developed tuberculosis.

Fig. 10-*A (lower left).* Roentgenogram of chest of patient who for thirteen years was an asbestos worker. Tuberculosis developed. (*Cf.* Fig. 10-*B.*)

Fig. 10-*B (lower right).* Tuberculous lesion in patient shown in Figure 10-*A.*

prepared from them, neither were any found in the sections prepared from the liver, spleen, and kidney. The fluid expressed from the lung was found to contain asbestosis bodies and also free asbestos fibers.

It is seen that these asbestosis bodies are found mainly in the alveoli of the lungs. They consist of a central asbestos spicule surrounded by a colloidal aggregate of blood protein and possibly an iron salt, and one wonders whether their formation may not be a protective action on the part of the body. One can easily imagine that these fibers which are inhaled right into the alveoli can set up an inflammatory reaction in the pleura. One of the first radiologic signs is often a dry diaphragmatic pleurisy.

RADIOLOGIC APPEARANCES

The radiologic appearances of pulmonary asbestosis are usually quite typical in the radiogram when the condition is well advanced. In the early cases they may easily be overlooked, especially when one observes the radiogram without any knowledge of the clinical history. This latter method of observation is always practised in our hospital, so as to give the physician an unbiased opinion of the radiologic appearances. A well-advanced case shows some of the appearances enumerated in the next paragraph.

The diaphragmatic movement is limited; its outline tends to be indistinct and is sometimes uneven. There is clouding in the costophrenic angle due to thickening of the pleura, a thickening often seen to extend along the costal margin on both sides to the apices. The heart outline is often poorly defined, showing a ragged outline due to changes in the lung around the pericardium (Fig. 4). It is not displaced in position unless the degree of fibrosis is greater on one side than on the other. A prominence has often been noted in the left auricular or pulmonary area. No appreciable displacement of the mediastinal contents is usually apparent. The lung fields at first show a slight relative increase in density in the lower zones, due to a lack of air entry, without any appreciable accentuation of the bronchial striations (Fig. 5). Sometimes some small calcareous deposits are seen scattered in the lower zones of approximately the same size as the calcifying tubercle, but they have a slightly lesser density and are irregular in outline. Later, there appears a patchy increase in density in the lower zones, which may also involve the mid-zones, producing a fine network of fibrosis (Figs. 6 and 7), which does not appear to be of a bronchial type but, rather, alveolar or perivascular.

These appearances are described in contrast to the coarse fibrosis seen in silicosis, combined with the occurrence of fairly large nodules of varying density in the lung fields and around the hila. The fact that there are comparatively few visible changes in the radiogram in asbestosis when compared with silicosis does not in any way mean that they are of a less serious nature or less advanced, as the density of the silicotic lesion would appear to be greater and the fibrosis of a coarser character, involving localized areas of the lung, whereas in asbestosis the fibrosis would appear to leave very little intermediate undamaged lung (Figs. 9, 10-*A*, and 10-*B*).

The study of this subject has emphasized to me the importance of comparative radiography of the chest (10). One hopes in the future that it will be made possible for us to examine the workers before they commence their employment in factories where asbestos is used, so that a comparative study can be made of the lung condition at yearly intervals. When making this examination it will be essential to employ the method of comparative radiography which we have at-

tempted with considerable success during the past three years at the City of London Chest Hospital.

All these patients, having symptoms, came voluntarily to the hospital, so it must not be expected that such gross changes will be discovered by a routine examination of a group of workers from an asbestos factory.

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