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PULMONARY ASBESTOSIS

A Report of a Case and a Review[1]

WILLARD B. SOPER

This case is presented because apparently it is definitely one of pulmonary asbestosis, a condition that would appear to be much more widespread than at present suspected and regarding which but little has as yet appeared in American medical literature. Only very recently has interest been aroused, stimulated particularly by a number of articles and studies in the medical press of England. Just as certain other dusts took their toll over a long period before the medical profession awoke to the true facts, so asbestos dust to a lesser degree appears to be taking its toll without sufficient recognition of the fact on the part of physicians.

Case Report

Mr. K., a man of 30 years, was admitted to the William Wirt Winchester Hospital on May 26, 1929, on account of symptoms suggesting pulmonary tuberculosis.

The family history was completely negative for tuberculosis. The patient had been well and strong as a boy; he had the usual childhood diseases and rather frequent head colds. At the age of ten years he suffered for a short time from an ailment diagnosed malaria, and recovered completely. He had an attack of burning micturition associated with haematuria about five years before admission. For the latter trouble he was cystoscoped without a diagnosis being made. The urinary symptoms then ceased entirely, never to recur. He began work in an asbestos factory at the age of 17 and continued in the industry with more or less constant exposure to the dust from then on.

The onset of the present illness was in the fall of 1928 with dyspnoea. Some malaise was also noted. He continued at work until February, 1929, having lost seven pounds in the interim. In February, following exposure to smoke in a factory fire, the sputum became blood-streaked and continued so off and on up to admission, although there was never any frank haemoptysis. On account of these symptoms the patient gave up his work in February and continued under his physician's care, with the dyspnoea rather increasing,

[1] From the William Wirt Winchester Hospital, West Haven, Connecticut.

a little morning sputum blood-streaked at times, malaise and a burning pain in the chest.

On admission to the hospital the symptoms were a little whitish morning sputum, about one dram in amount and blood-streaked occasionally; burning pain over the whole chest, quite severe at times; rather marked dyspnoea on exertion; nervousness; irritability and constipation of three months' standing. His best weight was stated to have been 150 pounds, usual weight 142 to 145; the admission weight was 142 pounds. The sputum had been examined for tubercle bacilli on several occasions between February and May and was always negative.

Physical Examination: The patient was fairly well nourished, a little pale and the skin cyanotic on pressure. He did not look particularly ill. The hands were clammy. The left pupil was slightly larger than the right but reactions were normal. Several teeth were missing and those that remained showed a moderate amount of pyorrhoea with congestion of the gums. The tonsils were a little large and a very few small cervical lymph nodes were just palpable along the margin of the left trapezius. The thorax was well formed and symmetrical and expanded equally on both sides. The measured expansion at the nipple line was 5 cm. The heart was in normal position and no abnormalities were noted except a slight arrhythmia. Percussion showed a little dulness above both clavicles and a practically normal note otherwise over the anterior chest. Posteriorly there was a little dulness over both tops down to the 6th dorsal spine and more marked on the right. The note at the bases was hyperresonant. Breathing was bronchovesicular over the upper third on both sides. There were a few dry crackling superficial râles at both bases about the whole circumference of the chest. There were also occasional râles of the same sort scattered over the whole of both lungs, but definitely more marked in number between the outer clavicle and the 3rd rib on the right. The abdomen was normal. There were no lymph-node enlargements except as noted above. The genitalia were normal. Reflexes were normal.

The fingers showed no clubbing. The hands, however, were interesting in that there was on the right hand between the thumb and the first finger a fibrous mass the size of the end of one's little finger, an outgrowth of the skin. This was fairly soft in consistence, about that of a fibroma; it was non-tender and was said to have resulted from a sliver of asbestos penetrating the skin. Also two of the fingers showed what were called asbestos corns, namely, small hard areas each about a millimetre in diameter and said also to have been of long standing and the result of asbestos slivers.

Laboratory: The blood count showed 4,000,000 red cells with 75 per cent haemoglobin, white cells 6,250, with a differential of polymorphonuclears 68 per cent, lymphocytes 22 per cent, large mononuclears 8 per cent, eosinophiles 2 per cent. The urine was normal and the Wassermann negative. Ten

specimens of sputum examined for tubercle bacilli, six by the antiformin method were negative. A second blood count at the time of discharge showed no essential change from that on admission.

X-ray: The striking feature of the films was a rather diffuse fine type of shadowing that was rather more marked over the lower two-thirds of both



FIG. 1. X-RAY OF LUNGS, PULMONARY ASBESTOSIS, MAY 26, 1929

lungs. The trachea was drawn a little to the right opposite the clavicle; the mediastinal shadow was broad; both it and the heart shadow were in normal position. The right diaphragm appeared somewhat higher than the left, but the exact location was obscured by a hazy shadow blending into the lung field above. ntinuous at the lateral margin with a shadow which filled the

costophrenic sinus. The same condition existed over the left diaphragm but to a lesser degree. Above both clavicles was a shadow, rather ground glass in character, suggesting apical pleurisy. Extending out from the hilum on both sides in all directions were very fine striations which were particularly marked toward the bases and along the left margin of the heart where, to quote Burton Wood (1), they presented a cobweb appearance. These markings were essentially the same in both lungs. The right lung in addition showed a definite linear thickening of the fissure between the upper and middle lobes in the outer half of the lung. The inner end of this fissure pointed toward a group of two quite definite dense shadows as of calcified tracheobronchial nodes.

The patient was discharged on July 2, 1929. During his stay in the hospital the dyspnoea became less marked. No cough ever developed. The sputum remained slight in amount, thin in character and was raised without effort in the morning. It was occasionally blood-streaked. There were no genitourinary symptoms. The chief complaint was the burning sensation in the chest experienced before admission. The temperature was normal with an occasional 99°, the pulse 80 to 90, and respirations were usually normal, with an occasional 20 to 22.

Following discharge the patient rested at home with no regular work but up and about the house more or less. He presented himself for examination again on April 9, 1930, about nine months after his discharge. He stated then that the dyspnoea had become definitely more marked, and that the little morning expectoration had persisted unchanged with a little morning cough added. He suspected that his condition was due to a sinus trouble for which he stated he was being treated at the time. The sputum had been blood-streaked on several occasions, but still there had been no frank haemoptysis. The burning pain had ceased about December and had been superseded by a dull, drawing pain above the left breast. A certain amount of malaise and definite weakness on exertion continued. Weight had increased from the 142 on discharge to 162 pounds. The credit for this gain the patient gave to the codliver oil and tomato juice which he took consistently during the whole period. He was under treatment for his pyorrhoea.

The striking feature of the reëxamination was the dyspnoea. Respirations were 25 and even ordinary speech produced shortness of breath. The complexion was slightly leaden and there was a very noticeable cyanosis of the ears, hands and skin on pressure. There were no other changes from the original examination except in the thorax; this was symmetrical and gave the effect of being rather flattened over the upper half in front. On full inspiration, expansion in the anteroposterior diameter appeared to be nil, there being no visible motion of the sternum. The epigastrium moved forward strikingly with inspiration. There was some lag in the lateral expansion of the right base. At the nipple line the chest measured 94.5 cm. on extreme expiration and 96

cm. on extreme inspiration, an increase of only 1.5 cm. The lungs showed an unusual degree of difference between the notes on light and heavy percussion. With the latter no dulness was noted, but on light percussion there was a very definite wooden note over the whole of both lungs, especially pronounced on the right to the 5th dorsal spine behind and from the 6th rib to the base, both in the axilla and anteriorly. Liver flatness began at the 6th rib and did not change in either phase of respiration. On auscultation the breathing was a little diminished overall but not strikingly so, with a frank bronchovesicular quality over the right upper lobe to the 5th dorsal spine and to the 4th rib. A few nonlatent râles were heard over the whole of both lungs, but they were quite indefinite. With cough, however, a striking type of râle was heard from the top to the bottom on both sides but most markedly in the right axilla and below the 6th rib in front; it was superficial, fine, dry and crackling in character. The quality of râle was essentially the same overall. Blood-pressure was 130 over 90, practically the same as during the hospital stay. The general impression was that the râles were considerably more definite and more widespread than nine months before. The increasing dyspnoea and cyanosis seemed in accord with the increase in the râles. The vital capacity was 1350 cc., 29 per cent. Unfortunately this had not been determined at the time of the original admission. The new X-ray films were a little less exposed than those previously taken and brought out the markings still better. Discounting this fact, it was impossible to determine a definite X-ray change.

ASBESTOS

Before entering upon a discussion, something should be said of asbestos. This word is from the same in Greek, meaning unquenchable.

Asbestos was known to the ancients for its fire-resisting qualities. Greek and Roman writers refer to its use for lamp wicks. Allusion is made to it by Marco Polo, and Charlemagne is said to have possessed a remarkable table-cloth of this material which was purified by being thrown into a fire. Only relatively recently, however, has it come into extensive use in industry.

Asbestos is a silicate of numerous varieties. It is always found associated with other minerals, especially with chrome, iron and magnetite. Cooke (2) gives the composition of the Canadian and Italian fibres as follows:

	Canadian Chrysotile	*Italian Fibre*
Silica	40 87	40 30
Magnesia	41 50	43 37
Ferrous oxide	2 81	0 87
Alumina	0 90	2 27
Water	13 55	13 72

From the standpoint of pathology, however, the mineralogical composition of asbestos is very important. The following analysis is generally accepted:

	Canadian	*Rhodesian*
Asbestos	92.50	98 85
Magnetite	6.75	0 70
Quartz	0.25	0.10
Residue	0.45	0.35

From the foregoing chemical and mineralogical analyses it becomes evident that asbestos is made up mainly of a magnesium silicate containing but traces of free silica (quartz).

Formerly asbestos was obtained almost exclusively from Italy and Corsica and the Italian workings still yield large quantities. South Africa also supplies large amounts. But the Canadian asbestos, since the recognition of its commercial value in 1877, has become the most widely used industrially. It is found in a small belt of serpentine in the Province of Quebec, chiefly near Black Lake and Thetford. Elsewhere it is widely distributed, but in quantities too small to be worked to advantage commercially.

Cooke states that the fibre microscopically is seen to consist of two different elements: the translucent glistening material of which the bulk of the fibre is composed, and black opaque angular particles. Minute black granules are also present, actually part of the fibre. The dust generated during manufacture is seen to consist of these sharp angular particles and minute granules. Analyses of several samples of dust showed that the dust containing the greater quantity of the black particles contained the largest amount of iron. These particles were found in very small number in the finished article. Whereas the dust showed 18.4 per cent of iron as ferrous oxide, the finished article showed only 0.1 per cent. Cooke concludes that the blackened brittle parts of the fibre are the iron-containing portions, the bugbear of the manufacturer, the cause of dust, and a danger to the health of the workers.

The rock is blasted in open quarries, crushed and pulverized. In this form it is often shipped. At the manufactory it is mixed with other fibres, especially cotton. Then there are the processes of carding, roving and spinning which may be carried out just as with cotton, wool or silk. All of the processes produce asbestos dust, some more than others. Our own patient was engaged in the manufacture of brake-linings. He stated that the bands were composed of 30 per cent of asbestos fibre and 70 per cent of cotton fibres woven together.

DISCUSSION

Prior to the appearance of the above-described case, pulmonary asbestosis was practically unknown to most of us in New Haven. However, on account of the history of prolonged exposure to asbestos dust and the several respects in which the patient's symptoms, signs and X-ray films differed from those of tuberculosis, the condition was immediately suspected as possibly due to the inhalation of asbestos dust. During that very month of May, 1929, there appeared in the current number of *Tubercle* an article on *Pulmonary Asbestosis*, with special reference to the radiographic appearance, by W. Burton Wood (1). The description of the composite X-ray findings in 15 cases of pulmonary asbestosis described by Wood was so nearly identical with the X-ray picture of our case and the symptomatology so similar that we immediately concluded that we were dealing with such a case, with possibly a minimal tuberculosis superimposed. To this study by Wood, later studies by the same author, and the limited literature on the subject I am indebted for most of the material of this presentation. Wood in the above cited article in *Tubercle* gives a clinical abstract of 16 cases with reproductions of the films. A description of the composite skiagram of all of these cases is repeated verbatim.

Most noticeable feature of skiagram in the workers exposed longest to asbestos dust is the presence of shadows suggesting a diffuse fibrosis affecting chiefly the lower two thirds of the lungs. The fine quality of the shadows is worthy of note. Some of the cases exhibit a ground glass appearance, though on close inspection a fine mottling is evident. Bronchial striations toward the lung bases are prominent and seem to spread out to form a fine network giving an appearance like that of cobweb. As the striations radiate outward from the lung roots, they cut the edge of the cardiac shadow, especially on the left side, giving a shaggy appearance as if the heart were encased in coarse felt. When more definite mottling is present it lacks the coarse quality described in the skiagrams of chests showing pneumoconiosis, for example in South African coal miners. Another feature is evidence of basal pleurisy, and the frequency with which the costophrenic angle on one or both sides is obliterated will be noted. Many of the cases also exhibit a thickening of the apical pleural cap. Annular shadows suggesting a possibility of cavitation were seen in three cases; in only one of these were tubercle bacilli found in the sputum. The region of the lungs most apt to show marked changes in pulmonary tuberculosis of the fibroid type is usually spared.

Wood (1) is also quoted in large part as to symptoms, physical signs, differential diagnosis, prognosis and duration of exposure to dust.

SYMPTOMS

The cardinal symptom of pulmonary asbestosis, like that of other forms of silicosis, is dyspnoea. The dyspnoea is progressive and in advanced stages of the disease may become extreme. Chest expansion may be reduced to one inch or even less. Dyspnoea may be accompanied by cyanosis and the complexion of most patients has a slightly leaden hue. Wasting is a notable feature and may proceed to emaciation. Cough is a variable symptom, but is seldom excessive and may be slight. Expectoration is usually moderate and may be altogether absent over a long period. Particles of asbestos are apt to penetrate the superficial layers of the skin of the arms and legs of workers. Irritation by these causes keratinization, and "asbestos corns" are produced around the central core of fibre.

PHYSICAL SIGNS

If pulmonary tuberculosis supervenes, the signs and symptoms of this condition would obviously modify the clinical picture. In the absence of such a complication the physical signs are those of a bilateral pulmonary fibrosis affecting the pulmonic bases. The adventitious sounds have a dry crackling quality, and in the axillary regions are often superficial. The latter probably have a pleural origin. A friction rub may be heard over the same area. As the disease affects both lungs, cardiac displacement if present is usually slight. Early clubbing of the fingers may occur but is seldom well marked. When seen it is usually represented by slight swelling of the skin surrounding the proximal ends of the nails.

DIFFERENTIAL DIAGNOSIS

Features which serve to distinguish asbestosis and pulmonary tuberculosis, though a combination of the two conditions may be often suspected and sometimes proved, are the leaden or dusky complexion, extreme dyspnoea, wasting or emaciation out of all proportion to physical signs, and the dry quality of the adventitious sounds. These with an absence of tubercle bacilli from the sputum, and a history of prolonged exposure to asbestos dust indicate the probable diagnosis.

PROGNOSIS

When the disease is established the prognosis appears to be grave.

DURATION OF EXPOSURE TO ASBESTOS DUST

In Wood's series of cases the shortest period of working was one year and the longest period fourteen years. One patient was exposed to dust for a period of eight years before the onset of cough. Another case remained in fair health for five years after commencing work, although frequent colds and a tendency to winter cough were apparent. It is significant that a patient may complain of symptoms of pulmonary distress only some time after ceasing work. Wood states that a reference to the radiograms will show that in general the density and extent of the lung shadows are proportional to the duration of exposure to the dust.

PATHOLOGY

In 1924 Cooke (3) published in England a brief autopsy report which he then thought was of the first case. The patient had worked in asbestos twenty years. Adhesions were extensive. The lungs were firm and small and cut with abnormal resistance. The extensive fibrotic process appeared distinct from the scattered tuberculous foci. Cooke speaks of particles of mineral matter in the fibrous tissue and in the caseous tuberculous areas. The bronchi were dilated, the lining epithelium gone and extensive peribronchial fibrosis was present.

This report by Cooke brought to him word of the first autopsy on a case of asbestosis by Murray in 1900, the essential features of which were the pulmonary fibrosis and "spicules of asbestos." In 1927 Cooke (2) wrote a second more comprehensive article describing the histology of the lungs of the case originally reported in 1924. He described for the first time and showed microphotographs of deposits of large angular pigmented particles which he judged to be the heavy brittle iron-containing fragments of asbestos fibre. He explained the greater involvement of the right lung on the theory that the fragments would more easily travel down the more vertical right bronchus.

McDonald (4) about the same time published a study upon what he termed "the foreign bodies," the material being from Cooke's case and one other.

In 1928 Simson (5) wrote an article entitled *Pulmonary Asbestosis in Africa*. In it he described the findings in sections of lungs of four asbestos workers. Two suffered death from tuberculosis and two from pneumonia. The first two cases showed considerable fibrosis and the latter two only a moderate amount. All, however, showed "curious

golden-yellow bodies," which the author states he never found in autopsies of miners dying from silicosis and tuberculosis in the Rand. Considerable space is given to a discussion of the "yellow bodies." Simson thinks there is considerable evidence in favor of asbestosis conducing to tuberculosis as a complication. He cites Collis who found that five deaths from phthisis occurred in five years among a staff of less than 40 workers employed at a factory where asbestos was woven.

The next autopsy reported was in July 1929 by Page and Wood (6). This case was studied before death and was especially interesting in that there was no complicating tuberculosis. A woman had worked nine years as a weaver of asbestos and gave up the occupation on account of ill health about four years before death. Dyspnoea, loss of weight and strength continued increasingly until her demise. A description of this autopsy is worth repeating in part:

Body was emaciated. The pleurae were uniformly thickened to a slight extent. In the upper third of the chest the layers were bound together by moderately firm adhesions; 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; and denser fibrous strands appeared as irregularly shaped nodules particularly in the right lower 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 most 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 fibres. 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-yellow bodies resembling those described by McDonald and Simson were seen. These bodies were present in sections taken from various parts of the lungs. The majority of them were imbedded in fibrous tissue; where the fibrosis was less advanced many were seen to be in clumps in the alveoli, usually radiating from the centre with their clubbed ends outward; numerous isolated specimens were seen in the fibrous tissue and elsewhere. Varying in size and shape the majority showed a large clubbed head, a segmented body and a tapering tail. Many circular bodies with

a diameter of about one and a half red blood corpuscle were also present. These were evidently of the same structure. The bodies were of a golden-yellow color both in unstained sections and in those stained with aniline dyes, but they gave the prussian-blue reaction with potassium ferrocyanide and dilute hydrochloric acid.

The hilum lymph nodes were pigmented, but were only slightly enlarged, and no golden-yellow bodies were found in sections prepared from them. One at the bifurcation of the trachea was caseous and slightly calcified; no tubercle bacilli and no giant cells were found in the sections prepared from it. No evidence of tuberculosis was found in other organs and no golden-yellow bodies were seen in those examined microscopically (the liver, spleen and kidneys).

As suggested by Stewart (7) the fluid expressed from the lung was examined and found to contain golden-yellow bodies. With the aid of dark-ground illumination, Gloyne (3) observed that the fluid contained also free asbestos fibres and, in examining it, he noted that the yellow bodies when dissolved in sulphuric acid while under observation contained a core of asbestos fibres.

The autopsy of a final case was reported by Wood and Page (8) in January, 1930. In the summary of their case they state that the three features of special interest are (1) rapid evolution of the tuberculous process in a patient who had been exposed to asbestos dust; (2) the presence in the lungs of a large number of asbestosis bodies within two years of the first exposure to the dust; and (3) the presence in the lungs of asbestos fibres and the various stages of the formation of typical asbestosis bodies.

The essential features then of the pathological changes appear to be the pleurisy, the marked diffuse fibrosis and contraction of the lungs, and the presence of asbestos fibres and asbestosis bodies.

Regarding the presence of the asbestos fibre in the lesions of asbestos workers, Gloyne (3) in 1929 published an illuminating paper. Summarizing the description of raw asbestos, he stated that it may be ground into a fine powder, calcined in a crucible and mounted on a slide as a wet preparation with concentrated sulphuric acid without showing any signs of destruction. Examined by the dark-ground illumination method with a 1/6 in. objective, a film thus prepared shows bundles of fibres and individual fibres of varying lengths. The fibres are highly refractile, they are straight and rigid like steel wire and have often a very fine saw-like

edge, while the ends are broken at different angles and occasionally split. The finest of them have a bluish tinge.

With these appearances in mind Gloyne demonstrated that asbestos fibres can be readily removed from the so called asbestos corns with a fine needle and, when examined by dark-ground illumination, show the characteristic appearances of the asbestos fibres. He then demonstrated asbestos in the mouth and nose of two operatives by a method consisting of digestion of the material with antiformin, centrifugation of the digested material, calcining of the deposit in a crucible to destroy all cellular debris, and finally the making of a wet preparation of the calcined deposit in concentrated sulphuric acid and examination by dark-ground illumination. By the same method he found asbestos fibres in the sputum of three of six cases examined. Furthermore, typical asbestos fibres were found in the lung and were present in the expressed juice of the lung of a case available. Finally he made wet preparations of the golden-yellow bodies in expressed lung juice for examination by dark-ground illumination. Concentrated sulphuric acid was very gently run under the cover-slip. The yellow bodies began to dissolve and in the middle of their structure were a number of typical asbestos fibres. At the end of half an hour practically all the material of which the bodies were composed had disappeared, leaving only a faint ghostlike outline of the bodies with highly refractile asbestos fibres within them. The asbestos fibres lay at different angles. Gloyne concluded that, in any event, whatever other material enters into the composition of the bodies the basic factor is the asbestos fibre. and he suggested the term "asbestosis bodies" as being the most descriptive.

Stewart (7) in 1928 stated that in one autopsy of his own of a case of pulmonary asbestosis and in three by a colleague asbestosis bodies were readily demonstrated in large number by simple microscopic examination of lung juice expressed upon a slide and covered with a cover-glass.

Stewart and Haddow (10) in 1929 wrote a preliminary report on their efforts to obtain for diagnostic purposes asbestosis bodies by lung puncture and from sputum. From two cases clinically of pulmonary asbestosis they withdrew from the base of the lung through a needle a minute amount of blood-stained material. Unmistakable golden-yellow asbestosis bodies were found in considerable number in the material from one. In the second case negative results were obtained. Sputum is usually lacking or is in but small amount except during attacks of bronchitis.

From each of two cases, however, about a half-ounce of mucopurulent sputum was obtained. This was dissolved in an equal quantity of undiluted antiformin, centrifugated, and the deposit washed and examined microscopically. After prolonged search through numerous fields two typical asbestosis bodies were found in one of the specimens examined. The other was negative.

In an article published in January 1930, Gloyne (11) gave the results of animal experiments to show whether the asbestos fibre is definitely toxic or whether it acts merely as an inert foreign body or a mild sterile irritant. His conclusions were as follows: (1) asbestos fibres when injected into animal tissues acted as a benign irritant, producing granulation tissue; (2) this granulation tissue contained many asbestosis giant cells, presumably an attempt to destroy the asbestos fibres by phagocytosis; (3) connective tissue was formed in due course, but the giant cells persisted; (4) the asbestosis giant cell was readily distinguished from the true tuberculosis giant cell; (5) asbestosis bodies were not found; (6) asbestos injected repeatedly intravenously appeared to have no toxic effects on distant tissues.

CONCLUSIONS

1. A case is reported which seems to be typical of pulmonary asbestosis.
2. When there has been exposure to asbestos dust the presence of pulmonary asbestosis should always be suspected.
3. The most common single symptom is dyspnoea. This and the other symptoms are essentially those of a progressive generalized lung fibrosis.
4. The physical signs of uncomplicated pulmonary asbestosis are substantially those of generalized fibrosis of both lungs and basal pleurisy.
5. X-ray examination is of great value in establishing the diagnosis.
6. Asbestos contains but a very small amount of free silica but probably conduces to a more hasty evolution of any accompanying tuberculosis, as in the better understood forms of silicosis.
7. An immediate diagnosis at autopsy is said to be made possible by simply squeezing out upon a slide a drop of lung juice from the fibrosed tissue and covering with a cover-glass. The asbestosis bodies in large number are readily visible under the microscope.

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