PULMONARY ASBESTOSIS IN SOUTH AFRICA.

(*With Special Plate.*)

BY

F. W. SIMSON, M.B., CH.B.ED.,

PATHOLOGIST AT THE SOUTH AFRICAN INSTITUTE FOR MEDICAL RESEARCH, JOHANNESBURG.

It has been known for some time that workers exposed to the dusty atmosphere arising from some processes involved in the preparation of asbestos materials suffer from pulmonary disability. The mining of the mineral itself is probably not a source of danger, as asbestos is mined mostly in open quarries. After sorting, there remains waste rock which still contains fibre in payable quantity. Crushing of this rock causes a considerable degree of dust exposure, and exposure to the dusty atmosphere is still more exaggerated in the carding and spinning of asbestos in the mills.

Very little concerning the pathological changes in the lungs of persons working with asbestos has been found in the literature, and it was thought that a record of the following cases might be of interest.

On September 2nd, 1926, the medical officer of an asbestos mine in Southern Rhodesia forwarded to the South African Institute for Medical Research three small specimens of lung from a *post-mortem* case for histological examination.

CASE I (No. 9026).

The subject was a male adult native, who had worked for twelve months in the asbestos mill, and, except for a short period before his death, had had a good record of health. For nine weeks before death he suffered from acute tuberculosis, and the *post-mortem* findings showed miliary tuberculosis involving the lungs, liver, spleen, and pericardium. The object of the histological examination was to ascertain if there was any evidence of fibrosis in the lungs which might be directly ascribed to the nature of his employment, since workers in the mill are exposed to a very dusty atmosphere.

Apart from the tuberculosis and associated fibrosis, sections of the lungs showed a certain amount of connective tissue change which had no obvious connexion with the tuberculous process. It was also found that curious golden yellow segmented structures, with rounded or club-shaped ends (Fig. 1), were embedded in this fibrous tissue, together with very minute, doubly refractile particles, the latter presumably silica. Although the curious segmented bodies were remarked upon, no further investigation was carried out at the time, and the new-formed fibrous tissue was attributed to the presence of silica.

CASE II (No. 11016).

It was not until September 29th, 1927, when a portion of lung from a second case was sent by the medical officer from the same mill, that the foreign bodies and fibrosis were more fully investigated. This piece of lung was from a male adult native who had been employed in the mill for a period of two years. About September, 1926, he was admitted to hospital suffering from pneumonia, and had a long illness. He was discharged, and later (September 19th, 1927) readmitted in a dying condition. He was very emaciated, and had the physical signs of pulmonary tuberculosis with fibrosis. At autopsy the lungs were firmly bound down by adhesions, the root glands enlarged and hard, and the lungs on section hard and fibrous with an almost leather-like consistence. The mesenteric glands were also considerably enlarged.

Histological sections showed a generalized but moderate degree of fibrous thickening of the pleura, trabeculae, and alveolar walls. In addition, there was a much more marked fibrosis arranged in irregular-shaped nodules, chiefly related to the blood vascular system and bronchi. This fibrous tissue was cellular, the elements arranged in an irregular manner, and included some small lymphocytic accumulations (Fig. 2). There was no resemblance to the orderly, whorled arrangement and sharp definition of the silicotic nodule (Fig. 3). The bronchi were the seat of slight catarrhal changes, and many of the alveoli contained "dust cells" filled with phagocyted particles. There was no evidence of tuberculosis or acute pneumonic consolidation. Embedded in the fibrous tissues, lying free in alveoli (Fig. 4), and contained in phagocytic cells, there were numerous golden yellow segmented bodies (Figs. 5 and 6), a few large angular black bodies, and very minute doubly refracting particles. Phagocytosis was a very striking feature. Large numbers of the bodies were contained within ordinary mononuclear cells and irregular-shaped multinucleated giant cells (Fig. 7). Phagocytosis was not confined to the smaller particles, but even large rods were seen completely enclosed and often bent in order to allow them to occupy the space within the cells (Fig. 7). It was thought that these foreign bodies, together with the crystalline matter, may have been the cause of the fibrosis, as the connective tissue, both in distribution and formation, was very suggestive of changes resulting from a dust occupation of the lungs. At the same time the history of pneumonia and protracted recovery must be taken into consideration, since an unresolved pneumonia with subsequent fibrosis is not an uncommon occurrence amongst natives working on the mines in South Africa.

CASES III AND IV (Nos. 6419 and 6731).

Consultation of the records and previous histological sections of material from the same mine has revealed two additional cases, both of which showed lobar pneumonia. There was very little connective tissue increase, but the unusual structures (Fig. 8) and refractile crystalline particles were present. No history of length of service in the mill was obtained with either of these cases.

Further study of the golden yellow bodies showed a variety of shapes, but the most common forms had rounded or club-shaped ends and a segmented body tapering to a finely pointed tail. They were non-refractile to polarized light, soluble in strong acids, and, on raising to a red heat, turned black and tended to lose their outline. In sections treated with hot dilute hydrochloric acid and potassium ferrocyanide they gave a well-marked Prussian blue reaction. No pigment except these structures giving the iron reaction could be detected in the sections examined. Strong hydrochloric acid, having been tested for the presence of iron and found negative, was used to dissolve out iron from fresh sections. After treating the sections the hydrochloric acid gave a very distinct red-pink colour with potassium thiocyanate, and the red-pink colour disappeared on the addition of a solution of mercuric chloride. The sections were re-examined and showed that the majority of the golden yellow bodies had been dissolved out. From these tests it was concluded that the structures contained a large percentage of iron.

As controls, sections of lungs from a large number of miners on the Rand who had died from silicosis and tuberculosis were examined. Bodies such as have been described above were found in none of these, nor was the Prussian blue reaction or other test for iron positive except in those cases where the pigment was obviously of haematogenous origin. A single case of a miner was also investigated; he had worked for twenty-eight years in the haematite mines in the North of England, and subsequently for seven and a quarter years in the gold mines of the Rand, South Africa. The cause of death was carcinoma of the gall-bladder. Macroscopically the pulmonary root glands were enlarged, pigmented, and fibrosed. The pigment was dark rust-coloured and gave a marked Prussian blue reaction with dilute hydrochloric acid and potassium ferrocyanide. Occasional rust-coloured subpleural islets were visible. On section the lungs showed a slight diffuse fibrosis and a few large areas of fibrosis. The whole lung gave a marked iron reaction. Histological sections of the large areas showed moderately well defined, but irregularly shaped, masses of well-formed acellular fibrous tissue (Fig. 9). The alveolar walls were slightly thickened, and there was a moderate degree of connective tissue increase round the blood vessels and bronchi. A large quantity of pigment of a reddish-brown colour was contained in phagocytic cells and lying free in alveoli, alveolar walls, and between the fibres of the newly formed fibrous tissue. The greater part of this pigment gave the iron reaction. In addition, there was another variety of pigment, in much smaller quantity, in the form of crystalline refractile particles. The latter was intimately mixed with the iron-containing dust. This lung was especially examined to determine, if possible, whether the same peculiar bodies were being formed from a deposit of ferric iron dust as in the case of an asbestos dust which contained both ferrous and ferric iron. A careful and thorough search was made, but no particles with a similar appearance were detected (Fig. 10). At the time of the patient's death this case of ironstone phthisis was complicated by underground work in the gold mines on the Rand, but in 1919 it simulated, by x-ray photography and physical examination, an early case of silicosis.

and confirms the findings of Sir Kenneth Goadby[1] and Dr. Cronin.[2]

In addition to the human lungs showing asbestosis, Dr. Mavrogordato of the South African Institute for Medical Research has supplied me with the lungs of a guinea-pig which died in December, 1927, from causes other than asbestosis. This animal was exposed to an asbestos dust atmosphere experimentally. The length of exposure was two hours a day on each of fifty days. The first exposure took place on February 4th, 1925, and the last on April 1st of the same year. The asbestiform compound used for the experiments was a chrysotile[3] obtained from the mine in Southern Rhodesia. Histological sections showed a slight generalized fibrosis and an increase in pigment, but the interesting feature was the presence of the golden yellow bodies (Fig. 11), similar to those seen in the lungs of human pulmonary asbestosis.

A comparison between the human cases and the experimental animal showed that the fibrosis was more rapid and extensive in the human cases than in the experimental animal. This is readily explained by the difficulty in reducing the tough asbestos fibre to a uniformly fine powder, and to the presence of a comparatively small proportion of the rock dust which is usually associated with asbestiform compounds. For "dusting" animals a limited amount of dust is available, and of this only a small proportion contains particles of sufficiently small dimensions to permit of their reaching the lung alveoli. In silicosis[4] the size of the majority of the particles[5] which reach the alveoli is between 1μ and 3μ; in the haematite lung, mentioned above, the particles appear larger, but even here are much smaller than the greater number of those prepared from asbestos for "dusting" experiments. In the working mill the conditions are very different from an asbestos dust atmosphere produced experimentally. Fine dust is continuously reaching the atmosphere, and only the very fine particles remain suspended for any length of time. These gradually increase in numbers until a maximum concentration is reached, then remain more or less constant during working hours. Thus it will be seen that, in order to produce changes in experimental animals showing the same degree of fibrosis in the same length of time, an atmosphere approximating that of the working mill will have to be obtained.

The amount of fibrosis in two of the human cases (Cases I and II) was quite definite, and, if due to the presence of asbestos dust, the initial rate of production was rapid when compared with present-day non-infective silicosis on the Rand. It is difficult to state a definite time for the production of an appreciable degree of fibrosis in pure non-infective silicosis, but modern observation tends to show that it is in the neighbourhood of ten years. In Case I a moderately marked fibrosis had taken place after one year of work in the mill, but this was complicated by tuberculosis. It is known that the rate of fibrous tissue production is very much greater in dust diseases complicated by infections, but even allowing for this the connective tissue increase in Case I was rapid. In Case II there was a still more definite fibrosis after two years of work in the mill with no evidence of tuberculosis.

Before this work was completed Drs. Cooke,[6] Stuart McDonald, and Oliver[7] published their papers dealing with pulmonary asbestosis, and previous to that a short article on the same subject appeared in the *Lancet*. The similarity between the case described and ours was suspected when the first article appeared, and our work goes far to confirm the findings. As in those recorded here, the asbestos dust responsible for the changes in the lungs in their case was a chrysotile. Whether other asbestiform compounds are capable of producing these changes it is impossible to say, as there appears to be no record of any such cases in the literature.

In the Union of South Africa[8] and Rhodesia there are many asbestos mines, from some of which chrysotile is obtained, but two other interesting varieties are found—namely, crocidolite and amosite. Amosite is a comparatively recent discovery, the chemistry of which has not been completely worked out, but it appears to be somewhat similar to crocidolite in composition. Both these compounds contain a large percentage of ferrous iron. Analysis of four samples of amosite[8] showed between 32 and 44 per cent. of FeO, and eight samples of crocidolite[8] between 16.5 and 40.5 per cent. Up to the present there has been no examination of *post-mortem* material from cases of death among those working in mills where these minerals are treated. No such material has been made available.

Chrysotile or serpentine asbestos usually contains 2 to 3 per cent. of FeO isomorphously replacing magnesia. The analysis of Dr. Cooke's case showed 3 per cent. of FeO, and the Rhodesian mineral, according to Mr. A. L. Hall,[3] 2.44 per cent. Dr. McCrae of the Government Chemical Laboratory, Johannesburg, analysed the FeO content of the asbestos used by Dr. Mavrogordato in his animal experiments, and found a much lower percentage—namely, 0.45. Apparently even with very small percentages of FeO the golden yellow bodies are formed in the lungs.

With regard to the unusual structure and chemical nature of the golden yellow bodies found in the lungs in pulmonary asbestosis, until many more cases have been examined and more experimental work has been done very little can be stated. There is no doubt that they are in some way associated with the asbestos, or, probably, more particularly with the FeO content, either of the asbestos itself or of the dust of the mill. This dust contains a much higher percentage of iron, which may be derived partly from the lode from which the asbestos is mined.

Three possibilities regarding the formation of the golden yellow bodies are worthy of mention:

(1) In the form of a gel, as suggested by Dr. Stuart McDonald.

(2) As particles of ferruginous quartz formed in the lungs under conditions similar to weathering, or ferruginous quartz changed in composition as a result of combination with constituents of the body fluids. In support of this it may be mentioned that crocidolite, which is an alkali silicate with ferrous iron, is especially liable to decomposition when exposed to weathering. Sodium is removed, the iron oxidized and hydrated to form limonite, and silica set free; there then results a ferruginous quartz which possesses the finely fibrous structure of the original mineral. It is extremely hard, and coloured a rich golden yellow. It is possible that a change such as this occurs with the small quantity of ferrous iron associated with chrysotile, and the colour of the bodies in the lungs is very suggestive.

(3) Phagocytosis of these structures is a very prominent feature in the lungs, and it was thought that the action of these cells may have been responsible, first, for some change in chemical composition, then a building up and moulding into the various shapes seen.

The fibrous tissue change in the lungs of the above-mentioned cases of workers in asbestos mills is probably the result of a reaction on the parts of the tissues to both the golden yellow bodies and a small quantity of silica.

Sufficient cases of pulmonary asbestosis have not been recorded to base an opinion upon the liability to secondary infection by specific inflammatory processes such as tuberculosis, but it is very suggestive that two of those now recorded, which have been examined histologically, have shown tuberculosis complicating the changes produced by an asbestos dust occupation of the lungs.

Besides these cases there is still further evidence in favour of tuberculosis being a complicating factor.

Dr. H. M. Murray reported a fatal case in the *Charing Cross Hospital Gazette* in 1900; and in the United States of America, from one source,[9] during the period 1907 to 1914 there were 13 deaths, 3 of which were from tuberculosis.

In 1910 Dr. Collis[10] reported on the relationship of asbestos dust to pulmonary tuberculosis, and found that 5 deaths from phthisis had occurred in five years amongst a staff of less than forty workers employed at a factory where asbestos is woven.

In conclusion, I wish to record my thanks to Dr. Mavrogordato for data and material from an experimental animal; to Dr. Irvine, chairman of the Miners' Phthisis Medical Bureau, Johannesburg, for material from silicotic cases and from the lungs of a haematite miner; to Dr. McCrae for his analysis of the FeO content of a sample of asbestos; and to Mr. P. Longmore for his valuable assistance in preparing the microphotographs.

FIG. 1.—Pulmonary asbestosis (Case I). Golden yellow bodies lying in the centre of a nodule of young fibrous tissue. (× 850.)

FIG. 2.—Pulmonary asbestosis (Case II). Irregular fibrotic nodule showing a moderate degree of cellularity. The bodies embedded in this tissue are just visible. (× 250.)

FIG. 3.—Silicosis. Nodule showing orderly whorled arrangement and sharp definition characteristic of this type of fibrosis. (× 250.)

FIG. 4.—Pulmonary asbestosis (Case II). Small phagocytes containing granular dust and golden yellow bodies. One large body is lying free in the alveolus. (× 850.)

FIG. 5.—Pulmonary asbestosis (Case II). Large golden yellow structures with globular ends and segmented body tapering towards one end. (× 850.)

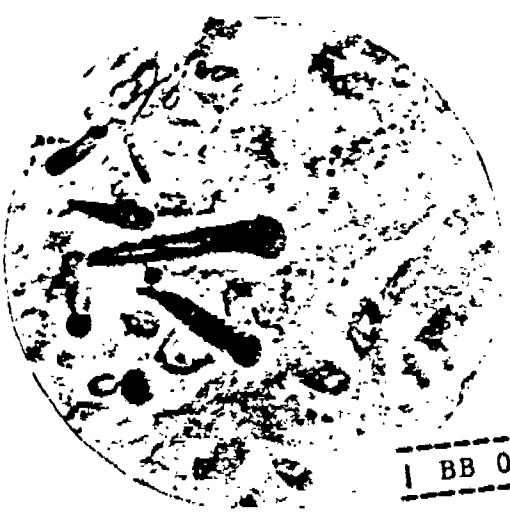

FIG. 6.—Pulmonary asbestosis (Case II). Large golden yellow structures with globular ends and segmented body tapering towards one end. (× 850.)

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ABDOMINAL PAIN AS EXEMPLIFIED IN ACUTE APPENDICITIS:

A CLINICAL AND BIOLOGICAL CONSIDERATION.*

BY

JOHN MORLEY, CH.M., F.R.C.S.,

HONORARY ASSISTANT SURGEON, MANCHESTER ROYAL INFIRMARY; HONORARY CONSULTING SURGEON, ANCOATS HOSPITAL; LECTURER IN APPLIED ANATOMY, MANCHESTER UNIVERSITY.

THE EVOLUTION OF OUR KNOWLEDGE OF ABDOMINAL PAIN.†

ALMOST the first considerable contribution to our knowledge of the mechanism of abdominal pain was made by J. Ross of Manchester[1] in 1887. Ross held that there were two kinds of pain in visceral disease—true splanchnic pain, felt in the affected organ; and associated somatic pain, referred to the cerebro-spinal nerves of the body wall. Lennander[2] established the insensibility of the exposed gastro-intestinal tract to the ordinary painful stimuli, and attributed all abdominal pain to stimulation of nerves in the sensitive parietal peritoneum. Mackenzie, whose views form the orthodox teaching of the current textbooks, accepted Ross's views on somatic pain, but rejected splanchnic pain as non-existent. He believed that painful stimuli passed from the viscera through the afferent splanchnic nerves to the spinal cord, but were only appreciated by the brain as arising from the sensory nerves of the parietes. Mackenzie's theories of a viscero-sensory and viscero-motor reflex have been widely accepted, and will be discussed later. Hurst[3] in 1911 published observations which have restored our belief in true splanchnic pain; he proved that this type of pain is produced by a single adequate stimulus—namely, increased tension in the muscular wall of the viscus concerned. Yet Hurst does not reject Mackenzie's views on the viscero-sensory and viscero-motor reflex mechanism.

My purpose in this paper is to discuss the light which a study of acute appendicitis throws on the mechanism of abdominal pain; and since Mackenzie's theories were based very largely on his observations in cases of appendicitis, this disease may reasonably be used to test those theories.

THE TWO PAINS IN APPENDICITIS.

The conventional teaching on the pain in acute appendicitis is that the attack is ushered in by pain referred to the region of the umbilicus or a little above it, and that after a few hours the pain becomes localized in the right iliac fossa or moves down into that region.

The Initial or Central Pain.

The first point that I wish to emphasize is that the initial pain is entirely different in character and in its mode of origin from the pain which appears in the right iliac fossa a few hours later. The initial pain is felt in the centre of the abdomen, and though the patient may refer it to the mid-line at or above the umbilicus, he often describes it as "all across," sweeping his hand evenly across the central region of the abdomen. It is, in short, very imperfectly localized. The pain is often described by the patient as "like an ordinary bellyache, but more severe." It is frequently griping in character and varies in intensity; the more severe spasms usually occur at more or less regular intervals, and last for a few seconds, during which the patient moans and writhes restlessly in bed. This early pain is entirely unassociated with any tenderness on palpation, and the patient may rub or press on his abdomen in a vain attempt to secure relief—a thing that he never does when the second pain has appeared.

This initial pain in appendicitis is, I believe, a true splanchnic pain, and, like all true splanchnic pain, is due to increased tension on the muscular wall of the viscus concerned. Some degree of obstruction to the lumen of the appendix, causing retention of inflammatory exudate distal to the obstruction, is an essential factor in its production. Sometimes—in fact, it is almost the rule in the most fulminating cases—we find a hard, laminated faecal concretion engaged in the relatively narrow base of the appendix, while distal to it a collection of foul pus under great tension rams the concretion home into the base. In this condition, so well described by Wilkie[4] as acute appendicular obstruction, the central pain in the umbilical region continues until general gangrene or localized perforation of the appendix relieves the tension within it and the pain disappears. In other cases a stricture at the base, the result of some former ulceration, gives rise to the same sequence of events. In others a kinking at the base combines with inflammatory swelling of the mucosa to occlude the outlet into the caecum. In yet rarer instances a foreign body is the occluding agent. A critical examination of the specimens removed in a large number of cases of early appendicitis has convinced me that wherever this splanchnic pain is present some degree of obstruction to the lumen, with retention of inflammatory exudate under tension beyond it, will be found. It is possible that the inflamed condition of the appendix lowers the threshold of painful stimulation and allows a relatively slight distension to cause pain; but without increased tension visceral pain is impossible.

Under certain conditions recurrent attacks of appendicitis may occur in which this splanchnic central pain is the only evidence of the disease, and great difficulty in diagnosis results. A striking instance of this difficulty was afforded by a man, aged 21, who gave a history of four severe attacks of epigastric pain, each lasting for a few hours, during the course of four months. There had been neither tenderness nor muscular rigidity during the attacks. Exploration a week after the last attack showed that the only viscus affected was the appendix, which was swollen and congested in its distal portion. There was a narrow stricture at the base, and, distal to this, a collection of thick yellow pus. In the ten months that have passed since the operation there have been no more attacks.

It is evident that this patient was suffering from recurrent attacks of acute obstructive appendicitis of too mild a type to cause perforation, and that his pain was of the pure splanchnic appendicular type. The reason for the entire absence of local pain and rigidity I shall discuss later, when dealing with the second pain. The brief intermittent colicky attacks of central abdominal pain so common in children, and described by physicians as "umbilical colic," are very commonly due to spasmodic efforts to expel a concretion or a nest of threadworms from the appendix, and form another example of pure splanchnic pain.

Although the splanchnic pain in acute appendicitis is felt in the umbilical or lower epigastric region, and not in the region of the appendix, it should not be described as a referred or reflected pain, as no radiation of pain or reflex process is involved. The appendix is developmentally a part of the mid-gut loop. The brain can only appreciate painful stimuli arising from any portion of the mid-gut as vaguely situated in the centre of the abdomen, and stimuli of appendicular origin are no exception to the rule. It is characteristic of this splanchnic pain that it is dull and aching in character—as patients express it, "like an ordinary bellyache"—and vaguely localized; in other words, it is a form of Head's protopathic pain.

*A post-graduate lecture delivered at the Manchester Royal Infirmary.
† For reasons of space this introductory section has been abridged.

In the present case no involvement of the medulla was found. Extensive fibrous meningitis resembling gummatous basilar meningitis has also been described by Askanazy,[11] Rosenblath,[12] Henneberg,[10] and others. As far as is known, the cysts are very little toxic, although it appears that pyrexia occurred at the onset of infection or early in the course of a case of disseminated cysticercosis described by Major R. Priest[13] in 1926.

The case we record proved fatal after nearly six years of symptoms; but the parasites, especially when localized in the eye or brain, may live for long periods, very rare cases being noted up to twenty years (Küchenmeister, Roth-Ivanoff, Pfeifer, and others).

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A CASE OF PNEUMOCONIOSIS.

RESULT OF THE INHALATION OF ASBESTOS DUST.

BY

H. E. SEILER, M.B., CH.B., D.P.H.CAMB.,

ASSISTANT MEDICAL OFFICER, PUBLIC HEALTH DEPARTMENT, GLASGOW.

(*With Special Plate.*)

THE asbestos industry has been in existence for over two thousand years, and it is remarkable that the possibility of damage to the lungs, arising from the inhalation of dust during its manufacturing process, has not been investigated until within comparatively recent years. Very few cases of pneumoconiosis due to asbestos dust have been recorded in medical literature, and for this reason the notes on the following case may be of some interest.

The first recorded case was that reported by the late Dr. Montague Murray of Charing Cross Hospital in 1900. No other case was reported, and the inference that asbestos dust might be the cause of an extensive pulmonary fibrosis was lost sight of for some time. In 1924[1] and 1927[2] Dr. W. E. Cooke published his exhaustive reports on another case, and described in detail the pathological changes and his "curious bodies," which are apparently a unique feature of the microscopical picture. This stimulated other investigations, and Simson[3] has reported similar findings in workers employed in the crushing of asbestos rock as apart from the manufacturing processes. In most of the cases hitherto reported the extensive fibrosis has been associated either with pulmonary tuberculosis or an unresolved pneumonia, and there has always been the slight doubt as to whether the inhalation of asbestos dust was the primary cause of the fibrosis.

The patient, a man aged 40 years, was sent for an opinion of his chest condition to the tuberculosis dispensary. The question of tuberculosis was raised as a consequence of his complaint of cough and breathlessness, combined with loss of weight and lassitude, extending over a period of several months.

Previous History.—There was no history of tuberculosis in the family. No history could be elicited of any illness in early life which might have led to a pulmonary fibrosis, and, apart from the symptoms which he considered to be associated with his occupation as an asbestos worker, he has had no other illnesses.

Occupation.—When a boy he had worked for a very short period in a glass works. Thereafter, for a period of twenty-two years, he has been associated with the asbestos industry. During this period he has been employed in the various processes of asbestos work: for four and a half years he worked at a carding machine, which is considered the dustiest part of the work; for seven and a half years at the weaving of asbestos cloth, and for the remainder of the period at the manufacture of the millboard. The various processes differ considerably in their degree of dustiness, and therefore in the amount of asbestos dust inhaled by the various types of workers. As, however, a number of different processes, evolving a varying amount of dust, may be carried out in the same room, the dust produced at any one process may affect workers at another process. It will be noted that this man must have worked in the asbestos industry for a period during which less importance was paid to the prevention of dust than at the present time.

History of Present Illness.—The patient has had a cough from within a few months of starting work at asbestos, but he states that this was "from the throat," and appeared to have been purely the result of dust irritation. Within recent years the cough has increased in severity, and there has been a little sputum first thing in the morning. The first symptom to cause anxiety was breathlessness, which developed five years ago, and this has become accentuated since then. His general condition has remained satisfactory, and his particular symptoms were not sufficiently severe to interfere with his employment until a few months ago. At this time he complained of lassitude, loss of weight, frequent night sweatings, and an occasional slight aching of the left shoulder. On no occasion has there been haemoptysis.

Notes on Examination.—He is a spare-built man; weight 7 st. 11 lb.; temperature, pulse, and respirations within normal limits. No obvious dyspnoea noted on examination. Cyanosis of lips and cheeks, but not a marked feature. Well-marked finger clubbing noticed by the patient for at least two years. Sputum scanty, and of a mucoid nature. Tubercle bacillus absent on frequent examination.

Examination of Chest.—Inspection showed expansion to be poor generally (one inch by measurement); there was hypertrophy of the extraordinary muscles of respiration; the supraclavicular fossae were markedly hollowed; the heart apex beat was in the fifth intercostal space within the nipple line. The percussion note was flattened throughout; there was relative dullness over the right upper lobe, both in front and behind. On auscultation the respiratory murmur was diminished generally, and of a harsh quality over the right upper lobe anteriorly and posteriorly; expiration was prolonged all over the chest; rhonchi were general, with fine pleural friction at both bases.

Other Systems.—Examination of other systems was negative.

X-Ray Examination.—The radiogram of the chest shows a fine mottling of a silicotic type throughout both lungs, more marked on the right side and at both bases, with indication of a dense fibrosis, especially on the right side. (See special plate.)

Progress.—The patient was admitted to hospital for observation, and has now been an in-patient for six months. During this period his general condition has improved considerably, and he has gained over 1 st. in weight. The cyanosis which had been noted on first examination has to a great extent disappeared, but there is still some evidence on the cheeks and lips. The cough, which is now manifestly less troublesome, is still present, and there is a scanty mucoid sputum. Physical examination of the chest reveals no new feature apart from the absence of bronchitic sounds. The temperature and pulse remained within normal limits during the period of observation and treatment in hospital.

Conclusion.

There seems little doubt that this is a definite case of pulmonary fibrosis, the result of the inhalation of asbestos dust. The recent development of symptoms of toxaemia, associated with long-standing pulmonary symptoms, was at first suggestive of a chronic pulmonary tuberculosis with an acute exacerbation. However, the physical signs, the persistent absence of the tubercle bacillus in the sputum, and the maintenance of temperature and pulse within normal limits demanded further investigation. The typical radiographic features (signs more marked in the basal portions of the lungs) and his long period of employment with asbestos point definitely in the direction of a fibrosis of occupational origin, especially as there is neither a history of other illnesses in childhood or adolescence to support a more common cause of pulmonary fibrosis, nor does this correlation of history, symptoms, and physical signs occur in these latter cases. Toxaemic symptoms in the absence of a tuberculous affection are of special interest; whether they are the result of the absorption of some constituent of the asbestos rock or not is a point which requires further investigation.

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[1] Cooke, W. E.: Fibrosis of the Lungs due to the Inhalation of Asbestos Dust, *British Medical Journal*, 1924, ii, 147.

[2] Cooke, W. E., Stuart McDonald, and Sir T. Oliver: Pulmonary Asbestosis, *British Medical Journal*, 1927, ii, 1024.

[3] Simson, F. W.: Pulmonary Asbestosis in South Africa, *British Medical Journal*, 1928, i, 885.

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sunshine there each day for three years, recorded by the month, ranged from 1.9 for December, the lowest figure, to 9.5 in July, the highest. The average percentage of maximum sunshine from 11 a. m. to 1 p. m. —the period during which infants can be most conveniently exposed—showed a similar increase from December to July. The sun's rays during December, January and February in the latitude of Toronto produce a slight but definite antirachitic effect on rats fed on a rachitogenic diet. A sharp increase occurs in the antirachitic effect of sunshine about the first of March. The antirachitic effect of sunshine in April and May is approximately eight times as great as in December, January and February. It would appear that the increase in the antirachitic effect of sunshine in March, April and May is due not so much to an increase in the amount of the ultraviolet rays of the length present in the sunshine in winter as to the presence of rays that are shorter than those found during the winter. The antirachitic effect of skyshine (reflected rays from the sky and clouds) is approximately from one half to two thirds as great as that produced by what is ordinarily termed sunshine (rays from the sun plus the reflected rays from the sky).[4]

Ordinary window glass is impervious to the antirachitic rays of sunshine. Consequently much effort has been devoted of late by glass manufacturers to the production of glasses that will transmit some of the rays in the region of the ultraviolet. Reports on some of the available products have already been published in THE JOURNAL.[5] Those now marketed are only partly pervious. The studies of Tisdall and Brown[6] on rachitic animals show anew that no antirachitic effect, or at the most a negligible effect, is produced by the sun's rays through ordinary glass. The antirachitic effect of skyshine through the special glasses is slight—in fact, almost negligible—except immediately adjacent to the window. Similar results were obtained with rays through an open window covered with ordinary fly screen. In order to obtain much benefit from rays through an open window or a window glazed with special glass it is necessary to receive the direct rays of the sun. Tisdall and Brown believe that the use of special glass during the winter months is probably of little value. Its use in the latitude of Toronto is justified from about the first of March on, as the inclement spring weather prohibits the exposure of patients to the sun's rays, which at that time have a great antirachitic effect. By means of this glass, these antirachitic rays can be utilized.

4. Tisdall, F. F., and Brown, Alan: Antirachitic Effect of Skyshine, Am. J. Dis. Child. 34: 737 (Nov.) 1927.
5. Report on Window Glass Substitutes, J. A. M. A. 88: 1563 (May 14) 1927. Sheard, Charles: Ultraviolet Radiation and Its Transmission by Various Substances, ibid. 88: 1315 (April 23) 1927. Henderson, H. N.; Lemon, H. B.; Falk, I. S., and Coade, E. N.: Ultraviolet Radiation from Sunlight and Incandescent Lamps, ibid. 89: 187 (July 16) 1927.
6. Tisdall, F. F., and Brown, Alan: Antirachitic Value of the Sun's Rays Through Various Special Window Glasses, Am. J. Dis. Child. 34: 742 (Nov.) 1927.

PULMONARY ASBESTOSIS

Apparently the first case of pulmonary asbestosis reported was in 1924. W. E. Cooke[1] has just made available the record of a woman who began to cough soon after she started work in an asbestos factory at the age of 13 years. Her general health did not suffer until she was 26. Five years later she was totally incapacitated and she died at 33. Her complaints had been cough, dyspnea, expectoration and lassitude, and the physical signs those of fibrosis and cavitation of the lungs. Postmortem examination of the patient's lungs[2] revealed diffuse interstitial pneumonia, chronic bronchitis, some empyema, anthracosis and extensive tuberculosis. Certain foreign bodies were unique. They were found in the alveoli, bronchi, bronchioles and interstitial fibrotic areas and apparently were neither of vegetable nor of animal origin. The hypothesis advanced is that these bodies are portions of asbestos fibers in the process of alteration and absorption by hydrolysis, either by direct chemical action or by enzymes. The question remains as to whether the fibrosis was the result more of tuberculous infection or of the presence of asbestos. Considerable fibrosis, however, has been found in one case without apparent tuberculosis.

Industrial dusts have been the objects of widespread research. In view of the fact that asbestos has been in use since five centuries or more before the time of Christ, it seems strange that it has not attracted attention. Asbestos is a silicate and is associated with other substances in the proportions given by Cooke as follows:

	Italian Fiber	Canadian Chrysotile
Silica	40.30	40.27
Magnesia	43.37	41.50
Ferrous oxide	0.87	2.81
Alumina	2.27	0.90
Water	13.72	13.55

Microscopically, asbestos consists of translucent glistening fibers, on the one hand; opaque angular particles and minute black granules, on the other. In the process of manufacture, much dust is generated, particularly in carding, and this dust is made up of the angular particles and black granules mentioned. Few of them appear in the finished product. Analysis shows:

	Per Cent
Finished article: Iron (as ferrous oxide)	0.1
Crude raw material: Iron (as ferrous oxide)	2.81
Dust from carding room: Iron (as ferrous oxide)	18.4

"From these results it appears conclusive that the blackened brittle parts of the asbestos fiber are the iron-containing portions—the bugbear of the manufacturer, the cause of 'dust' and a danger to the health of workers in the process of manufacture."

Although Cooke's case is only the second reported, many other cases seem to have occurred. One patient, for instance, who had worked in a carding room for ten years bef re his admission to a hospital said that

1. Cooke, W. E.: Pulmonary Asbestosis, Brit. M. J. 2: 1024 (Dec. 3) 1927.
2. McDonald, Stuart: Histology of Pulmonary Asbestosis, Brit. M. J. 2: 1025 (Dec. 3) 1927.

he was the only survivor of ten men who had started work with him. Conditions are better now, for modern factories are equipped with devices for removing the dust. Nevertheless, asbestosis, because f its dangers and its unique pathologic features, deserves more attention than it has had.

FOREIGN OBJECTS IN THE CIRCULATION

The protective devices at the command of the body are remarkable and varied. They help to avert disaster and sometimes death under conditions that would tax the resources of the most ingenious physician to the utmost if his assistance alone were depended on to devise relief. Some of the mechanisms on which the organism depends involve chemical reactions. Thus, acids and alkalis tend to become neutralized and consequently less harmful. Toxic organic products, whether introduced from without or produced endogenously, may be oxidized, conjugated or otherwise altered chemically so that the resulting compounds are more readily tolerated and eliminated. Some substances, like certain of the heavy elements, may become innocuous through deposition in tissues. The outcome in all these typical instances tends to result in diverting danger.

There are other protective reactions that depend almost entirely on mechanical effects. This applies to the disposal of foreign materials, particularly such as are chemically inert and insoluble in the body fluids. Metallic pieces somehow manage for the most part to traverse the alimentary canal without serious injury to it. The swallowed open safety pin has given many a mother well justified worries until it emerged without detriment to the unsuspecting infant that ingested it. Accidents occasionally introduce metallic particles not only into the soft tissues but even into the blood stream. More than one bullet or projectile fragment has entered a large vein and gradually proceeded along the path of the vessels. Where does it go?

A review of the literature by Warthen[1] has left him with the impression that wandering foreign bodies in the venous circulation are carried by the blood stream to the heart, where they are kept in motion by the churning action of the heart or become entangled in the chordae tendineae of the right ventricle and gradually embedded in the endocardium. No mention is made of foreign bodies reaching the lungs. In his own studies at St. Elizabeth's Hospital, Richmond, Va., to determine the course taken by foreign bodies in the venous circulation and their ultimate lodging place, objects of various sizes and shapes were inserted in the veins of a series of animals. These investigations showed clearly that the foreign objects within the lumen of a normal vein of a dog are carried by the blood stream toward the heart. This migration may take hours or days, but eventually these wandering bodies reach the heart. It was surprising t learn further, however, that the majority of these objects do not remain in the heart but are pumped into the pulmonary artery and lodge in the lungs, where they produce infarcts with apparently little disturbance to the pulmonary circulation. The presence of these foreign bodies within the lungs appears to cause little discomfort. Abscess of the lungs rarely occurred. Apparently sterilized foreign objects in the venous circulation do little harm to the heart or lungs, and unsterilized objects which are smooth and symmetrical rarely cause infection. Unsterilized, irregularly shaped bodies, as nails or fragments of nails, are most dangerous and give rise to abscesses.

It will come as a surprise to learn from Warthen that it is possible for objects to pass from the venous to the arterial circulation, this transition probably occurring in the lungs, without leaving any macroscopic evidence of pulmonary injury. He notes, as others have observed, that the smaller, more symmetrical and lighter objects in the venous circulation tend to remain in the heart, while the larger, more irregular and heavier bodies are carried into the lungs. Foreign bodies that remain in the heart appear to lodge near the apex of the right ventricle at the base of the chordae tendineae. As this is the most accessible region of the heart in man, the newly developed possibilities of intracardiac surgery may be requisitioned whenever foreign objects thus lodged demand removal.

THE DANGERS OF ULTRAVIOLET RAYS

The tendency of therapeutic methods to be adopted and discarded in great waves of popularity and forgetfulness has never been so completely manifested as in the current attention given to the use of physical therapeutic methods, particularly the ultraviolet ray. Not only do barber shops swindle prospective victims of baldness with incandescent lamps colored purple, not only do electrical corporations sell, as ultraviolet ray devices, contraptions delivering hardly any ultraviolet radiation at all, but some manufacturers of apparatus actually delivering ultraviolet rays of potency endeavor to place these devices wherever a sale can possibly be made. Regardless of the fact that practically every method in medicine that may do good can also do harm, these machines are being sold to bath institutes, swimming pools, massage parlors, beauty parlors, clubs, barber shops and innumerable other businesses in which medical supervision is certainly not probable, indeed, hardly possible. Th sales are made notwithstanding the fact that scientific literature has already revealed that the rays may in some instances depigmentize or bleach the skin, that they possess dangerous effects for the eyes, that some people who do not tan

1. Warthen, H. J.: Fate of Foreign Bodies in Venous Circulation, Arch. Surg. 15:712 (Nov.) 1927.

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This is but an example of the matters which remain to be dealt with. They are details, and only details by comparison with the arrival of a great central scheme, but there will be room in their adjustment for the display of adequate leadership, and it is not too soon to give them consideration.

PULMONARY ASBESTOSIS.

THE introduction of that extremely useful substance asbestos into industry has unfortunately given rise to a new occupational risk and to yet another form of chronic pulmonary fibrosis. The material has in one form or another been known since the days of Greek and Roman civilisation—the ancient writers refer to it as "unquenchable stone"—and CHARLEMAGNE is said to have had a table-cloth made of it, which was cleansed by throwing it in the fire. In earlier days the hornblende variety of asbestos was often referred to as amianthus. Sir THOMAS BROWNE, the author of Religio Medici, in one of his poems sighs that he might have amiantine toes which would not suffer from the gout. The indestructibility of asbestos has no doubt been the primary reason for its introduction into trade processes; asbestos will resist great heat, strong acids, and alkalis, whilst owing to its fibrous structure certain varieties of it can be carded and spun like a textile. A report[1] just published by the Home Office deals with the effects of asbestos dust on the lungs and on the dust suppression in the asbestos industry, and is written by Dr. E. R. A. MEREWETHER and Mr. C. W. PRICE, respectively medical and engineering inspector of factories. Dr. MEREWETHER'S report is based upon data obtained in an extensive investigation made during 1928 and 1929, initiated by the Home Office, following the discovery of non-tuberculous fibrosis in the lungs of an asbestos worker, of sufficient severity to necessitate treatment in hospital. Prior to this the Home Office had knowledge of only two deaths in which the inhalation of asbestos dust was a contributory cause; a survey of the industry in 1910–11 had not revealed any serious hazard. The well-known triad—exposure to dust, fibrosis of the lungs, and high mortality-rate from pulmonary tuberculosis—has long been recognised in industrial processes involving the use of free crystalline silica, but, as Dr. MEREWETHER remarks, the fact that a serious, even fatal, degree of fibrosis can be produced by other dusts has not been generally appreciated. A peculiar feature of the fibrosis produced by asbestos dust is the occurrence in the sputum or lung-juice of bodies, described by Prof. MATTHEW STEWART, which have a curious resemblance to a minute crustacean.[2] These consist, as BURTON WOOD and GLOYNE have shown, of a series of discs around a central spicule of asbestos.

The manufacturing processes fall into one of two groups according as they employ (more or less) pure asbestos and asbestos along with other dusty substances. The former group has been the subject of this inquiry on the ground that the effect of asbestos dust alone must be worked out before the complicated factors can be assessed. The population at risk in this group of the industry is not exactly known, but is estimated at 2200. A certain amount of selection was used in the examination, new-comers and long-time workers being chosen. The men examined were with one exception at work on the day of examination; their number (excluding 11 cases who had previously worked in other dusty occupations) was 363, or 16·5 per cent., of the estimated total workers in the industry. Of these, 95 (or 26·2 per cent.) showed a diffuse fibrosis of the lungs attributable to the inhalation of dust; 21 others had precursive signs of the disease. In 133 cases radiograms were made; 62 of these showed definite diffuse fibrosis, 25 were only suggestive. Dr. MEREWETHER is careful to emphasise the fact that only a proportion of the workers have been examined and that it would be unfair to assume that roughly 1 in 4 asbestos workers have fibrosis; the group examined was loaded with a high proportion of men who had been five years or more in the industry. The actual figures were these. There were no cases of fibrosis among those who had been employed less than 4 years, 36 cases in those employed from 5–9 years, 27 cases in those employed 10–14 years, 15 cases in those employed 15–19 years, and 17 cases in those employed over 20 years. Considering the relation of fibrosis to age the largest number of cases were found in the age-groups 30–39 and 40–49; Dr. MEREWETHER deduces that the important factor is the length of exposure, and that the age factor is negligible. The difficulties in ascertaining the incidence of fibrosis amongst workers in particular processes seem insurmountable as many processes are housed in one room and workers are transferred from one process to another. But there is clearly a lower incidence and probably a later onset among the spinners as a group than among those who crush, card, or weave asbestos. No evidence was found to indicate that one variety of asbestos is more potent than another in producing fibrosis. Concentration of dust and length of exposure are interdependent factors. For a number of years disablement, it seems, is curiously slight, even more so than in the case of silicosis. This may be due in part to the character of the disease, and in part to the fact that the work is not as a rule strenuous. The affected worker often continues at work with occasional breakdowns from bronchitis until he his aware of shortness of breath upon exertion. A terminal broncho-pneumonia or other acute infection may supervene while the man is still at work; with other there may be a period of invalidism. Particulars are given of ten cases up to the end of 1929 in which a advanced degree of abestosis without tuberculosis was the primary cause of death, nine being verified post mortem. The length of exposure in these cases varied from 9 to 24 years. The rate of progress seems to vary within wide limits. With continuous exposure to high concentrations of dust the fibrosis may be fully developed in from seven to nine years with a lesser the fibrosis may take 15 to 25 years to mature. So far as the available data go there is no special liability to pulmonary tuberculosis among asbestos workers.

Preventive measures are dealt with by Mr. C. W. PRICE in the second part of the report. The factories and workshops concerned number at least 160, of which 18 are textile factories. Small premises are the rule. The majority are well-housed and well-ventilated, but there are some open-sided sheds which are difficult to keep clean, and some which are congested with material. The arrangements for ventilation vary much. Dust removal systems where used effect a measure of general ventilation; over-head extracting fans are unsuitable and little used. Extraction by downward displacement has been considered for weaving rooms and Mr. PRICE considers it might, with suitable safeguards, replace localised exhaust ventilation, although it could not be successful without efficient heating. While many dusty processes are already recognised, he points

[1] H.M. Stationery Office. Pp. 34. 1s. 3d.
[2] THE LANCET, March 1st, p. 445, and March 29th, p. 701.

is represented by the effort to find a nonvolatile agent for complete narcosis or, failing that, a nonvolatile agent to serve for so-called basal anesthesia, supplemented by the use of a volatile anesthetic. Lendle's paper seems to show that neither Avertin nor the barbital derivative "Pernocton" is a satisfactory solution of the problem. Safe and efficacious new methods of anesthesia may result from the modern work in this field; but all the evidence indicates that the products thus far proposed for so-called basal anesthesia are hypnotics or sedatives and should be used as such. They cannot be safely used for complete anesthesia and can be safely used in combination with other agents for the production of complete anesthesia only by those thoroughly experienced in the administration of anesthetics and closely familiar with the studies of the use of nonvolatile agents for anesthesia. Thus far their intravenous use must be considered unsafe.

PULMONARY ASBESTOSIS

In 1928 THE JOURNAL,[1] commenting on an article by W. E. Cooke,[2] suggested that pulmonary asbestosis, because of its dangers and its unique pathologic features, deserved more attention than had been accorded it. Two and a half years later Mills [3] reported the first case from the United States. His patient had worked in an asbestos mine in South America prior to 1898, had returned to the United States, and again had been employed in South America, this time in an unstated occupation, between 1911 and 1913. He died in Minnesota in 1929. The history of the case indicates that the asbestos which was found in the subject's lungs at necropsy was inhaled prior to 1898. If so, the profound pulmonary changes which evidently were induced by the asbestos, and which seemingly contributed to the patient's death, were present thirty-two years after the agent that caused them was introduced. Even if the exposure to the asbestos occurred at the time of the patient's later visit to South America, the pulmonary injury endured at least seventeen years. Accordingly, Mills has concluded that pulmonary asbestosis is an incurable disease, from which the patient may or may not die. The cause of death, if tuberculosis is avoided, seems to be pulmonary fibrosis with its indirect effect on the heart. At necropsy of Cook's patient, pulmonary fibrosis and evidence of extensive tuberculosis were found; it was not certain whether the fibrosis was due more to the tuberculosis or to the asbestosis. In Mills's case, evidence of tuberculosis was not found but the fibrosis was severe. The conjecture seems warranted, therefore, that asbestos is capable of producing two types of injury, somewhat as iron dust is capable of producing two types of pulmonary siderosis. According to Bohrod,[4] who cited the work of Hart, the red type of iron lung is found in persons who work in iron mines and in factories using certain iron pigments: pneumonoconiotic induration predominates and there is little tuberculosis. Metal grinders and polishers display the black type of iron lung; induration is less evident and tuberculosis is unrestrained. That siderosis and asbestosis are allied seems likely in view of the evidence that the iron-containing [1] portions of asbestos constitute the danger to health in the asbestos industry. Concerning the relation of physicians of the United States to this industry, Mills pointed out that asbestos is mined and manufactured in many parts of this country and that pulmonary asbestosis surely will be encountered. In England the workmen's compensation act has recently been extended to include this condition.[5]

1. Pulmonary Asbestosis, editorial, J. A. M. A. 90: 119 (Jan. 14) 1928.
2. Cooke, W. E.: Pulmonary Asbestosis, Brit. M. J. 90: 1024 (Dec. 3) 1927.
3. Mills, R. G.: Pulmonary Asbestosis: Report of a Case, Minnesota Med. 13: 495 (July) 1930.
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INSENSIBLE PERSPIRATION AND BASAL METABOLISM

The estimation of basal metabolism has proved to be unexpectedly valuable as a diagnostic aid. The measurements of the respiratory exchange, which are now depended on to furnish the requisite information, have been greatly simplified in recent years, so that the procedure can be made applicable even in the office of the individual practitioner. There are, however, refinements of technic that ought to be applied more widely than they usually are. This refers particularly to the proper preparation of the patient and to the maintenance of strictly "basal" conditions. In the case of infants and children, obvious difficulties present themselves in the measurement of respiratory exchange as well as in direct estimations of heat output. Fortunately an alternative procedure has been proposed in the relatively simple method of ascertaining the insensible losses represented by perspiration. The determination of loss by insensible perspiration as an index of metabolism was discussed in detail in 1926 by Benedict and Root [6] in its application to adults. Its use in relation to children has been championed by S. Z. Levine and his co-workers [7] in the Department of Pediatrics of the Cornell University Medical College, New York. Basal standards of insensible perspiration were proposed by these workers in reference to the normal infant. The latest researches of Levine and Marples [8] have been concerned with the validity of the proposed methods and their basis of reference to predictions. A statistical study was made of the published data responsible for the standards at present available for the basal metabolism and basal insensible perspiration of normal infants. The investigation included consideration of the correlation between the two variables in simultaneous calorimetric measurements as well as in independent measurements with the respiration chamber and the balance. The evidence secured points to a high positive correlation between the physiologic mechanisms of heat production and insensible perspiration in the human subject.

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7. Levine, S. Z.; Wilson, J. R., and Kelly, Margaret: The Insensible Perspiration in Infancy and Childhood: I. Its Constancy in Infants Under Standard Conditions and the Effects of Various Physiologic Factors, Am. J. Dis. Child. 37: 791 (April) 1929; II. Proposed Basal Standards for Infants, ibid. 39: 917 (May) 1930.
8. Levine, S. Z., and Marples, Eleanor: III. Basal Metabolism and Basal Insensible Perspiration of the Normal Infant: A Statistical Study of Reliability and of Correlation, Am. J. Dis. Child. 40: 268 (Aug.) 1930.

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and Bode would group under the separate heading of erythroleukæmia, but there seems little necessity for this further group. All cases of erythræmia may be regarded as potentially leukæmic. Frequent and copious bleeding and phenylhydrazine are regarded as the best means of treatment, and a special warning is given against splenectomy.

It is doubtful if the third group of Parkes Weber and Bode—polycythæmia hypertonica—needs to be differentiated from the first. Polycythæmia associated with high blood pressure was first described as an entity by Gaisböck, and on the continent rejoices in an eponym. High blood pressure is found in many cases of polycythæmia, both secondary and essential, and renal disturbances are not uncommon. All cases of polycythæmia might perhaps be brought together under the first and second of the authors' groups.

ANALYSIS OF PSYCHOTICS.

THE discussion at the Royal Society of Medicine on psychotherapy in mental disorder, which we reported last week (p. 625), revealed a divergence of view which may be explained in part by differences in scientific method and in part by differences of training and experience. On the one hand are psychiatrists whose work has been concerned principally with institutional material; on the other hand are psycho-analysts who for the most part have not had much to do with the type of psychotic found in mental hospitals and whose training in psycho-pathology has been predominantly on Freudian lines. Dr. Devine from a wide experience of confirmed psychoses again advocated the view, so ably expounded in a recent book, that the basic cause of psychotic manifestations is primarily "organic." He uses the term "organic" in a special sense as pertaining to the vegetative aspects of the organism, not necessarily the same as the usual clinical sense of "structural." With such a point of departure it is clear that the modifications which psychotherapy tends to produce—primarily at the psychological level of the integrated activities of the organism—cannot be considered as cardinal but must be regarded as symptomatic palliatives. Psycho-analysts, however, feel capable of explaining all the characteristic psychotic symptoms in psychological terms alone, deduced from psycho-analytical interpretation of the mental processes involved. The fact that these interpretations have not received by any means a universal acceptance, and that even those psychiatrists who are sympathetic towards much that is psycho-analytic are still sceptical of some of the most fundamental of the assumptions involved, naturally does not deter psycho-analysts from pursuing their working hypotheses. Dr. Glover refrained from doing much more than defining the ways in which by the special psycho-analytic methods of approach psychotics may become accessible to psychotherapy. He explained that the cases so far dealt with were few and the time that has elapsed too short to justify final claims. We might add that a perusal of Abraham's pioneer psycho-analytic work on some depressive psychoses, mentioned by Dr. Sylvia Payne in her contribution, does not compel conviction of the validity of his interpretations.

But there is a viewpoint which in the discussion between organicists and psycho-analysts hardly received sufficient attention, although it is widely held among continental psychiatrists. For example, a distinction has long been made between the "fundamental disease process" and the "accessory symptoms" of schizophrenia. The fundamental process would correspond to Devine's organic substratum, while the accessory symptoms are presumably accessible to psychotherapy. Similar interpretations would make [illegible] cases of manic-depressive type and also many [illegible] idiopathic epilepsy explicable on the basis of [illegible], sometimes inherited, responses to psycho[illegible] factors. Here also psychotherapy should have a [illegible] application in the resolution of the precipitating [illegible]. Another view regards schizophrenia, paranoia, and paranoid conditions as frequently the result of habit-deterioration, the psychotic development being traceable in the life-history, where traits of personality emerge of a type that leads to increasing maladjustment and finally to the rupture of compensation that we call the psychosis. Looked at in this way the therapeutic problem of the psychoses, as Dr. Gillespie pointed out, seems more hopeful, principally from the preventive aspect. This would involve working with children and adolescents in the pre-psychotic stages. Some confirmation is forthcoming both from recent psychiatric experience with children and also, it is interesting to find, from some instances of the psycho-analysis of children such as those quoted by Dr. Melanie Klein.

What the discussion principally brought out is the need for much closer rapport between the different investigators, psychiatrists and psycho-analysts. It is unfortunate that there should be such a division of opinion as springs from imperfect collaboration. It is clear also that there is much room for research into the predisposition to psychosis and into the possibilities of modifying the various factors while they are in the making. It would be most useful if a number of people, apparent candidates for psychopathy, could be followed and studied from childhood into adult life. Furthermore, an attempt should be made to disentangle the congenital or inherited and presumably less modifiable predispositions from those which are acquired and presumably more modifiable. Our county asylums, drawing upon a defined area of population, should be peculiarly well situated for the study not only of mental disease in families but of the inheritance of individual traits and tendencies. In the psycho-pathology of the developed psychoses themselves there remains a wide field for study. The relative dearth of articles on this topic published from mental hospitals in this country is in striking contrast with the number of researches on the physico-chemical condition of mentally sick patients. The problem of accessibility has not been sufficiently studied; a continuous search should be made for a technique of access. When these questions have been better explored it will become possible to discuss treatment, psychotherapeutic and otherwise, more effectively.

PULMONARY ASBESTOSIS.

IT is now recognised that the inhalation of dust generated in the manufacture of asbestos often gives rise to an insidious and progressive pulmonary fibrosis which is distressing in its effects and disastrous in its consequences. The value of asbestos depends upon its well-known resistance to heat and to the action of strong acids and alkalis. Its importance in the manufacture of fire-resisting articles, whether designed to protect against risk or for use in connexion with machinery, and the absence of any efficient substitute is the cause of ever-increasing demands upon the factories where the fibre-containing rock is crushed and the fibres spun and carded. The number of persons employed in the factories is already large and, as Sir Thomas Oliver[1] points out, many of the workers are young. In the past too little attention has been paid to their welfare, and the need for rigid standards of protection is urgent if further suffering and loss of life are to be prevented. The effects of the inhalation of asbestos are due to the silica which it contains, but the resulting fibrosis differs histologically from the nodular form seen in other types of silicotic lung—e.g., in gold miners' phthisis. The whorled formations seen in the latter are absent and are replaced by a diffuse fine interstitial pneumonia. The difference is also reflected in radiograms of the two conditions, the gold miner's lung showing scattered dense rounded opacities which contrast with the fine basal mottling and linear striæ which are characteristic of the asbestos worker's lung.

[1] Oliver, T., Arch. f. Gewerbepath. und Gewerbehyg., 1929, i., Heft 2.

A peculiar feature of pulmonary asbestosis is the presence of the so-called asbestosis bodies. These were discovered by Prof. Matthew Stewart in the sputum of workers suffering from the disease, and they have been demonstrated by a number of investigators in sections from the lungs, and in lung juice removed during life by exploratory puncture or expressed at autopsy. The asbestosis body, which bears a curious resemblance to a minute crustacean, consists of a central core of asbestos fibre which appears to be embedded in a series of discs. One end of the fibre is often buried in a bulbous extremity, and this with the succeeding discs produces the appearance of a minute screw. The nature of the aggregation around the central spicule is *unknown*, but Gloyne[2] has been able to demonstrate the central fibre by exposing the containing body to the action of sulphuric acid.

THE SCIENTIFIC EXAMINATION OF PICTURES.

THE famous Italian pictures, which returned to their various homes last week, after being on crowded view in Burlington House for the past two months, evoked enormous general enthusiasm and, as was inevitable, certain discussions on the attributions. For more than ever is the authenticity of old masters becoming a serious financial matter, while the prices for genuine canvasses soar upwards under American desire of possession. It is significant of much that is happening in clinical medicine that recourse should be had to exact scientific methods in diagnosing the period of a painting and possibly the actual painter—critical acumen is taking its place with *tactus eruditus* as something personal whose value can be added to by employing the methods of laboratory technique. Dr. A. P. Laurie, professor of chemistry to the Royal Academy of Arts, has contributed to this month's issue of *The Analyst* an article—a record of a recent lecture delivered before his Society—upon the identification of pigments used in painting at different periods, in the course of which he gives a brief account of other methods of examining pictures. The technique employed closely resembles that of the pathological laboratory, and it is not suggested that the verdicts of these investigations are to take the place of the judgments of the instructed. Knowledge of the art of painting, its history, and the manners of the various schools, of brushwork, and of idiosyncrasies of well-known executants will remain the medium of identification, but the chemist and the radiologist can be drawn upon for assistance. Dr. A. Martin de Wild (see THE LANCET, Nov. 2nd, 1929, p. 932) has gone over the ground, when investigating the pigments used by the Dutch and Flemish masters from the fifteenth century onwards, and such a system of inquiry may give art critics a feeling, shared by many medical practitioners, that, with a disputed diagnosis as a possibility, the calling in of the chemist and radiologist is a measure of ordinary prudence.

PYELOGRAPHY WITHOUT CATHETERISATION.

GOOD results are claimed[3] for a new method of examining the urinary tract by means of a preparation called uroselectan. Since the advent of cholecystography urologists have been stimulated to search for a similar method applicable to their specialty. After trying various substances Lichtenberg and Zwick have hit upon an iodide compound which they call uroselectan, and it promises to be very useful for the purpose. As it is badly tolerated by the stomach it has been given by intravenous injection. Two injections are made at three minutes' interval, the first 40 c.cm. and the second 60 c.cm. of a 40 per cent. solution of the drug. Films may be taken a quarter, three-quarters, and one and a quarter hours after the final injection, and it is said that as a rule good shadows are obtained of the whole urinary If, however, the renal function is depresse shadow may appear for six hours or more, an rapidity with which a pyelogram is obtain therefore a rough guide to the efficiency of the k The method may be regarded as a definite adv in urology. Where it is sufficient for diagr purposes it is obviously a great gain to the pa who is spared ureteral catheterisation, and it al also be useful in those cases in which the catheté tion is attended by special difficulties, a advanced tuberculosis of the bladder. Urosel has already been tried by a number of surgeo Germany and France, and is being tested in Eng Mr. Frank Kidd records some observations i current number of the *Journal of Urology*.

[2] Wood, W. Burton, and Gloyne, S. Woodhouse: THE LANCET, March 1st, p. 445.

[3] Valley-Radot, P., Dalsace, J., Nemours-Auguste, and Dérot, M.: Presse Méd., March 19th, p. 385.

A FRIPP MEMORIAL.

A PROPOSAL has been made in the public that a memorial to the late Sir Alfred Fripp sh be instituted to perpetuate his memory, and we received from the popular novelist, Major Beith, an outline of the scheme which has beer in hand. It is proposed to invite public subscript to a fund to be devoted to the development extension of the Children's Department of G Hospital, the endowing of cots, the building of a ward, or in such other manner as the governor the hospital and Sir Alfred's own relatives decide. The trustees of the fund will be I Lonsdale, Mr. Wilfred Godfrey, and Major Hay Be Subscriptions may be sent to, and will be gratef acknowledged by the Hon. Secretary, the "Alfred Fripp" Memorial Fund, 115, Cheap London, E.C.2. Major Beith writes: "Two cha teristic donations have already been received by a cheque for £1000 from those truly pract philanthropists, the Froth Blowers, and a sum 10*s.* raised by farthings among the children o a County Council school in one of the poorest distr of London." The total sum subscribed will handed intact to the Governors of Guy's Hospi an institution, as our readers know, with which Alfred was closely associated as a student, surge and governor for over 40 years.

VITAMIN DEFICIENCY AND ANÆMIA OF PREGNANCY IN INDIA.

AN anæmia of pregnancy is common in India ama all communities except the European. The bl picture closely resembles that of pernicious anæm and treatment with liver extract is often successi If untreated the disease is frequently fatal or there m be recovery after delivery. The anæmia is differ tiated from true pernicious anæmia by this occurrer of spontaneous recovery and by the rarity of achl hydria, regarded as an invariable concomitant of t pernicious anæmia. The condition has received a go deal of attention without any light being thrown up its ætiology so far. A new study, however, has be made by L. Wills and M. M. Mehta, working under t auspices of the Indian Research Fund Association f the Maternal Mortality Inquiry, and they offer sor interesting suggestions in the course of a prelimina report.[1] Their first important contribution is t recognition that the disease is not essentially a anæmia of pregnancy, but widely affects wome throughout the population. It is only exacerbated b the exigency of pregnancy. Hearsay evidence quoted that it is even common also among men. Th wide prevalence of the condition and the equally wi prevalence of poor nourishment, deficient both i quantity and quality, readily suggest the likelihoo that an association between the two is of fundament importance in ætiology. The report is only a pr liminary one, chiefly occupied with the accurate defin

[1] Wills, L., and Mehta, M. M.: Ind. Jour. Med. Res., 192 xvii., 777.

of London. Although the surgeons of the time usually came of a humbler social class and received a less liberal education than the physicians, they based their practice on sound anatomical lines and were rapidly improving their position in the medical world; they not unnaturally resented the action of the physicians, some of whom also advised surgery, in prohibiting them from giving internal remedies. Ambroise Paré, who wrote treatises on medicine and midwifery, is regarded by Dr. Herbert Spencer as "the reformer of surgery and midwifery."

PULMONARY ASBESTOSIS.

DR. E. R. A. MEREWETHER and Mr. C. W. Price have carried out with commendable success an official inquiry into the effects of asbestos dust in inducing fibrosis of the lung. Their results are embodied in a report submitted by the Chief Inspector of Factories to the Home Secretary on March 17th, and laid before Parliament on March 24th. Before February, 1928, when a case of non-tuberculous fibrosis of the lungs was discovered in an asbestos worker (Seiler's case), the Factory Department of the Home Office had knowledge of only two deaths about which there was an expert opinion that the inhalation of asbestos dust had contributed to, if not caused, the fatal outcome. In the first of these, described by Dr. Montague Murray in 1906 before the Departmental Committee on Compensation for Industrial Diseases, the post-mortem examination confirmed the clinical diagnosis of extensive non-tuberculous pulmonary fibrosis; in the second, which was described by Dr. W. E. Cooke, director of the Pathological Department of the Wigan Infirmary, in the *British Medical Journal* of July 26th, 1924 (p. 147), the illness showed a progressive dust fibrosis together with chronic tuberculous infection, but the evidence for an etiological relation between inhalation of asbestos dust and fibrosis of the lung would have been stronger if tuberculosis had been absent. The inquiry by Dr. Merewether and Mr. Price was initiated when, investigation of Seiler's case having definitely excluded other industrial and infective causes of fibrosis, it became necessary to decide whether the supervention of this disease in an asbestos worker was an exceptional occurrence or evidence of a grave industrial risk. Dr. Cooke's observation that microscopical examination of sections made from the lung of his case revealed the presence of curious annular and club-shaped bodies, and clinical and pathological observations that have since then been published from Leeds, London, and South Africa, have shown that free silica cannot be regarded as the only dust capable of producing pulmonary fibrosis, and that the inhalation of silicate of magnesium, the constituent of asbestos dust, is, if anything, more effective in producing a like condition. Dr. Merewether and Mr. Price at the beginning of their investigation tried to discover how many persons in the country are constantly exposed in the course of their daily work to the influence of pure or almost pure asbestos dust. Such information was necessary in order that they might check the adequacy of the sample of workers examined, and envisage in proper perspective the problem confronting the industry. The number of exposed workers they believe to be about 2,200, and of these they examined 363, in 133 the examination including radiology of the chest. Diffuse pulmonary fibrosis attributable to the inhalation of dust was found to be present in 85 of the workers examined, representing 26.2 per cent. of the series. By distributing the workers according to the process in which each had been longest employed, and grouping similar processes together, the authors of the report were able to make certain broad generalizations about the relative effects of work in different dust concentrations. Correlating dust counts with the history and the medical and radiological features of each case, they found that the two factors of greatest importance in the development of fibrosis were length of exposure and dust concentration. Age and sex played no part. While no definite case of fibrosis clearly due to asbestos dust was found among workers with less than five years' exposure, the authors are not prepared to rule out the possibility that such cases might occur on exposure of workers to high concentrations of dust. They lay stress on the fact that for the production of generalized fibrosis a definite minimal quantity of dust must be inhaled, and that the lower the concentration the lower will be the time that elapses before fibrosis develops. If, as seems probable, this view is correct, the practical inferences are very important, for it follows that the application of measures resulting in a reduction of the concentration of dust in the neighbourhood of asbestos processes will bring about, first, a great increase in the latent period preceding the development of disabling fibrosis, and secondly, as measures for the prevention of dust are perfected, the almost total disappearance of the disease. The increased liability of silicotics to pulmonary tuberculosis does not appear to be shared by asbestos workers as a class, or even by those of them who have developed pulmonary fibrosis. In this respect, and in the mode of distribution of the fibrous tissue in the lungs and in the radiological findings, pulmonary fibrosis among asbestos workers differs from silicosis. At the same time as the report by Dr. Merewether and Mr. Price was issued Earl Russell introduced a bill in the House of Lords to extend Section 47 of the Workmen's Compensation Act, 1925 (relating to silicosis schemes), to industries involving exposure to asbestos dust, and to amend the provision concerned with medical arrangements and examinations. In our issue of March 23rd, 1929 (p. 562), we drew attention to inconsistencies in the four existing schemes applicable to groups of "dusty occupations"; this bill aims at making a general scheme to cover all industries and processes to which compensation schemes apply.

[1] *Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry.* By E. R. A. Merewether, M.D., and C. W. Price. H.M. Stationery Office. 1930. (1s. 3d. net.)

DELAYED LETHAL EFFECT OF RADIUM ON TISSUE CULTURES.

A FURTHER step in the investigation of the destructive influence of radium on certain types of new growth is reported by Dr. F. G. Spear.[1] In the *Journal* of September 14th last (p. 508) we referred to the discovery by Drs. Canti and Spear that after a short exposure of a tissue culture to gamma irradiation there was a marked fall in the number of cells in mitosis (40 per cent. that of controls), and that this was followed by an increase (160 per cent. that of controls), which compensated almost exactly for the diminution seen during the first period and subsequent to irradiation. Dr. Spear now reports experiments showing that the gamma rays have a delayed lethal effect on tissue cultures *in vitro*. While the resistance of individual cultures to irradiation was found to vary considerably, it seemed to be established that the greater the intensity of irradiation the sooner the lethal effect could be observed. When 300 mg. of radium was used at a distance of 0.5 cm. the probable life of a culture was approximately doubled if the duration of exposure was halved; with 100 mg. of radium, however, halving the exposure had a less pronounced tendency to double the life of the culture. Comparison of the action of the two doses of radium showed that the exposure of a culture to 300 mg. of radium for six hours produced a lethal effect six days (three subcultures) earlier than did exposure to 100 mg. radium for eighteen hours, though in each case the dosage was the same, amounting to 1,800 milligram hours. It was also found

[1] *Proceedings of the Royal Society*, B, vol. 106, 1930, p. 44.

tomy is still in its infancy. Post-operative observations may be changed as the survival period increases, which, if it does, will alter the surgical indications and the selection of cases. Close cooperation between laboratory investigators, clinicians and neurosurgeons is imperative in order to make the best selection of cases for surgery and to evaluate accurately the results obtained, whether they are failures or successes.

ASBESTOSIS

A. J. LANZA, M.D.
Assistant Medical Director, Metropolitan Life Insurance Company
NEW YORK

Asbestosis is a pneumoconiosis caused by the inhalation of asbestos dust. It is distinct from silicosis in its pathology and clinically. Whether asbestosis will remain a distinct form of pulmonary dust disease or will prove to be of a type common to a number of dusts remains to be seen. So far, it is the only pneumoconiosis, other than silicosis, that has received any considerable amount of study from pathologists, clinicians and industrial hygienists.

Whereas silicosis has been recognized for many centuries, asbestosis is a newcomer. Asbestos (Canadian) is a hydrated magnesium silicate containing no free silica but about 44 per cent of combined silica, 43 per cent magnesium, nearly 13 per cent of water, and traces of iron and nickel. While asbestos was known to the ancients, the fabricating of asbestos on a large scale is comparatively new; it received a tremendous impetus from its use in connection with automobiles and as an insulating material and a heat resistant for a great variety of mechanical purposes.

While Hoffman[1] called attention to the possible harmfulness of asbestos dust in 1918, it was not until February 1927 that asbestosis was, so to speak, officially recognized in this country by the filing of a disability claim for workmen's compensation in Massachusetts. The claimant was a foreman in the weaving department of an asbestos plant, and the claim was upheld by the Massachusetts Industrial Accident Board. This was twenty-seven years after the first fatal case was reported in England by Dr. Montague Murray. In 1910 and again in 1934 a fatal case was reported in England, and in 1928 the British Factory Department conducted an investigation and enacted laws for the protection and compensation of the worker against this hazard. The whole subject in England has been summarized by Merewether.[2] At the present time, as nearly as can be estimated, there are about 12,000 individuals employed in the chief asbestos plants in the United States, of whom 10,000 might be exposed to asbestos dust.

In 1927 a fatal case of uncomplicated asbestosis was reported to the Medical Society of South Carolina,[3] and since then, including this case, there have been eleven fatal cases reported in the United States, eight uncomplicated and three complicated by tuberculosis. These reports, together with the fact that asbestosis figured in the extraordinary occupational disease litigation that has spread over this country, resulted in both laboratory and field studies of this new hazard. Gardner and Cummings[4] at Saranac Lake, N. Y., conducted animal experimentation with asbestos in dust and reported their observations in 1931. These two authorities in the United States and Gloyne[5] in England have described the pathology. While silicosis is predominantly parenchymatous, asbestosis is mainly interstitial; nor is asbestosis characterized by the nodular formation so distinctive of silicosis.

A search of all the death records on file in the Metropolitan Life Insurance Company revealed that asbestosis had been given as a cause or contributing cause of death in only nineteen cases. The first case noted was in 1924, there were two in 1927, one occurred in 1931 and the rest have occurred since 1933. The diagnosis was supported by autopsy in only six. In one of the six the primary cause of death was carcinoma, in another glioma of the brain, and in another pulmonary tuberculosis. In the other three asbestosis was given as the primary cause, with cardiac failure as the contributing cause. Some of these cases were reported in the literature.

Asbestosis complicated by heart disease was given as the cause of death in seven cases. There were no autopsies in these seven and pulmonary tuberculosis was diagnosed in several by the attending physician, so perhaps too much weight cannot be given to these certificates.

Diabetes and pulmonary tuberculosis were also mentioned in the remaining six cases, in which there were no autopsies. All nineteen patients were males and with one exception all were white.

In our studies of asbestos mines and fabricating plants,[6] the clinical picture of asbestosis was milder than that of silicosis. To be sure, the individual patient with marked asbestosis will greatly resemble the individual with silicosis. There is the same dyspnea on exertion, the same dry cough, and the more or less indefinite physical signs elicited by the stethoscope. The patient with asbestosis is apt to have clubbed fingers—not usually seen in silicosis—and he is apt to be pasty faced and even show a slight cyanosis, while the silicotic patient is apt to be fairly robust looking. Of course, in each instance I refer to patients whose disease is not complicated by infection.

We did not find in communities in which asbestos was mined or fabricated the familiar picture of disability and tuberculous infection so characteristic of hard rock mining communities. Our observations were supported by the statements of physicians practicing in these communities. All the patients with asbestosis that we detected were, with one exception, working steadily at their trades. In only one case did we find evidence of active tuberculosis and that diagnosis was based only on the roentgen appearance. Several showed healed tuberculosis. Gardner and Cummings in their reports called attention to the difference between the action of asbestos dust and silica dust in relation to tuberculous infection in experimental animals, and their observations tend to bear out our clinical study.

In all, our clinical data are based on 126 physical examinations of asbestos workers, all of whom had more than three years' exposure and who were selected at random. Sixty-three of these presented a roentgen appearance which we thought indicated a pneumoconiosis.

Read before the Section on Preventive and Industrial Medicine and Public Health at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 12, 1935.

1. Hoffman, F. L.: Mortality from Respiratory Diseases in Dusty Trades, Bull. 231, U. S. Department of Labor, June 1918.
2. Merewether, E. R. A.: A Memorandum on Asbestosis, Tubercle 15:69 (Nov.), 109 (Dec.) 1933; 15:152 (Jan.) 1934.
3. Lynch, K. M., and Smith, W. A.: Pulmonary Asbestosis, Am. Rev. Tuberc. 23:643 (June) 1931.
4. Gardner, L. U., and Cummings, D. E.: Studies on Experimental Pneumonokoniosis: VI. Inhalation of Asbestos Dust: Its Effect upon Primary Tuberculous Infection, J. Indust. Hyg. 13:65 (Feb.) 1931.
5. Gloyne, S. R.: The Morbid Anatomy and Histology of Asbestosis, Tubercle 14:445 (July), 493 (Aug.), 550 (Sept.) 1933.
6. Lanza, A. J.; McConnell, W. J., and Fehnel, J. W.: Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers, Pub. Health Rep. 50:1 (Jan. 4) 1935.

[illegible] but the symptoms were [illegible] and [illegible]. We called these cases [illegible] presenting evident; [illegible] symptoms and [illegible] roentgenograms, we termed second stage. Of these sixty-seven patients, twenty had been exposed more than ten years and thirteen more than fifteen years. We are still continuing our asbestos studies and hope to secure additional information on the time element in development and the tendency and rate of progress.

One feature that has impressed us is that the British investigators found asbestosis more severe and more menacing than we did. This difference may be more apparent than real, but it is possible that the English factories may be more dusty than ours. There were not available any comparative dust counts, but this impression is based on their reports and on statements made to myself and my colleagues by persons familiar with the English conditions. One process, described in the British reports as "mattress making" and stated to be extremely dusty, does not appear to have a counterpart in this country. In both countries, energetic steps have been taken to control the dust hazard in asbestos plants, so that it is probable that further cases of disabling asbestosis will be rare.

As in silicosis, the diagnosis centers on the roentgenogram. However, the whole matter of attempting to interpret these films and correlate them with the clinical evidence, if any, is difficult and elusive. If, in our studies, we had found a more clear-cut and severe type of pneumoconiosis with marked symptoms and disability as well as a distinctive roentgen appearance, such as my colleagues and myself had been accustomed to find in our previous investigations of silicosis, our task in attempting to make a positive diagnosis and estimate the extent of the disease would have been easier. There is no doubt that, especially in the beginning, we were handicapped by endeavoring to evaluate asbestosis with a silicosis foot rule.

The x-ray appearances are not clear cut or distinctive as in silicosis and do not lend themselves to ready grouping into progressive stages. There are less evident pathologic changes in these films and the shadows are finer, more granular and softer than in silicosis. The asbestosis film gives the impression of ground glass, and there is no nodulation with the consequent tendency of the nodules to coalesce and give dense opaque areas in the films. The distribution of the shadows is somewhat different, occupying the lower third of the lung, except in far advanced cases, when the shadows may occupy the major portion of the lung. We noticed frequently the well marked outline of the interlobar septum on the right side. We also noticed that a number of films in cases of asbestosis showed enlarged hearts, and this might be expected when one considers that the pathologic process tends to constrict the pulmonary blood vessels as they ramify along with the bronchioles.[7]

It is possible that the x-ray appearance of asbestosis may not be distinctive of this disease alone but uniform in appearance with pneumoconiosis due to other silicate dusts. Much more investigation and study of roentgenograms of industrial workers exposed to all sorts of silicate and other dusts are needed before it will be possible to speak definitely on this, the most important phase of the diagnosis of pneumoconiosis.

One thing is certain. The utmost care and patience are needed to elicit the occupational history of the patient and to correlate this and the clinical picture with the roentgenogram before a diagnosis of asbestosis is justified. As noted in relation to other occupational health hazards, there [illegible] too frequent tendency to make a diagnosis of a specific occupational disease because of presumptive or actual exposure without the corroboration of other essential factors to a correct diagnosis. One is not justified in making a diagnosis of asbestosis any more than of silicosis in the presence of a roentgenogram showing no distinctive pulmonary pathologic changes.

Associated with exposure to asbestos dust is the occurrence in the sputum and pulmonary tissues of a peculiar formation known as asbestos bodies. These asbestos bodies have been described by a number of observers and are due apparently to the action of the tissues on the asbestos fiber. Their exact significance is doubtful, but it is commonly agreed at the present time that they are not diagnostic of pulmonary fibrosis and indicate merely that the individual has been exposed to asbestos dust.[8] Some observers believe that, when these asbestos bodies appear in the sputum in clumps, they indicate actual disintegration of lung tissue.

Dr. Miller[9] of the United States Public Health Service describes the technic that he employs in the intraperitoneal injection of finely divided dusts in a state of suspension. Miller defined three types of reaction: absorptive, inert and proliferative. The first is produced by relatively harmless or inactive dusts, the second by dusts that might cause pulmonary fibrosis, and the third as the typical reaction of silicosis. Recently Miller[10] described the effects of the injection of three varieties of asbestos; namely, chrysotile, crocidolite and amosite. Chrysotile is Canadian asbestos and, as previously stated, is largely magnesium silicate. Crocidolite and amosite contain only a small quantity of magnesium, containing instead iron silicate in approximately the same quantity.

The three types of asbestos produced the same type of reaction; namely, the one described by this investigator as inert. He states:

> We have been assuming that the dusts producing this inert reaction cause pneumoconiosis of the diffuse fibrosis type as distinct from the proliferative reacting dusts which cause nodular fibrosis. This assumption, as you know, has not been proven by corresponding animal experiments with the same dusts, but results from observation of pathologic material from autopsies. . . . I believe that the gross behavior of asbestos in the tissues has further strengthened the value of our classification of dusts and the intraperitoneal test as a means of determining the harmful dusts by its clinical correlation.

Much work remains to be done before asbestosis is spoken of as authoritatively as is silicosis. In the meantime, asbestos plants are being cleaned up and the dust is being controlled. This, together with the smaller number of persons employed, implies that there will probably never be the wealth of clinical material that has been available in silicosis. It is by no means certain that asbestosis progresses as does silicosis after withdrawal from dust exposure, nor does infection seem to be as closely and intimately associated with asbestosis as with silicosis. The answer to these and other problems resulting from exposure to silicate dusts demands further study both in the field and in the laboratory.

Metropolitan Life Insurance Company.

7. [illegible], J. L., in discussion on McCann, W. F.; [illegible], Alberto; [illegible], [illegible], and [illegible], R. W.: The [illegible] Functional [illegible] in the Pulmonary [illegible], J. A. M. A. 102: [illegible] (Sept. [illegible]) [illegible].

8. Merewether, [illegible] Wood, W. B., and Gloyne, S. R.: Pulmonary Asbestosis, Lancet 2: 1383 (Dec. 22) 1934.

9. Miller, J. W., and Sayers, R. R.: The Physiological Response of the Peritoneum to Dusts Introduced as Foreign Bodies, Pub. Health Rep. 49: [illegible] (Jan. [illegible]) [illegible].

10. Miller, J. W.: Personal communication to the author.

ASBESTOSIS BODIES IN SPUTUM AND LUNG*

KENNETH M. LYNCH, M.D.
AND
W. ATMAR SMITH, M.D.
CHARLESTON, S. C.

Asbestosis has, of comparatively recent time, assumed a position f prominent and perhaps peculiar interest in the field of pneumonoconiosis and occupational disease. While the asbestos industry is more than two thousand years old, it is only within the past few years that it has assumed the prominent place which it now occupies in the industrial arts. Until recently asbestosis has been assumed to be essentially silicosis, and the free silica has been thought to constitute the dangerous factor in pneumonoconiosis as contrasted with the relative harmlessness of other dusts.

It has come to attention within the last three or four years that there is a peculiar characteristic in the state of asbestosis, not found in silicosis, and it is our purpose in this report to record some data that have come to hand, since American medicine thus far has no report on this peculiar condition and to the end that particular interest may be stimulated in its study in the extensive asbestos industry. It is pected that a further report of our studies be made at a later time.

In 1924, Cooke[1] recorded an autopsy on an asbestos worker who had been exposed for twenty years, the last five years intermittently, showing extensive fibrosis of the lungs and chronic tuberculosis. At this time there was seen in the lungs only black particles, from 3 to 393 microns in length, of various shapes.

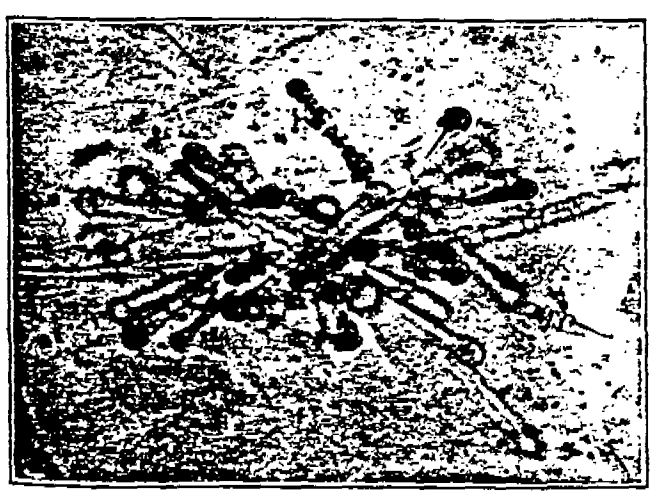

Fig. 1.—Asbestosis bodies in sputum concentrate; reduced from a photomicrograph with a magnification of 705 diameters.

In 1927, Cooke[2] again recorded this case and cited another in which a patient who had worked in an asbestos mill for ten years showed at autopsy pulmonary fibrosis and spicules of asbestos in the lungs. These two cases are apparently the only records of autopsies in cases of asbestosis up to that time.

In a companion article, McDonald[3] recorded a histologic study of the lung tissue in Cooke's case and stated that the appearances were practically the same as in another case the sections of which were in the possession of Dr. I. M. D. Grieve of Armlay, Leeds. McDonald found, in addition to well marked diffuse interstitial pneumonia, chronic bronchitis and emphysema, anthracosis and extensive tuberculosis, certain peculiar foreign bodies in the alveoli, bronchioles and

Fig. 2.—Asbestosis bodies in sputum; magnification about 950 diameters.

interstitial fibrotic areas. This is the first recorded observation of these objects, which have since been called "asbestosis bodies." There was considerable disagreement among zoologists, botanists and others as to their nature; McDonald advanced the hypothesis that they are portions of asbestos in process of alteration and absorption by hydrolysis, with the passing of the silica into a colloidal state and later into a gel.

In 1928, Simson[4] followed with a report of four autopsies on asbestos mill workers in South Africa, the first being done in 1926 without discovery of the asbestosis bodies at the time. One of these subjects worked for twelve months in the mill, developed tuberculosis and died; the second worked for two years in the same mill and had a prolonged pneumonia during this time. This patient had neither tuberculosis nor pneumonia at autopsy but fibrosis of the lungs. The length of exposure of the third and fourth patients is not given, but both died of lobar pneumonia and both showed very little fibrosis. In the lung tissues of all of the four were found "golden yellow" segmented structures with rounded ends.

The descriptions and illustrations by McDonald and Simson make it clear that they were dealing with the same object and that it is the same as we have found in the lungs and sputum of asbestos workers.

In October, 1929, there occurred in the service of one of us (K. M. L.) an autopsy on a patient dead of

* From the Departments of Pathology and Internal Medicine, Medical College of the State of South Carolina.

1. Cooke, W. E.: Fibrosis of Lungs Due to the Inhalation of Asbestos Dust, Brit. M. J. 2: 147 (July 26) 1924.
2. Cooke, W. E.: Pulmonary Asbestosis, Brit. M. J. 2: 1024 (Dec. 3) 1927.
3. McDonald, S.: Histology of Pulmonary Asbestosis, Brit. M. J. 2: 1025 (Dec. 3) 1927.
4. Simson, F. W.: Pulmonary Asbestosis in South Africa, Brit.

a gunshot wound. This was a Negro man who, at the time of his death, was a worker in an asbestos mill, having done such work for a total of twenty-eight months during a period of about three years. At the autopsy there was nothing of significance beyond the gunshot wounds of the chest and abdomen. In microscopic examination of the tissues, however, the peculiar bodies under consideration here were found in the sec-

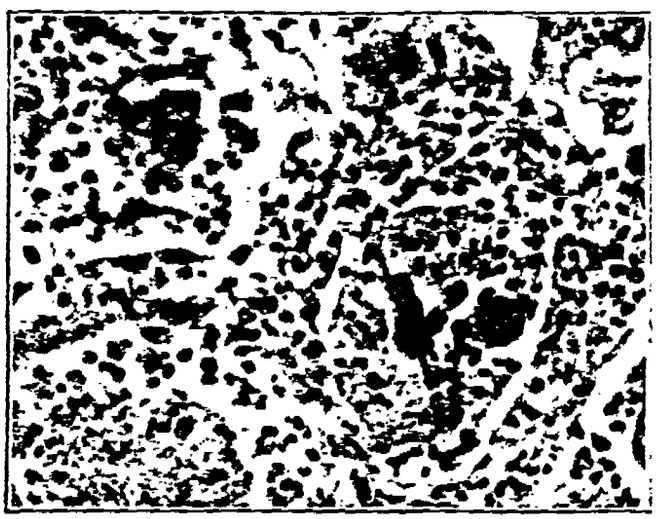

Fig. 3.—Asbestosis bodies in lung with phagocytes and black granular pigment within alveoli in pneumonia; reduced from a photomicrograph with a magnification of 475 diameters.

tions from both lungs, in alveoli, associated with fine black granular pigment and mononuclear cells, some being intimately associated with large mononuclear phagocytes, in the bronchioles, where there was also the black granular material and some mononuclears, in the alveolar walls, which were thickened, and within the connective tissues of the interlobular areas along the course of vessels and bronchi. Here there was a definite increase of fibrous tissue and much accumulation of black granular pigment.

At about the same time occurred another autopsy on a Negro man who was working in an asbestos mill at the time of development of lobar pneumonia from which he died. He had been in this occupation almost continuously for a period of four and one-half years. In this case, also, it was in the microscopic study of the tissues from the autopsy that the asbestosis was discovered, gross examination revealing only massive pneumonia of the whole right lung and upper left lobe.

Sections of all parts of the lungs from this case revealed the "asbestosis" bodies. There were great numbers of them in the alveoli, the walls of the alveoli, the deeper pleural tissues, the bronchi and the interlobular framework tissues, and they even occurred in thrombi of veins and in the peribronchial lymph nodes. Associated with them was much black granular pigment, in the alveoli, interstitial tissues and lymph nodes, and in these nodes was also a heavy deposit of yellow-brown granular substance of the same color as the asbestosis bodies. Mononuclear and polynuclear giant cell phagocytes were prominent in intimate connection with the asbestosis bodies in the alveoli, bronchi and veins. They contained much of the black granular material in the alveoli and bronchi, also. In the walls of the alveoli and bronchi and in the framework structures where these bodies lay was a cellular increase in fibrous tissue.

The peculiar bodies in the lungs of these two patients were first suspected of being mycelia and spores of a fungus, possibly *Aspergillus*. On investigation of the subjects and after we had learned of their occupation, the reports of McDonald and Simson were encountered and identification with the objects observed by them was established.

It then occurred to mind that these bodies should be found in the sputum of such patients, since they occurred in considerable numbers in the alveoli and bronchi, and that sputum examination might prove to be an important measure in the diagnosis of asbestosis. Consequently search was made for them in sputum of asbestos workers from the clientele of one of us (W. A. S.) Specimens of sputum were examined by direct wet smear preparation and by examining the concentrated sediment of sputum after digesting it in 10 per cent sodium hydroxide solution, kept warmed to about 80 C. until the mucus was well digested and the mixture watery.

The first sputum examined came from a man who by clinical and roentgen examination had active fibrous caseous tuberculosis and also a moderate grade of pneumonoconiosis. He had not worked in asbestos for approximately a year but had been previously so occupied for about ten years. The sputum contained great numbers of tubercle bacilli. Asbestosis bodies were readily found in direct wet slide preparations and in large numbers in the sodium hydroxide digest concentrate.

It was at this point that it came to attention that such sputum tests had already been made. In 1929 Stewart and Haddow,[5] and in 1930 Wood and Gloyne[6] showed that they could be found by digesting the sputum with equal parts of concentrated antiformin and examining the centrifugated deposit. Subsequently we have found the asbestosis bodies in the sputum of two other asbestos workers and have failed to find them in one.

Fig. 4.—Asbestosis bodies with phagocytes and black granular pigment in fibrous lung; reduced from a photomicrograph with a magnification of 280 diameters.

The second patient was a Negro woman who had worked in an asbestos factory from 1918 to 1924 but not since. She had no evidence of pulmonary disease and was under treatment for syphilis. None of the asbestosis

5. Stewart, M. J., and Haddow, A. C.: Demonstration of the Presence of Bodies ("Asbestosis Bodies") in Material Obtained by Lung Puncture and in the Sputum, J. Path. & Bact. 32: 172 (Jan.) 1929.

6. Wood, W. B., and Gloyne, S. R.: Pulmonary Asbestosis, Lancet 1: 445 (March 1) 1930.

bodies were found in direct unconcentrated wet preparation. In the sodium hydroxide digest concentrate they were sparse and were not as long as those found in the first case. The sputum was mucopurulent.

The third patient had not worked in asbestos for approximately two years. He was employed in an asbestos factory for seven months in 1924 and for about two years from 1926 to 1928. He had active fibrocaseous tuberculosis of the lungs, with tubercle bacilli in the sputum but no condition distinguishable as pneumonoconiosis. The first sputum was mucoid in character and insufficient for concentration. No asbestosis bodies were found in direct wet unconcentrated preparations. In this specimen were large numbers of dust cells, with fine black particles and numerous fine needle-like crystals. The second sputum was mucopurulent. A few asbestosis bodies were found in the sodium hydroxide digest concentrate but none in the unconcentrated preparations. These sparse bodies were usually small, but some were long and thick.

The fourth sputum examination was done in the case of a white man who had worked in an asbestos factory for about fourteen years but not since 1926. He had advanced fibrosis of the lungs, thought to be a late stage of pneumonoconiosis, without any evidence of tuberculosis, and was in a state of advancing cardiac failure. Examination of sputum on three separate occasions by both direct and concentrated preparation failed to reveal any asbestosis bodies.

It was anticipated that this case, on account of the length of exposure and the extent of pulmonary disease, should have the best opportunity of exhibiting these bodies, but this expectation has not been realized. The first patient had been exposed within about a year, and it showed by far the greatest number of asbestosis bodies in the sputum, but the other two patients had not been more recently exposed, one had not worked in asbestos for about five years, and neither of these two gave any distinguishable evidence of pneumonoconiosis.

It remains a subject for further study as to the relation of the occurrence and time of duration of these bodies in the sputum to the extent of exposure to asbestos dust, and as to the state or stage of asbestosis or other associated conditions in relation to their expulsion in the sputum.

The asbestosis bodies are quite variable in size, color and form but generally are of a characteristic structure. In the sputum, from which they may be better studied alone, they have a central filament of a transparent, slightly greenish tinged needle-like crystal. This is taken to be an asbestos crystal. On this filament is deposited, in varying quantity, nodules, blobs and segments of a homogeneous refractive substance, varying from a shining light yellow or greenish-yellow to a deep mahogony brown, depending on the thickness of the deposit. In general this deposit occurs more on the extremities, usually in a spherical blob here, tapering toward the middle of the filament, which may be bare, giving the appearance of two clubs or baseball bats placed small end to small end. Again there may be a ball of the deposit on the extremities and the shaft have cylindric deposit of segments or disks of uniform or irregular size, giving the whole body the appearance of a dumb-bell. The clubbed form may break in the middle, leaving two indian-club forms. Again, the shaft and extremities may be the seat of more or less symmetrical deposits of globules, piled up, to give a variety of architectural figures. The segments or disks of deposit along the crystal filament may be pressed closely together or they may be separated by an interval at which the needle-like central crystal may be seen. Sometimes the shaft is covered by a homogeneous non-segmented deposit.

In the lungs they are of the same forms, but their full length may not be seen and they usually appear shorter. The size of the body varies widely in length and thickness, with the length of the filament and the amount and uniformity of the deposit. No extensive measurements have been made in this study, but in the lungs they have been measured from 12 to 100 microns long and from 1 to 12 thick, the shaft being narrower than the ends. In sputum they have been measured from 1 to 12 microns thick and up to 140 microns long.

Asbestosis bodies do not take the ordinary tissue and sputum dyes and they have been found in their natural form in one of these cases in slide preparations of sputum stained by the ordinary carbolfuchsin method for tubercle bacilli.

They may be stained on the slide by the prussian blue reaction for iron, the heavier and darker brown bodies taking a deep indigo blue, this shading to a lighter color in proportion to the heaviness and darkness of the yellow-brown substance. This reaction indicates a considerable iron content to the deposit. This iron has been thought to signify that the asbestosis body comes from the iron content of asbestos. Since the material is evidently a deposit within the lung on an asbestos crystal, this does not appear to be a satisfactory explanation. It is suggested that the iron content, if not the whole substance of the deposit, is of tissue origin, probably from the blood.

Clinical Notes, Suggestions and New Instruments

THE EXECUTION OF ROBERT H. WHITE BY HYDROCYANIC ACID GAS

EDWARD E. HAMER, M.D., CARSON CITY, NEV.
State Health Officer

Robert H. White was executed by hydrocyanic acid gas at the Nevada State Prison, June 2, 1930. A study of the heart and respiratory action was made at the time of the execution, a Bowles stethoscope being applied on the bare chest wall over the apex of the heart. The tube leading to the ear pieces on the outside of the prison wall was connected to the stethoscope while the prisoner was being strapped to the chair.

This was at 4:36 a. m. The heart action at that time was 108, strong and regular. The gas was started generating at 4:37½. At 4:38 the pulse rate was 120, regular and strong. A small inspiration was taken at 4:37¾, at which time the prisoner indicated that he smelled the gas. At 4:38 he took a very deep inspiration, turning his head toward the gas. He gave a spasmodic cough, his head fell forward, and he became unconscious. Following this first deep inspiration there was a complete stopping of the heart action for fifteen seconds. After that short period of time, or at 4:38½, the heart again began to beat in an irregular manner, continuing thus for fifteen seconds, when it became regular and strong. There was no apparent loss of power in the heart action. After this time, for two minutes the heart became slower, beating 100 times a minute at 4:41½ and eighty times a minute at 4:44. At 4:46½ the heart beats were distinctly regular but becoming very weak. The last heart beat was noted at 4:47.

Respirations during this time after the first deep inspiration were convulsive and irregular.