Gothenburg, where epidemic catarrhal jaundice was extremely prevalent.

3. The clinical history of the case here described is similar to that of many recorded from Sweden—an onset resembling in every way that of a benign case of epidemic catarrhal jaundice: improvement, then quite suddenly the appearance of symptoms pointing to an acute liver insufficiency.

4. The pathological changes of epidemic catarrhal jaundice found in sections of the liver taken from living patients by Nordmann (1925) and Eppinger (1922), and confirmed by sections obtained from patients who had died from other conditions, are those of a hepatitis grading into acute atrophy. Eppinger states that severe cases of acute catarrhal jaundice are forms of acute atrophy of the liver in miniature. The pathological changes in many of the cases reported by Bergstrand (1930) are also very similar to those found in the present case.

5. Bergstrand (1930), and Findlay, Dunlop, and Brown (1931) have expressed the view that epidemic catarrhal jaundice is probably due to a virus which, though pathogenic for man, cannot be transmitted to monkeys or to the smaller laboratory animals, while acute necrosis of the liver is due to some superadded toxæmia in a patient whose liver has not fully recovered from the primary attack of the virus. In support of the view that a toxin and an infection together produce a more widespread necrosis of the liver than either alone may be cited the classic work of Opie (1910), who found that acute hepatic necrosis resulted from the combined action of chloroform and *Bacillus coli*. Hurst and Hurst (1928) also found that the toxic action on the liver of manganese chloride is reinforced by injections of *Bacillus coli*, while Findlay, Dunlop, and Brown (1931) showed that arsphenamine injections, combined with injections of *Bacillus coli*, produce far more hepatic necrosis than either the coliform infection or the arsphenamine alone.

6. The presence of secondary bacterial infections in association with acute necrosis of the liver has been recorded by McDonald (1918), while it is of considerable interest that in a number of Swedish cases Bergstrand (1930) has isolated a *Streptococcus viridans* in pure culture from the duodenum. This is a further point of similarity to the present case, in which a streptococcal focus was located in the wall of the transverse colon.

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SILICO-ANTHRACOSIS PRESENTING CURIOUS BODIES SIMILAR TO THOSE IN ASBESTOSIS

BY

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(With Special Plate)

The arguments against anthracosis ever being a primary condition are so strong that the time would seem opportune to give to the disease a designation which is more correctly descriptive. Cummins,¹ Cummins and Sladden,² and Jousset³ have shown that the primary factor in the production of coal-miners' lung is silicosis, and that coal dust is retained in large amounts only when there is a high silica content. And the converse is true: without a high silica content there is no important retention of coal dust in the lungs. Cummins and Sladden conclude that coal dust is retained only when the lymph drainage in the lungs has been impaired by the action of silica, and that anthracosis is a special variety of silicosis depending upon the accumulation of coal dust in the silica-damaged lung. The following case supports their contention.

CASE HISTORY

A man, aged 64, was admitted to the Wigan Infirmary on September 17th, 1931, with the history that at the age of 10 he had commenced work as a "half-timer" in a cotton mill; at 16 he was employed at the Park Lane Collieries, near Wigan, and worked there continuously as a drawer and collier until the collieries closed in 1926. He had had no other employment, nor had he lived outside the Wigan district. The seams in these mines are all soft coal, no anthracite being present. On admission the patient was found to be suffering from multiple myelomatosis, with collapse of the bodies of the fourth and fifth thoracic vertebrae and paraplegia. He gave a history of dyspnoea and chronic cough, with mucoid viscid sputum. The urine contained a large amount of Bence-Jones proteose. No tubercle bacilli were found. The radiologist's report on the chest stated that there was extensive fibrosis of both lungs, but no mottling or evidence of silicosis, and the right base showed pleural thickening. The patient died on September 27th, 1931.

POST-MORTEM FINDINGS

There was invasion of the sternum, ribs, and vertebrae by neoplasm. The bodies of the fourth and fifth thoracic vertebrae were collapsed and surrounded by a tumour mass.

Lungs.—There were dense pleural adhesions on both sides, more marked on the right. The thoracic glands were black and enlarged, but not hard. The lungs were black and uniformly fibrosed, with the exception that four or five small hard nodules could be felt in each lung substance. There was no gross anthracotic consolidation.

The diaphragm, the capsule on the upper surface of the liver, and the pre-aortic glands showed deposits of carbonaceous matter.

HISTOLOGICAL APPEARANCES

The bone tumours present the histological characteristics of myeloma multiplex.

Lungs.—There is marked excess of dust throughout the sections, and the interalveolar septa are thickened and laden with dust cells. The perivascular and peribronchial areas show extensive fibrosis with large accumulations of dust—the result of blockage of the lymphatics; an occasional fibrous whorl is present. There is a considerable degree of emphysema. The pleura is thickened, and there are subpleural carbonaceous deposits, but no subpleural lymphoid hyperplasia is seen in the sections. By polarized light doubly refractile particles are seen in large numbers. In addition, some sections show masses of golden yellow pigment (? altered haemoglobin) and large black angular particles measuring from 5 to 80 μ in length. Some of the shapes are seen in the illustrations as the bases of the curious bodies.

The Curious Bodies.—In sections and after digestion of the lung with trypsin, some of the large angular particles are

seen to have a golden brown material deposited around them. Many, especially the bodies that have slender spicules as central cores, present the double and single bulbous ends and the annular arrangement of the deposit so frequently seen in the bodies in asbestosis. In asbestosis these bodies are present in myriads, and are of all sizes and shapes, from minute forms resembling streptococci to bodies 130 μ or more in length, as in Fig. 1 on Plate. Their size depends upon the length of the fibre and the size of the particles of asbestos dust which act as nuclei. In chrysotile dust there are black angular particles of biotite and magnetite, and in asbestosis some of these particles have the same appearance as those found in this case, and shown in Figs. 7, 8, 9, and 10. The particles in powdered iron pyrites closely resemble the large spicules. The curious bodies are not nearly so numerous or so varied in form as in asbestosis.

Tylecote and Dunn⁴ have recently published the finding of similar bodies in a case of a coal miner who had worked in Alabama as well as in English coal mines. According to the Geological Survey of Alabama, the iron ores are associated with the coal measures in an area of over 2,000 square miles, and these haematites contain silica in amounts up to 43 per cent. In the present case no iron ore except pyrites (FeS_2), or any strata containing large amounts of silica, were encountered during the course of the man's working life.

CHEMICAL ANALYSIS OF THE LUNGS

The analysis was as follows:

	Per cent.	
Mineral matter	11.5	of dried lung.
Insoluble silica	15.6	of ash
Soluble silica	24.6	"
Total silica	40.2	"
Ferric oxide	7.2	"
Phosphoric acid	0.66	"
Alumina	28.7	"
Calcium oxide	0.2	"
Magnesium oxide	0.3	"
Total sulphur (probably organic) ...	0.45	"
Alkalis	22.25	"

The silica figure is as high as the average analysis of the lungs of six quartz miners in South Africa given by McCrae⁵—namely, 39.7 per cent.—and approximates the highest figure in Cummins and Sladden's series of coal miners' lungs—42.1 per cent. The iron content is also high, but further analyses showed that the sulphur present was organic and therefore neither the iron nor the sulphur was due to the presence of unaltered iron pyrites. In this connexion it must be remembered that the total amount of the large black spicules seen in certain sections and in the digested deposit was infinitesimal compared with the bulk of the ash.

DISCUSSION

Curious Bodies.—The presence of silica and altered haemoglobin may be important in the ultimate solution of the problem of the curious bodies. The mode of formation of these bodies in asbestosis was discussed at the Annual Meeting of the British Medical Association in 1929.⁶ The bodies consist of slender cores of asbestos fibre, upon which colloidal aggregates of blood proteins plus a soluble fraction of asbestos are adsorbed. Syneuresis occurs, and with the loss of water the adsorption is rendered irreversible, and the fibre becomes permanently ensheathed. It was then suggested that the bodies were not peculiar to asbestosis, but that any spicule of insoluble material might have colloid deposited around it and become moulded into a curious body. The point is illustrated by Figs. 4, 5, and 6, which are of bodies having cores of biotite spicules. The constancy of the bodies in asbestosis has led them to be called "asbestosis" or "asbestos bodies," but in view of the case described by Tylecote and Dunn, and the present one, any title which implies pathognomicity would appear inaccurate. Schuster⁷ has reported a case of asbestosis in a wire-haired terrier that lived in an asbestos factory nearly all its life of 10 years. Although asbestos fibres were present in large numbers in the lung sections, not a single curious body was found.

Coal-miners' Lung.—The work of Cummins and Sladden, and of Jousset, has given us a new conception of anthracosis. The cases described by Cummins and Sladden were in anthracite workers, but the present case shows that the same conditions obtain in soft-coal miners. The present view of the pathology of anthracosis would appear to necessitate a readjustment, not only of the accepted radiological interpretation of silicosis and the pneumoconioses in general, but also of the clinical and industrial aspects of the disease. As an aid to the new conception the title "silico-anthracosis" has been suggested independently by Cummins and myself.

I should like to express my gratitude to Mr. T. H. Byrom for undertaking the analyses in this case.

The photomicrographs reproduced on the plate are by C. F. Hill and W. E. Cooke. Magnification 400 diameters.

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PAROXYSMAL TACHYCARDIA ASSOCIATED WITH PREGNANCY

BY

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In a recent issue of the *British Medical Journal*, James¹ records a case of asthma in which the attacks were in abeyance during pregnancy, only to recur shortly after delivery. The following notes of a case of a different nature may prove of interest, as here also there is illustrated the beneficial effect exercised by the pregnant state—possibly by the stimulation of the endocrine system which is thereby induced.

A woman, aged 22, pregnant for the first time, was admitted in labour to the Glasgow Royal Maternity and Women's Hospital on the evening of October 30th, 1931. On general examination the only points of note were that the skin was very pale, although the mucous membranes were of fairly good colour, and that there was a supernumerary breast on the left side. The heart sounds were pure and of good quality: there was no oedema. The patient had been a regular attender at the ante-natal dispensary, and, during these months, the progress was uneventful. She would not admit any ill-health, and the only item of interest gleaned from the family history was that her father was classed C3 during the war on account of a weak heart.

At 1.40 a.m. on October 31st a healthy female mature child was delivered spontaneously. The puerperium was uneventful until 5 a.m. on November 6th, when the heart suddenly began to beat rapidly. It was impossible to count the pulse at the wrist. The apical rate was ascertained to be 220 per minute. The heart continued beating at this rate until about 5.45 p.m. on November 11th, when the patient saw everything suddenly becoming black; she had a feeling as if the heart stopped for a couple of seconds, and as if something in the praecordial region snapped, whereupon the heart commenced to beat at about half its previous rate. The pulse and apical rate were then found to be 104 per minute. Breast-feeding was discontinued at the onset of the paroxysm, when the patient was seen by the consulting physician to the hospital, Dr. Wm. MacLennan.

Throughout this attack, lasting just over five and a half days, the cardiac rhythm was perfectly regular; this was confirmed, as was also the rate, by polygraphic tracings,

G. M. FINDLAY & J. L. DUNLOP: NECROSIS OF
LIVER WITH EPIDEMIC CATARRHAL JAUNDICE

FIG. 1.—Acute necrosis of the liver associated with epidemic catarrhal jaundice in a child.

W. E. COOKE: SILICO-ANTHRACOSIS WITH
"CURIOUS BODIES"

FIG. 1.—Curious body from a case of asbestosis, 137 μ in length. FIG. 2.—Curious body in asbestosis; colloidal coating fractured to show fine asbestos filament forming the core. FIG. 3.—A common form in asbestosis; elongated dumb-bell with annular or discoid appearance of deposit. FIGS. 4, 5, and lower figure in FIG. 6.—Curious bodies from a case of asbestosis showing biotite nuclei. FIGS. 7, 8, 9 and 10.—Curious bodies from the present case of silico-anthracosis. In FIGS. 8 and 10, discoid arrangement of deposit is seen.

WILLIAM HUNTER: UNUSUAL BONE INJURY
IN A CHILD

FIG. 1.—Greenstick bowing of ulna.

T. H. CROZIER: EXOPHTHALMIC GOITRE
IN A CHILD

FIG. 1.—Shows classical "bulging" eyes and startled expression; also scar of dog-bite on right cheek.