showed numerous "heart-failure" cells, many containing haemosiderin.

In the fourth case, that of an adult female, we draw attention to the development of a miliary appearance, localized to one mid-zone, after an attack of acute pulmonary oedema. Again lung puncture showed numerous pigmented macrophages.

Laubry's finding that lung puncture is a valuable diagnostic procedure has therefore been confirmed in our cases.

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A new diagnostic method using ultrasonics—high-frequency mechanical vibrations above the pitch of audible sound—was briefly described in the *British Medical Journal*, 1951, **1**, 931. A short editorial review of the subject in the *Journal of the American Medical Association*, July 14, p. 1033, discusses more recent work which has interesting possibilities, including the accurate localization of intracerebral new growths, the detection of intracranial foreign bodies, and the production of ventriculograms without the need to inject air. It is also claimed that carcinomatous growths of the breast can be distinguished from other tumours of that organ by ultrasonic echo-sounding. The basis of the method is that the transmission of ultrasonic vibrations depends on the density, elasticity, and viscosity of the tissues; when, for instance, an ultrasonic beam passes through a muscle and reaches the surrounding fascia, which is denser and less elastic than the muscle fibres, some of the ultrasonic energy will be reflected, and the rest will pass on with diminished force. Thus a picture somewhat like a radiograph can be obtained, but giving very different information, since it is built up from differences in the penetration of a mechanical vibration and not from varying transmission of electromagnetic rays from an *x*-ray tube. If instead of a beam a short ultrasonic pulse is used, the transmitter can be used to receive echoes during its quiescent period between each pulse, and, since it is about the size of a stethoscope bell, it can be applied to any part of the body's surface.

IDIOPATHIC PULMONARY HAEMOSIDEROSIS

REPORT OF A CASE IN AN ADULT

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Idiopathic pulmonary haemosiderosis (essential brown induration of the lung) is now a well-recognized clinical syndrome. Wyllie *et al.* (1948) review the 17 cases in the literature previous to 1947 and add seven further cases from Great Ormond Street Hospital for Sick Children. Further cases have since been reported by Nancekievill (1949), McLetchie and Colpitts (1949), and Gertrude Luther (1949). With few exceptions the cases so far reported have been in children (but see previous paper). Borsos-Nachtnebel described the condition in a man aged 38, and Belfrage and Waldenstrom reported a case which developed at 16 years of age and ended fatally at 19 (Wyllie *et al.*, 1948). We record a further case which began at the age of 16 and in which death occurred at 19.

Case Report

The patient, a boy aged 16, was first seen by one of us (A.A.W.) in April, 1947, when he was admitted to hospital with anaemia. A blood count showed: haemoglobin, 54%; red cells, 3,000,000; colour index 0.9; white cells 11,000 (polymorphs 59%, eosinophils 2%, lymphocytes 35%, monocytes 4%). This improved on iron therapy. A radiograph of the chest at this time was normal. Later he noticed that he was rather more short of breath when playing football.

In 1948 he was conscripted into the R.A.F. He found that drills, marches, and physical training made him very short of breath and exhausted. Mass radiography at the time of his arrival in the R.A.F. showed a widespread ground-glass mottling, accompanied by hilar flare, most marked in the right mid-zone, and also slight cardiac enlargement. At this time he was symptomless. He reported sick on December 1, 1948, and was admitted to an R.A.F. hospital. He was acutely ill. He had been coughing up blood-stained sputum, was pyrexial, and there were showers of fine crepitations scattered through both lung fields. The red cells numbered 3,800,000, sputum was negative for tubercle bacilli, and the Mantoux test was strongly positive at 1/1,000. Further *x*-ray examination showed extension of the shadowing. At that time it was considered that the *x*-ray findings were consistent with an acute bronchopneumonic spread of tuberculosis. Accordingly he was given a course of streptomycin, 100 g. Later a bronchoscopy was found to be normal. The anaemia responded to iron. On one occasion he developed languor and headaches, but examination of the cerebrospinal fluid was normal.

On a later review of the radiographs the possibility of idiopathic pulmonary haemosiderosis was considered. He was discharged from the R.A.F. on June 18, 1949, and reported to one of us (M.W.).

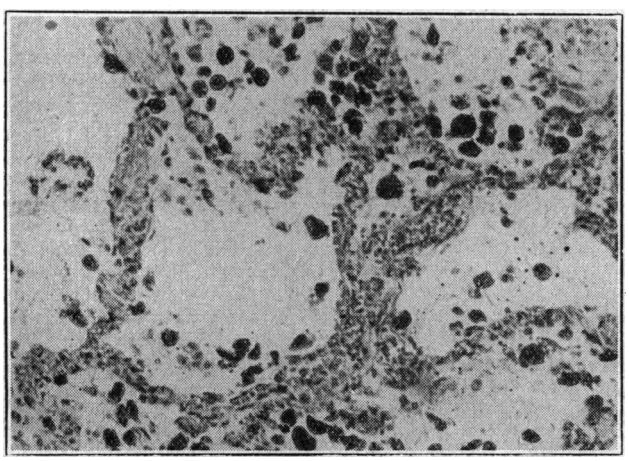
After being invalided from the R.A.F. he was seen at two-monthly intervals. He continued to complain of breathlessness. In October, 1949, he had several small haemoptyses. The physical signs and radiological appearances continued unchanged until in June, 1950, he complained of epigastric pain, and his liver and spleen were enlarged. There was engorgement of the neck veins but no evidence of a cardiac

lesion. His blood pressure was 95/75. Other systems were normal. Blood investigations during this period of observation were: July 11, 1949: Hb 85%, white cells 6,500 (polymorphs 67%, lymphocytes 28%, monocytes 4%); September 28: Hb 98%, white cells 6,700 (polymorphs 52%, lymphocytes 47%, monocytes 1%); July 28, 1950: Hb 102%, white cells 11,600 (polymorphs 68%, lymphocytes 29%, monocytes 3%). Sternal puncture showed a hyperplastic normoblastic marrow. Radiographs of the chest showed fan-shaped, bilateral, symmetrical shadowing occupying mid-zones from hilum to periphery, enlarged pulmonary conus, and enlargement of the right side of the heart. The shadowing appeared to be made up of multiple fine densities.

On July 16, 1950, signs of congestive cardiac failure were more pronounced. The patient was very apathetic, there were sacral oedema and oedema of the ankles. He died on August 17.

Post-mortem examination was performed by Dr. P. Coleman (assistant pathologist, Middlesbrough Central Clinical Laboratory), who gave the following report.

"The body was that of a young man aged 19. There were no external marks or injuries. The heart weighed 2½ lb. (1.1 kg.) and was dilated, although there was little



Photomicrograph of section of lung showing haemosiderosis. (×240.)

hypertrophy of the muscle. There were no pulmonary adhesions, there was free fluid in the pleural and pericardial cavity, and the surface of the lungs showed a most peculiar mottled brown appearance. On section the lungs were solid and fibrous. There was a peculiar rusty brown staining of the whole cut surface. There was considerable enlargement of the tracheo-bronchial glands, which were pigmented. The liver was enlarged and weighed 4 lb. (1.8 kg.); the cut surface had the appearance of a nutmeg liver, the appearance being in an almost exaggerated degree. The spleen was enlarged, weight 14 oz. (400 g.). Alimentary canal, kidney suprarenal —nothing abnormal. Cranial cavity—nothing abnormal discovered."

Histological Examination.—Lungs: There were large numbers of haemosiderin-containing phagocytes in the alveoli and the alveolar-wall epithelium. The latter also showed some red cells and free haemosiderin, indicating both old and recent small haemorrhages. Throughout this section there was a fine diffuse pulmonary fibrosis. The picture was compatible with a pulmonary haemosiderosis (see illustration). *Tracheal Bronchial Gland:* This also contained haemosiderin associated with gross congestion and some anthracosis. *Spleen:* This was grossly congested; no evidence of iron-containing pigment. *Liver:* Typical gross nutmeg appearance; no evidence of iron-containing pigment.

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THE EMPHYSEMATOUS-BULLOUS FORM OF BRONCHIAL CANCER

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[WITH SPECIAL PLATE]

In a previous communication (Castex and Mazzei, 1941a, 1941b), when referring to the "true congenital bronchopneumopathies and the radiological appearances given by bullous emphysema," we recalled the fact that the necropsies of most cases of suspected polycystic disease of the lung showed that the real cause was an acquired disease—chronic bullous emphysema. We also stated at the time that among our statistics of "gigantic bullous emphysema" there had been two cases of bronchial cancer of dyspnoeic type. Later (Castex *et al.*, 1942) we insisted upon this occurrence (which we have not found mentioned in the available literature) when speaking of the "alveolar insufflation, bullous emphysema, and emphysematous spontaneous pneumothorax in bronchopulmonary tumours," and pointed out that among our cases of lung emphysema were some in which a bronchial tumour could pursue its clinical course masked by a disease of the bullous emphysema type.

Here it is necessary to insist that we are not speaking of the ordinary emphysema, nor of that produced by pre-atelectatic alveolar insufflation (well described by Chevalier Jackson, by Pasteur Vallery-Radot and Israel, by Overholt and Ray Rumell, by Maxwell, and by us), but of the *gigantic bullous emphysema.* The same clinical picture was later described by Pruvost *et al.* (1943), Even and Lecœur (1943), Guichard *et al.* (1943), Pruvost (1945), Santy *et al.* (1946), and Maurer *et al.* (1946).

From the pathogenic point of view, such bullous emphysema could be due to the bronchial cancer itself or its adenopathy. Acting as a valve mechanism, both could give rise to a regional disturbance of expiration, thus causing increased broncho-alveolar pressure, the subsequent insufflation producing an anatomical emphysema which would result in the bullous emphysema. This is similar to what we have obtained experimentally in dogs by means of cannulae (Castex *et al.*, 1947).

In the past two years we have found and followed three new cases of gigantic bullous emphysema caused by bronchial cancer.

Case 1

The patient, a man aged 48, smoked 15 to 20 cigarettes a day. He was admitted in October, 1948. His disease started in the middle of July with a temperature of 102.2° F.