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.

(39° C.) and profuse sweating; this episode lasted three days and was followed by generalized weakness, anorexia, and spasmodic cough; the latter was more marked at night, and was so severe that it interrupted sleep and compelled him to sit up in bed. In spite of different treatments his cough and loss of weight continued. There was a marked effort dyspnoea, and the cough became worse and proved resistant to any form of medication. He lost 11 lb. (5 kg.) in weight.

Examination showed emaciation and pallor; there was constant dyspnoea. The chest was emphysematous and hyperresonant, with marked decrease of the vesicular murmur. Two radiological studies were carried out:—(1) August, 1948: marked hili; increased translucency of the right upper third due to insufflation or emphysema; below there was diffused opacity; right hilum enlarged. (2) September 3, 1948 (Plate, Fig. 1):—Right lung: atelectasis of the basal lobes (lower and medial); bullous emphysema of the apical sector of the upper lobe (dissociated bronchial obstruction). Bronchoscopy of right side revealed a neoplasm on the main bronchus.

The patient died in November, 1948, from cachexia and respiratory failure.

Case 2

A 52-year-old male clerk was admitted in October, 1948. Since July he had had a persistent cough and ever-increasing effort dyspnoea; the dyspnoea was sometimes paroxysmal. There had been no haemoptysis. In three months he had lost 22 lb. (10 kg.) in weight, and felt very run-down.

On examination he had dyspnoea even while resting; the dyspnoea was both objective and subjective, with an increased respiration rate (30 a minute). Marked cyanosis of the lips, cheek-bones, and pinnae was present. There was venous congestion in the neck. The thorax was emphysematous; elasticity and excursion were greatly decreased. On palpation there was a marked decrease of respiratory murmur in the lower third on both sides of the chest. Percussion revealed a hyperresonant right hemithorax; the left side was dull in the upper two-thirds and resonant in the lower third. On auscultation there was silence in the resonant area, and in the lower half decreased vesicular murmur and prolonged expiration.

Radiological examination on October 29 (Fig. 2) showed:—Right lung: gigantic bullous emphysema in the upper field which pushed down both lobes (middle and lower) and the right hilum; in the base there were horizontal linear images. Left lung: a homogeneous shadow occupied more than the upper half; with the patient in a transverse position this was seen to correspond to the left upper lobe. The patient died in cachexia.

Case 3

This case was followed up by Dr. Mario Dreyer.

A married Argentine man aged 52 had felt increased loss of strength since the end of 1946. He was run down and had had frequent pyrexia. He did not improve under a tonic medication, and his blood sedimentation remained constantly high. Some months later the neck glands became enlarged, some of them to the size of an egg; this complication subsided. The gland enlargement episode was followed by a septic antrum, which required puncture. Later the cough reappeared, and there was increased expectoration, which was sometimes blood-stained. An x-ray film taken in January, 1947, showed:—Right lung: an air chamber of bullous emphysema in the upper half, hilum dense and proliferative. Left lung: increased network; left subclavicular shadow.

The patient's general condition became progressively worse, his dyspnoea increased, being present during rest. Metastases were found in several parts of the body, and one was removed from the abdomen for the purpose of an examination; Dr. Mosto reported "metastases of alveolar carcinoma."

The last x-ray film (1947) (Fig. 3) showed:—Right lung: large air chamber of bullous emphysema, dense egg-shaped hilum; in the lower field there was another supradiaphragmatic horizontal shadow. Left lung: the abnormalities seen in the former x-ray film have become worse.

Comment

These are three new observations of bronchial carcinoma in old chronic bronchial cases that, besides a severe general condition, presented a lung picture of emphysema and respiratory failure (dyspnoea and cyanosis). From the radiological point of view they showed bullous emphysema, sometimes of segmental or zonal type, sometimes unilobular, and, in others even more extensive, bilobular. There can be associated atelectasis of the other lobe or lung sector (dissociated bronchial obstruction).

From the clinical standpoint effort dyspnoea, dyspnoea during rest, and that of the paroxysmal type were the most outstanding and distressing features for the patients, respiratory failure being one of the causes of death.

Summary

Three new cases of gigantic bullous emphysema due to bronchial cancer are recorded.

This clinical picture, described by us in 1941, has been mentioned in a series of papers by French authors.

A relative number of cases and their common pattern led us to believe that they constituted a clinical form within a broncho-pulmonary neoplasm.

The radiological appearance of gigantic emphysema is associated with a clinical picture in which the most marked features are the respiratory failure with marked dyspnoea and cyanosis.

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The American medical profession is examining the question of educational employment of television (*J. Amer. med. Ass.*, June 2, p. 479). The number of available television channels is limited, and none have been assigned for educational broadcasts; stations are reluctant to offer free time for educational broadcasts because of high costs. The Bureau of Health Education has prepared a television manual which is free to county medical societies, and they recently telecast a programme which was "kinescoped" (a kinescope is a film reproduction of the original telecast with sound) and has since been shown in 45 television communities. The Bureau has also prepared television "packets"—a supply of scripts, titles, and explanatory material on charts or filmstrip—for local societies. These are available free of charge to medical societies and cover a variety of topics such as child care, preventive medicine, and cancer. There are three ways of providing television facilities for the medical profession in America: kinescopes prepared by the A.M.A., the television packets, which require more local preparation, and sponsored broadcasts. All these possibilities are being explored.

P. ELLMAN AND A. GEE (*contd.*)

J. E. CATES AND
M. B. McILROY:
TRYPANOSOMIASIS

FIG. 7.—Skiagram of Case 5 in February, 1948.

FIG. 8.—Skiagram of Case 5 in 1950. Resolution of miliary opacities in right lung and middle zone of left lung.

FIG. 1.—Photomicrograph of section of cervical lymph node in trypanosomiasis. ($\times 75$.)

M. R. CASTEX AND E. MAZZEI: EMPHYSEMATOUS-BULLOUS FORM OF BRONCHIAL CANCER

FIG. 1.

FIG. 2.

FIG. 3.

J. A. NIGHTINGALE: PULMONARY ARTERIOVENOUS FISTULA

FIG. 1.—Postero-anterior film of chest.

FIG. 2.—Right lateral film of chest.

FIG. 3.—Angiocardiogram—2-second film.