MASSIVE ATELECTATIC BRONCHIECTASIS ASSOCIATED WITH BRONCHIAL STENOSIS

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The application of thoracic surgery in children must eventually contribute to the knowledge of the pathogenesis of bronchiectasis. In this report clinical and pathological data concerning a case in which pneumonectomy was performed are presented.

F. B., a boy, aged five years, was well until 1933 when he had an illness diagnosed as pneumonia. The pulmonary lesion gradually resolved and an x-ray photograph taken later showed a patchy consolidation of the left lung, but no evidence of collapse (fig. 1). He recovered from this illness and was well until January, 1936, when he had a similar episode. Subsequently he
had a cough which was for a time associated with a little sputum and breathlessness. Later the cough was unproductive. There were no malaise, fever or other constitutional symptoms. In May, 1936, he was admitted to Westminster Hospital for investigation. On clinical examination it was noted that he was a well-nourished, lively child of good colour with slight clubbing of the fingers. The temperature was normal and there was only occasional cough without sputum. Meunier's method of searching for tubercle bacilli by gastric lavage gave negative results. The left side of the thorax showed restricted movement, complete dullness on percussion, medium-pitched bronchial breathing, and whispering pectoriloquy. The right border of the heart could not be percussed, and the cardiac apex lay opposite the fifth rib in the left mid-axillary line.

![Fig. 2.—Plain skiagram taken on admission.](http://adc.bmj.com)

X-ray examination showed that the left thorax was opaque from apex to base and small, scattered, calcified foci (not visible in the reproduction) were seen in the upper lobe (fig. 2). In view of a strongly positive intracutaneous tuberculin test (1 in 1,000 old tuberculin) it was thought that tuberculosis might be either the underlying or a contributing factor. The displacement of the mediastinum towards the affected side (fig. 2) seemed to rule out the diagnosis of epituberculosis or effusion. The presence of fluid was further excluded by thoracentesis which also demonstrated a greater negative pressure than normal on the affected side. There was no leucocytosis and the sedimentation rate was not increased.
At this stage of the investigation the diagnosis was an interesting one, and it appeared probable there might be a massive collapse or a fibroid lung; the presence of bronchiectasis was suspected and tuberculosis could not be excluded.

Bronchoscopy (Mr. F. C. Ormerod) revealed narrowing of the left main bronchus just proximal to the opening of the left upper lobe bronchus. No secretion was seen in the bronchus and beyond the stenosis the bronchi appeared to be normal. Bronchography with lipiodol then provided evidence of the greatest importance in the elucidation of the case (fig. 3 and 4). The trachea was displaced to the left. The opaque oil entered the bronchi on the left side and demonstrated cylindrical bronchiectasis, but did not penetrate far into the lung, showing that the lung parenchyma was not aerated. There was also much distortion of the bronchi. The first division of the right lower main bronchus was directed into the left side of the chest, showing herniation of the mediastinum by the right lower lobe. A partial stenosis of the left main bronchus was seen about one inch from the bifurcation.

**Treatment**

In consultation with Dr. Donald Paterson and Mr. Tudor Edwards it was decided that pneumonectomy should be performed. This decision was made because it was felt there was much risk of repeated infection taking place in a functionally useless lung. At operation Mr. Tudor Edwards found the lung to be extremely shrunken and adherent. The structures in the root of the lung were secured by mattress sutures and no enlarged glands were noted. After operation shock was counteracted and a blood transfusion performed. In the post-operation phase there was considerable pyrexia, attributable to infection of blood clot in the left thorax. Eventually the fever disappeared and healing began to take place satisfactorily. The general condition at the present time is excellent.

**Pathological report**

The specimen of the left lung removed at operation was fleshy and fibrous and contained numerous areas of calcification (fig. 5). Some of the bronchi showed dilation together with massive peribronchial fibrosis. Examination of the left main bronchus showed that the stenosed portion had been left behind in the ligatured stump.

Microscopic sections from various parts of the lung revealed marked peribronchial fibrosis and dilation of the bronchi. There was also dense lymphocytic infiltration chiefly around the bronchi. The alveoli were for the most part collapsed, although many contained blood. Some areas showed dense calcium deposits well circumscribed and encapsulated. Serial sections taken through one area suspicious of a tuberculous process, finally revealed a giant cell of the Langhans type with a surrounding area of epitheliod granulation tissue and lymphocytes (fig. 6 and 7). This histology was suggestive but not diagnostic of tuberculosis.
Two x-ray photographs taken at different times after lipiodol, showing displacement of the trachea to the left, cylindrical bronchiectasis and stenosis of the left main bronchus. In fig. 4 note the first division of the right lower main bronchus is directed into the left side of the chest showing herniation of the mediastinum by the right lower lobe.
FIG. 4.

MASSIVE ATELECTATIC BRONCHIECTASIS
Fig. 5.—Left lung removed at operation showing both cut surfaces. Note areas of calcification, peribronchial fibrosis and bronchial dilatation.
Fig. 6.—Microphotograph of lung containing an area resembling a tubercle.

Fig. 7.—High power magnification of fig. 6 showing giant cell of Langhans type.
Both etiologically and pathologically the interpretation of this case is difficult. The important points are the finding of a fibrotic and bronchiectatic lung in a patient in whom the tuberculin test is strongly positive. In children, tuberculosis is not usually regarded as a cause for widespread bronchiectasis. It may be remarked that Wallgren has described a localized and benign form of bronchiectasis occurring at the site and after the healing of a primary focus. There can be little doubt that tuberculosis of the 'childhood type' leads to varying degrees of induration and fibrosis, and it is a point worth considering if this type of tuberculosis may also contribute in the etiology of certain cases of bronchiectasis. The evidence available concerning the present case does not permit a final conclusion; the microscopic and histological findings, though suggestive of tuberculosis are not conclusive.

Another important positive finding in this case is the partial stenosis of the main left bronchus. It is not possible to say what caused this stenosis, though its presence might be an explanation for the failure of the left lung to expand and recover after the pulmonary infections which from the history are known to have taken place. The stenosis itself may have resulted from an ulcerative bronchial lesion or from the effects of a caseous gland in its neighbourhood.

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REFERENCE.

Massive atelectatic bronchiectasis associated with bronchial stenosis

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