*Surgical aspects of Pulmonary Tuberculosis in African children

by

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The Role of Surgery in the Relief of Bronchial Obstruction and in the Prevention of its Effects

The main purpose of this paper is to present a plea for a rational approach to hilar lymphadenectomy in the prevention of bronchiectasis, destroyed lung, emphysema and a massive tally of crippling respiratory disease. Operation should be considered **at an early stage** if drug therapy and physiotherapy are not clearly relieving bronchial compression or perforation by mediastinal lymph glands, either where the bronchial obstruction in itself is causing symptoms or where the resulting pulmonary lesion may be regarded as still reversible. This is an infinitely more attractive proposition to me as a surgeon than to be presented with the late effects of bronchostenosis and to be asked to salvage a respiratory cripple. I want to condemn in the strongest possible terms the indiscriminate use of drugs and bed rest in pulmonary tuberculosis when an obvious mechanical problem exists.

The Primary Complex and Bronchial Obstruction.

In the lung the glandular component of the primary tuberculous complex in young Africans is often florid in the extreme. Figure I. demonstrates a not unusual radiological appearance of this florid nature and it also demonstrates the common bilateral distribution of the lymph node enlargement. These features, the floridity and the bilateralcy of the lymph node involvement, in primary tuberculosis, together with the high incidence in African children help to explain the remarkable frequency of pathological states due to bronchial obstruction.

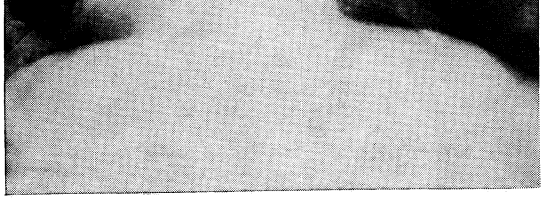

Figure I. The florid, bilateral hilar lymphadenopathy often seen in African patients with primary tuberculous infections in the lung.

The Effects of Bronchial Obstruction by Enlarged Lymph Nodes.

Some examples of the pathological states seen in the thoracic surgical clinic at Mpilo Central Hospital are illustrated in Figures II—VII. The lesions represent, in the main, changes which in themselves are not tuberculous but are the consequence of bronchial obstruction by enlarged lymph nodes.(1).

Bronchiectasis. The role of bronchial obstruction by tuberculous hilar lymph nodes has long been recognised in the causation of bronchiectasis. (2)(3)(4). The vulnerability of the soft, pliable bronchial walls of children is an

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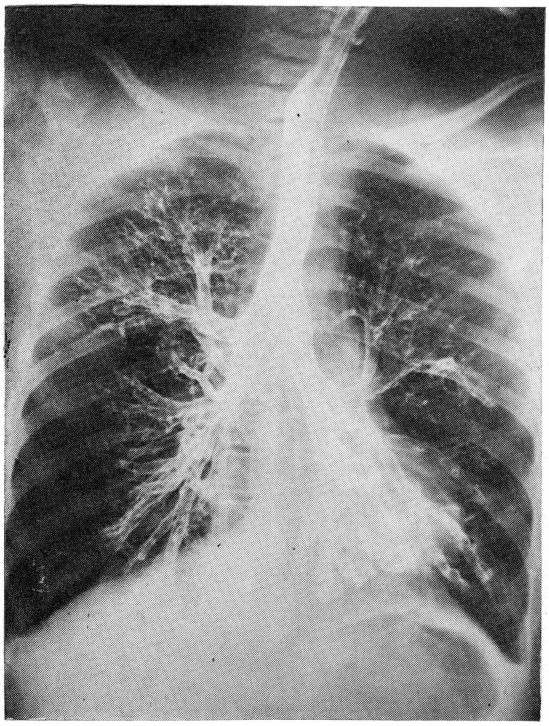

Figure IV. Bronchiectasis in the left lower lobe and lingula The hilar lymph nodes were tuberculous.

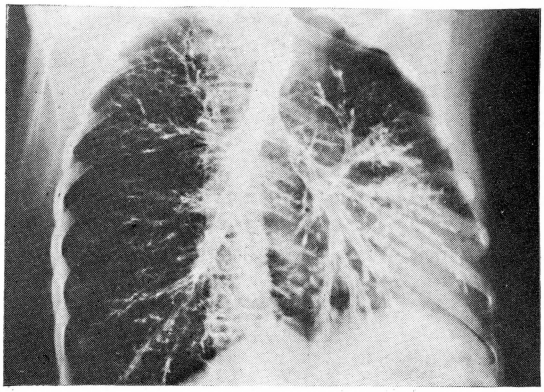

Figure V. Stricture of the left main bronchus due to ulceration by infection from caseating tuberculous lymph nodes.

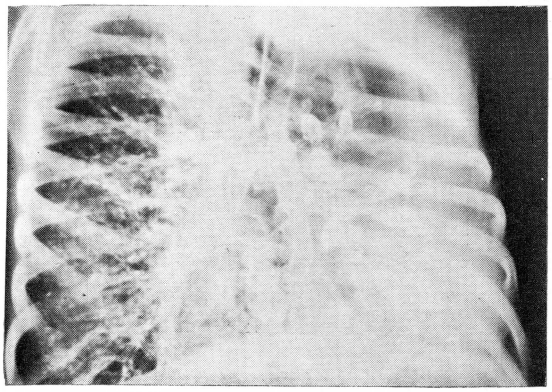

Figure VII. So called "destroyed lung" most often due to stenosis of the left main bronchus by duberculous hilar lymph nodes.

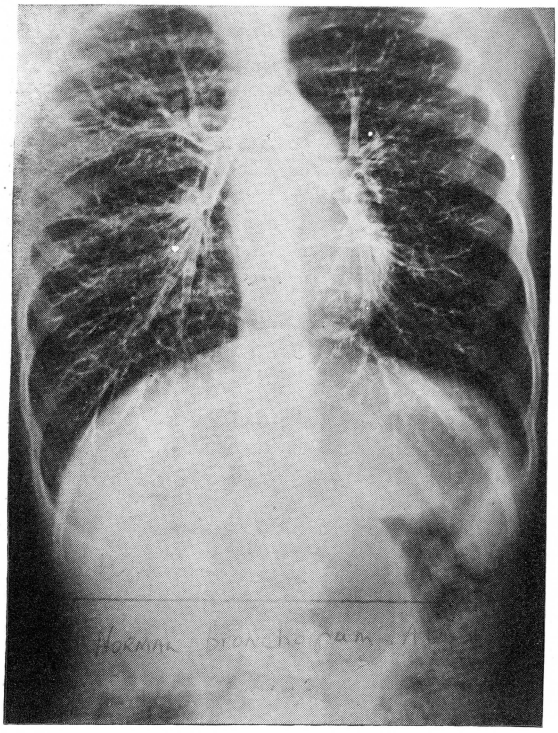

Figure VI. Stricture of the right intermediate bronchus due to compression by caseating tuberculous lymph nodes.

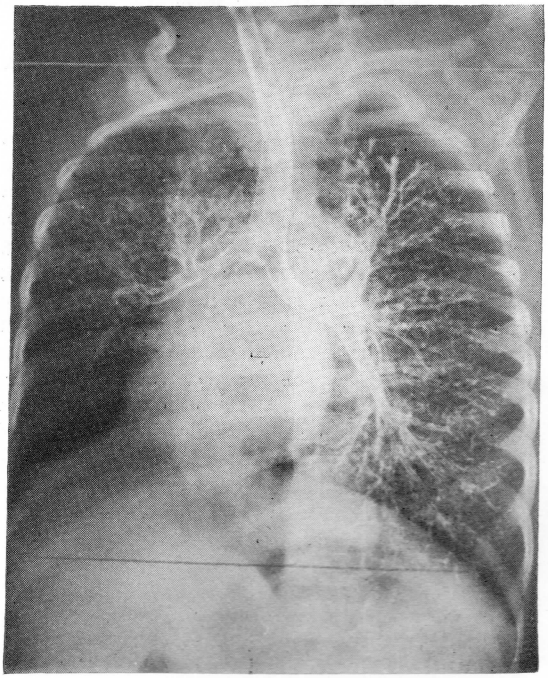

Figure VIII. The left main bronchus in a normal bronchogram. It is narrower and four times as long as the right main bronchus.

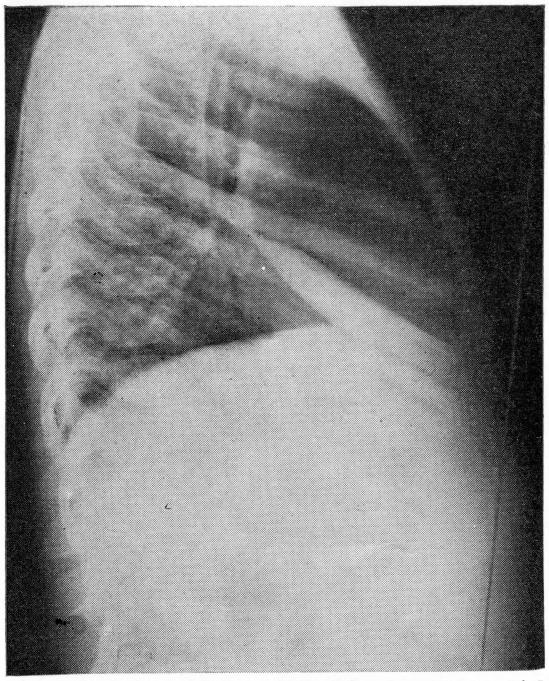

Figure II. Collapse of the middle lobe due to bronchial obstruction by enlarged tuberculous lymph nodes

additional factor at play. It is also well to remember that what appears to be a 'healed primary tuberculous complex' is not necessarily so for reactions around even calcified lesions in lung parenchyma or in the lymph nodes in the hilum can occur and do so after the lesion has been quiescent for a considerable numbers of years.(1)

The appearances in Figure III of "radiographic

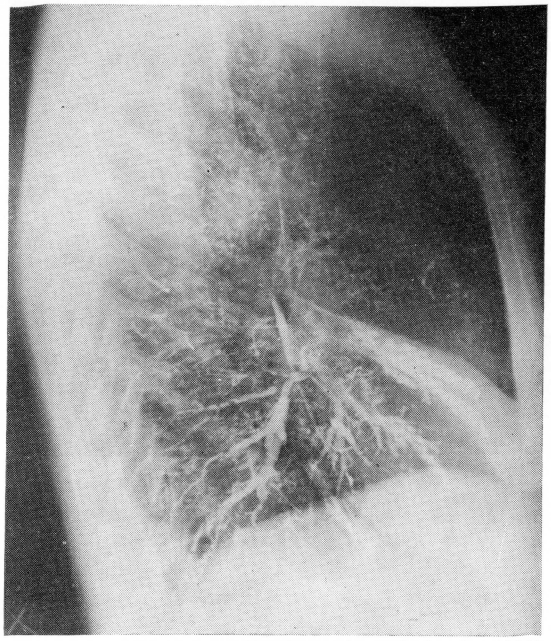

Figure III. "Radiographic" bronchiectasis in the middle lobe. This, as bronchiectasis elsewhere, is reversible in the early stages

bronchiectasis" can be explained as due to either the distension of the bronchi after the damming up of mucus or a compensatory mechanism for the loss of volume of the collapsed lobe.(5). Whatever may be the true explanation, in the early stages, before infection of retained secretion with consequent mucosal ulceration, submucosal inflammation and weakening, erosion and distortion of the bronchial wall **the changes are reversible** provided the bronchial obstruction is relieved.

In many instances adequate drug therapy together with adequate physiotherapy will achieve the re-expansion of the collapsed lung tissue if they are promptly instituted. If time is lost in starting treatment the condition becomes irreversible. Moreover, in not a few instances, the glandular component of the primary complex may go on enlarging in spite of drug therapy.(6)(7)(8). I have watched the development of bronchiectasis over a period of eighteen months in such a patient under bronchographic control. She subsequently required a lobectomy in addition to hilar gland resection for the protection of the remaining lobe. Measles is a well known factor enhancing the risk of bronchial obstruction in children with primary tuberculous infections.(9).

The anatomical distribution of bronchiectatic lesions is of great interest and is further evidence for the role of bronchial obstruction by enlarged lymph nodes in their etiology. Brock(10) has written about the disposition of the lymph glands around the middle lobe bronchus rendering it liable to compression as a result of disease and enlargement of the sentinel gland. "As the very acute angle formed by the origin of the middle lobe bronchus from the main stem begins to widen it is occupied by a lymph gland **as is usual with all such angles formed by the branching of bronchi" (My underlining).** Herein lies an explanation of the common distribution of bronchiectasis—middle lobe, left lower lobe and lingula—all fed by bronchi branching at very acute angles. This is in marked contrast with the distribution of lung abscess which predominantly involves segments of the lungs fed by bronchi originating at 90° or more from the parent bronchi.(11). The acute angle predisposes to compression of the bronchi by the enlargement of the lymph node wedged in the dependent angle.

The "destroyed lung" syndrome (Figure VII) or "chronic fibroid lung" (12) is tragically common in young Africans and usually it is not, as it is often stated to be, the result of extensive "adult" or "fibrocaseous" pulmonary tuberculosis. There may indeed be a focus of old tuberculosis to be found in the resected specimen but I believe that this is only the pulmonary component of a primary tuberculous complex and that the extensive bronchiectatic lesion of the lung is due essentially to the mechanically obstructive effects of the accompanying lymph node component.

Here again anatomical considerations are of great value. Figure VIII is a normal bronchogram in a child and it demonstrates the vulnerability to compression of the left main bronchus by reason of its length, comparatively small lumen and its course between the unyielding aortic arch on the one hand and the left

tracheo-bronchial lymph nodes on the other. When these nodes enlarge the left main bronchus is at risk and sure enough "destroyed lung" occurs on the left side in nine cases out of ten. It is clear that the right main bronchus by contrast with the left is shorter, wider and relatively unfixed.

When caseating tuberculous lymph nodes become adherent to the wall of a bronchus and the disease process invades it ulceration and **stricture formation** may occur. This process is illustrated in Figures V and VI. Attempts at the removal of caseous material or calcific matter through a bronchoscope are fraught with danger uniformly unsuccessful and usually make matters worse. (3)(12). Obviously when the lung beyond the stricture is permanently damaged resection is indicated but there is a time when the surgical removal of the diseased lymph nodes by thoracotomy can save valuable lung tissue from destruction. (13)(14)(15).

Surgery in the Prevention of Pulmonary Tuberculosis in Children.

The final point I want to make in discussing the surgical aspects of pulmonary tuberculosis in African children takes us back to first principles. Prevention is preferable to having to cure. It seems to me questionable that it is wise to persist with antituberculous drug therapy alone beyond six months in the face of persistent cavities in adult patients in developing countries. These patients become the reservoir of infection in the community. I do not refer to those confined to hospital with gross bilateral disease but rather to those who become "the good chronics" and are "treated" as out patients. The term "good chronic" is too often a euphemism for "fit positive tuberculotic"(16) and his numbers increase with a rising standard of living. Surgical resection offers a valuable measure(17) of rendering such patients less menacing to the uninfected children of the community. (18).

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