

BRONCHIECTASIS AND ATELECTASIS : TEMPORARY AND PERMANENT CHANGES

BY

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INTRODUCTION

The introduction of iodized oil as a contrast medium in radiology by Forestier and Sicard made it possible for the first time to demonstrate dilated bronchi in the living body. Thus the diagnosis of bronchiectasis could be made with certainty during life and was no longer dependent on post-mortem verification. Cases of bronchiectasis became apparent which did not show the classical symptoms of long-continued ill-health, cough, and copious sputum, with clubbing of the fingers and signs of disease at the bases of the lungs. Wall and Hoyle (1933) were able to recognize cases of "dry bronchiectasis." The intimate association between atelectasis of the lung and bronchiectasis was recognized and commented on by many authors—Singer and Graham (1926), Anspach (1939), Warner and Graham (1933), and Warner (1934). Finally, cases of bronchiectasis were diagnosed within a short time of their onset, and subsequent restoration of the bronchi to normal calibre was shown (Findlay, 1935 ; Jennings, 1937 ; Lander and Davidson, 1938a ; Fleischner, 1941 ; Ogilvie, 1941 ; and Blades and Dugan, 1944).

This paper presents four cases of atelectasis with bronchiectasis in which subsequent re-expansion of the lung was attended in three cases by return of the bronchi to normal, and in the fourth by an alteration in the bronchographic appearances with a persistence of bronchiectasis. The experimental work which has a bearing on this problem is reviewed, and an attempt made to explain some of the anomalies and misunderstandings which have arisen.

Case 1.—C. G., aged 12½ years, attended Brompton Hospital complaining that for three months he had had cough and thick, rather offensive sputum. At first he had been febrile, with cough and thick sputum, and had remained in bed for one month. Then the fever settled and the cough improved but did not completely disappear. A radiograph at this time showed a diffuse patchy opacity at the right base consistent with an incomplete atelectasis of the right lower, and possibly of the middle, lobe.

A bronchogram on Sept. 19, 1939 (Fig. 1) showed dilatation and bunching together and some shortening of all the bronchi of the right lower and middle lobes. On his first attendance at the hospital the boy looked healthy, his colour was good, and there was no wasting. There was slight clubbing of the fingers. Examination of the chest showed some diminution of movement over the right lower thorax, with dullness and coarse râles in this area both anteriorly and posteriorly. The patient was admitted to Brompton

Hospital. On admission, temperature was 98.6° F., pulse and respiration rates were normal, and sputum amounted to 1 oz. daily. Blood-count was within normal limits. A radiograph showed a condition identical with that previously described. Throughout his six weeks' stay in hospital he was afebrile except for two very short spells of three days each, when his temperature rose to 99° F., and on each occasion his sputum increased from a trace to 1 oz. daily. He was treated by breathing exercises and postural drainage.

The radiograph taken three weeks later showed clearing of the base of the right lung, and the diaphragm was a full space lower. Bronchogram three days later, Jan. 10, 1940 (Fig. 2), showed a marked alteration. The bronchi of the right middle lobe were almost normal; one bronchus only in this lobe remained dilated, and there was no bunching of the bronchi. The bronchi of the right lower lobe showed a very different appearance; they were much lengthened and no longer bunched, but they remained dilated in the middle portions of their length, the upper and terminal portions being of normal calibre. In the next five months the patient had three further bronchograms performed with no change from these appearances.

Subsequent reports over the next four years were that the boy remained perfectly well, played Rugby football for his school, and took part in a seven-mile steeplechase. He did not attend the hospital again, and there has been no news of him for the last two years.

Comment.—The infection, which was coincident with the collapse, appears from the duration of the fever and the presence of purulent offensive sputum to have been a fairly virulent one. The collapse was still present after three months, but in the course of the next three weeks it cleared. The persistence of bronchial dilatation after the collapse had cleared suggests that the infection had permanently damaged the bronchi, rendering them incapable of return to normal calibre.

Case 2.—S. T., aged 12 years, had no previous history of chest illness. On Jan. 4, 1940, he had an operation for nasal obstruction. The lungs were then normal. Two days after operation cough and a high temperature developed. The whole of left side of the chest was dull on percussion and air entry was absent. The heart and trachea were displaced to the left. On Jan. 16 he was transferred to the Royal Free Hospital. There was some cyanosis and the temperature was 103° F. The same physical signs persisted. Radiographs showed the left side of chest to be obscured, the heart being displaced to the left.

On Jan. 19, 1940, a bronchogram showed that the left main bronchus had become obstructed and iodized oil did not penetrate beyond the division into upper and lower lobe branches.

On Jan. 30 the patient had become afebrile, and a bronchoscopy was performed. The left main bronchus was almost completely obstructed by swelling of mucous membrane. Pus was secreted copiously from the left main bronchus; suction was applied and the mucous membrane cocaineized, and then iodized oil was introduced. Radiography then showed that the left upper lobe had aerated, but the left lower lobe showed bronchiectasis and collapse (Fig. 3).

On Feb. 22 there was no cough or sputum and no abnormal physical signs. A bronchogram (Fig. 4) showed that the bronchi in the left lower lobe were now normal. The child remained well after this. There were no abnormal physical signs in the chest and radiographs of the lungs remained normal. He was last seen in 1941.

Comment.—The collapse in this case was of short duration, not more than seven weeks, but was associated with a virulent infection producing a high continued fever. After the main bronchial obstruction had been relieved, atelectasis

persisted for a time in the lower lobe. At this time the bronchi in this atelectatic lobe were shown to be grossly dilated. After the lung had become completely re-aerated, the bronchi returned to normal calibre, without evidence of residual changes.

Case 3.—A. A., a soldier aged 26 years, was admitted to a military hospital in June, 1943, with a temperature of 102° F., pulse-rate 100, respiration-rate 28 per minute. He had been feeling ill for one week with pyrexia, headache, and malaise. On admission he was complaining of pain in his right chest, cough, and sputum. There was dullness at the base of the right lung with absent air entry. Sputum examination showed pneumococci and streptococci. No tubercle bacilli were found. Leucocyte count was 14,000 per c.mm. Radiography showed an opacity at right base. He was treated with sulphapyridine (amount unknown) and the temperature subsided.

On June 23, 1943, temperature, pulse, and respiration were normal. Cough and sputum persisted. There was dullness with loud bronchial breathing over both right lower and right middle lobes with many râles.

On July 2, 1943, he was admitted to Brompton Hospital complaining of cough, worse on lying down, with about two drachms of inoffensive, thick green sputum daily. The fingers were not clubbed. There was dullness over the base of the right lung anteriorly and posteriorly, with bronchial breathing and râles. Radiograph showed a high right diaphragm, displacement of the heart and mediastinum to the right, and an opacity at the right base suggesting collapse. Leucocyte count was 14,000 per c.mm., with 70% polymorphs.

On July 7, 1943, a bronchogram (Fig. 5) showed the bronchi of the right lower and middle lobes to be dilated.

On July 17, 1943, a bronchoscopy was performed. The right main bronchus was inflamed and contained mucopurulent secretion. The middle lobe orifice was larger than normal, and the bronchus contained mucopus. The left bronchial tree was normal.

It was then decided to remove the middle and lower lobes on the right side, and as a preliminary measure an artificial pneumothorax was established and silver nitrate was introduced into the pleural cavity. The intrapleural pressures were —16—8, changing after 250 c.cm. air had been introduced to —8—4.

On Aug. 18, 1943, a radiograph showed clearing of the shadow at the right base, under the pneumothorax, though some atelectasis was still present. The artificial pneumothorax was maintained for two months, and a further injection of silver nitrate was given on Sept. 20. During this period the patient was afebrile except for the days immediately following the injections. His symptoms had subsided, and he had only a trace of sputum.

On Sept. 17 radiography showed that the opacity at the right base had completely disappeared, and in November a further bronchogram (Fig. 6) was performed, and all the previously dilated bronchi were shown to be normal.

Comment.—The collapse was attended by a severe infection, as shown by the continued fever and leucocytosis. It was present from June 1, possibly a few days before, until some time between August 18 and September 17—that is, between eleven and fifteen weeks. A pneumothorax had been established seven weeks after the onset of the collapse. The part played by this measure in the re-aeration of the lung and the prevention of permanent bronchiectasis is arguable. It is feasible, however, that a reduction of the negative intrapleural pressure produced by the pneumothorax may have loosened the tug on the small obstructions in the finer bronchi and allowed their ejection and consequent aeration of the lung.

Case 4.—J. Y., a girl aged 10 years, had had a hacking cough every winter for eight years. She had had whooping-cough as an infant, and pneumonia with haemoptysis two years ago. She was brought to Brompton Hospital on Mar. 4, 1931, on account of the winter cough. There was dullness at the base of the left lung with coarse râles. Radiography showed the outline of a collapsed left lower lobe. Her weight was 4 st. 6½ lb. There was no clubbing of the fingers.

On July 13, 1931, she was admitted to Brompton Hospital. Her weight was 4 st. 5 lb. and the signs in the chest were unaltered. Slight clubbing of the fingers was present. A bronchogram on July 20 (Fig. 7) showed fusiform dilatations of bronchi at the left base. The temperature rose to 99° F. on several occasions in one month during her stay in hospital. Postural drainage and breathing exercises were given. On return from convalescence on Jan. 2, 1932, her weight was 5 st. 5 lb. and there were no symptoms and no abnormal signs in the lungs.

In Oct., 1932, the Mantoux reaction was negative up to 1 in 1,000. Radiographs no longer showed the left lower lobe collapsed. She was seen again in March, 1934, when she had had a cough for three weeks. Râles were still present at the left base. Her weight was 6 st. 4 lb. A bronchogram at this time was difficult to interpret, but the bronchi of the left lower lobe appeared to be normal. After this, she was seen at infrequent intervals. She attended several times after "colds" when she had a cough each time; no râles were discovered at the left base.

In March, 1946, she was seen again; there were no symptoms and she felt perfectly well. The weight was 9 st. 2 lb. There were no abnormal physical signs, the left base being free from râles. Bronchograms showed the bronchi of the left lower lobe within normal limits, though they were of great calibre than those of the right lower lobe (Fig. 8).

Comment.—In this case, even an approximate date for the re-expansion of the left lower lobe and the return of the bronchi to normal cannot be given. It can be stated, however, that the lobe was collapsed for at least four months. The patient was afebrile from the start and the evidence for the presence of any infection was slight.

DISCUSSION

Early theories of the pathogenesis of bronchiectasis were many and varied; most were based on the idea of a weakening and destruction of the elastic tissue of the bronchial wall, and a few only were compatible with a reversible process. One theory postulated the destruction of the bronchial wall and ulceration of the surrounding lung tissue with a relining of this abscess cavity by epithelium, a process clearly incompatible with recovery; this theory still finds adherents (Lisa and Rosenblatt, 1943).

Clinically it has been long recognized that bronchiectasis is accompanied by alveolar dilatation (emphysema) and displacement of the thoracic boundaries and of the thoracic viscera, and that in the majority of cases there is no obstruction of the main bronchus to the bronchiectatic area. Any theory which attempts to explain the process of bronchial dilatation must also explain these associated phenomena.

The theory that bronchiectasis is one of the compensatory mechanisms called into play by the shrinkage of part of the lung is based on two fundamental

FIG. 2.

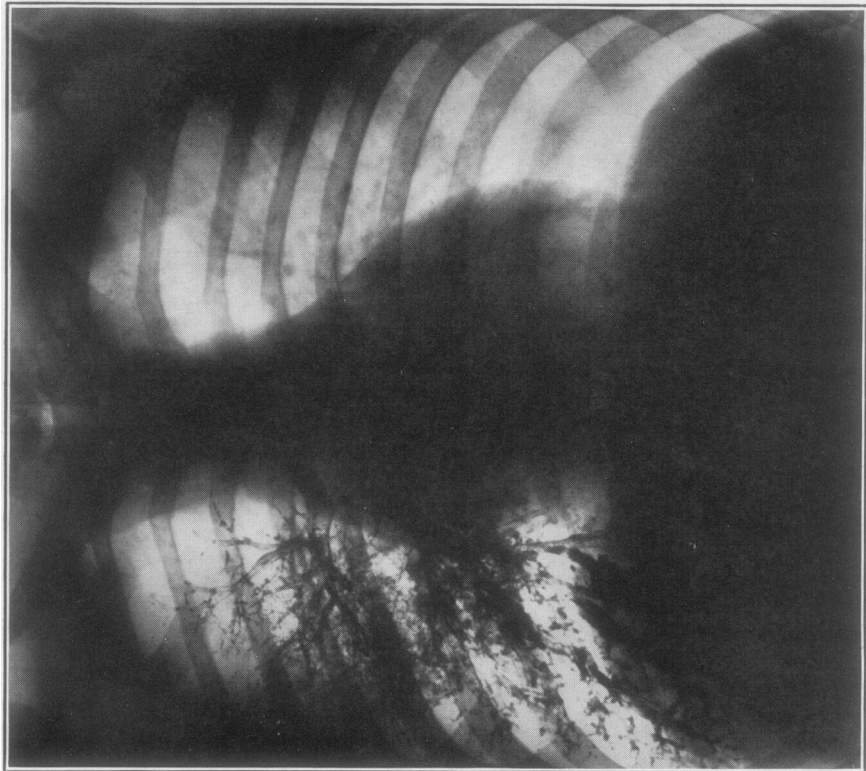


FIG. 1.

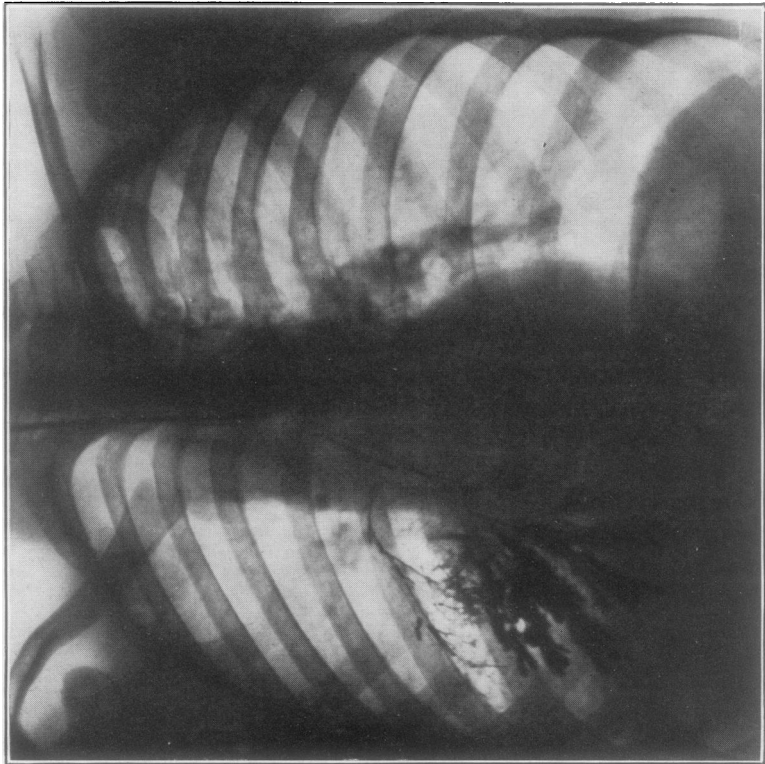


FIG. 4.

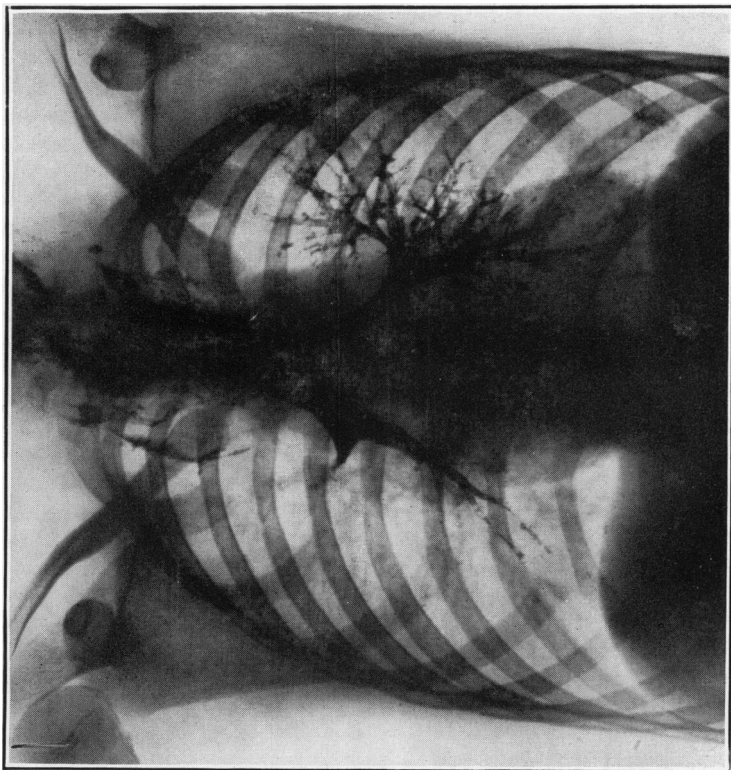


FIG. 3.

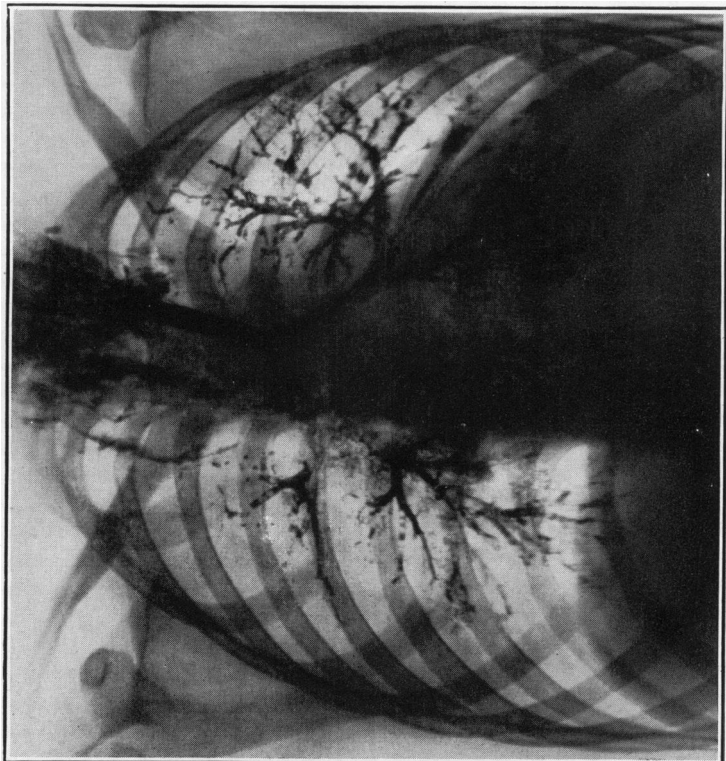


FIG. 6.

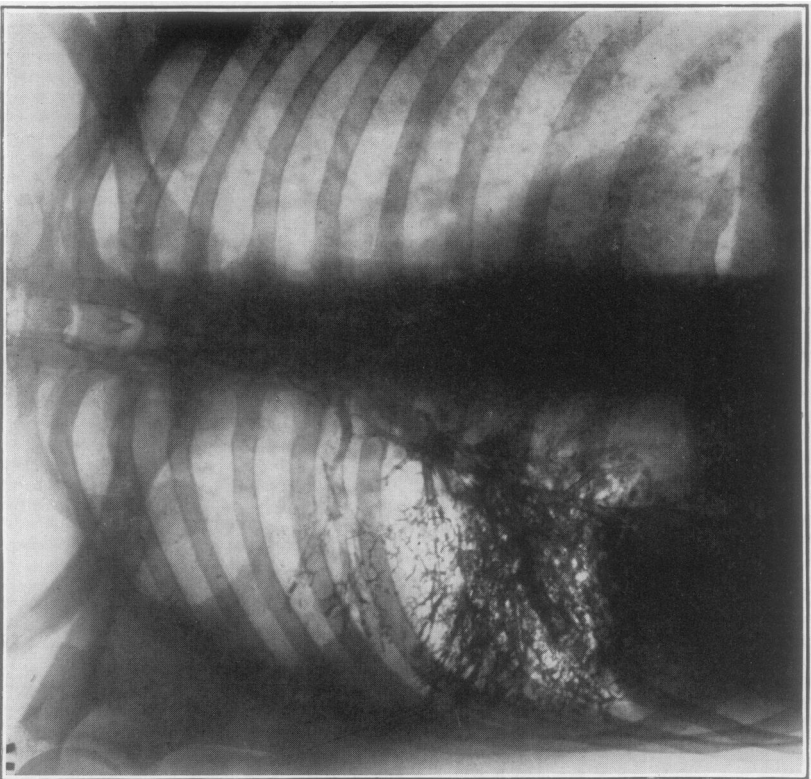


FIG. 5.

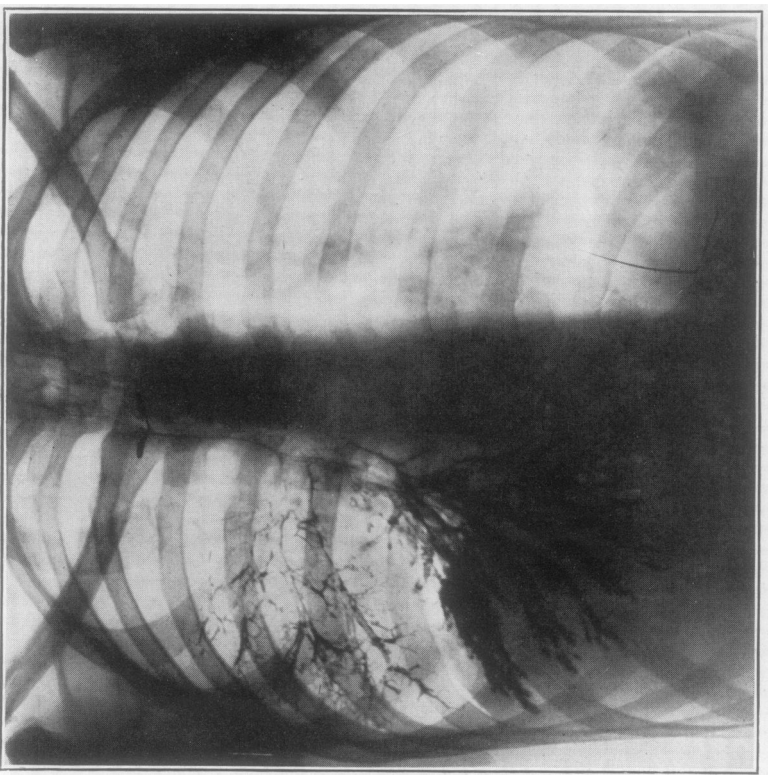


FIG. 8.

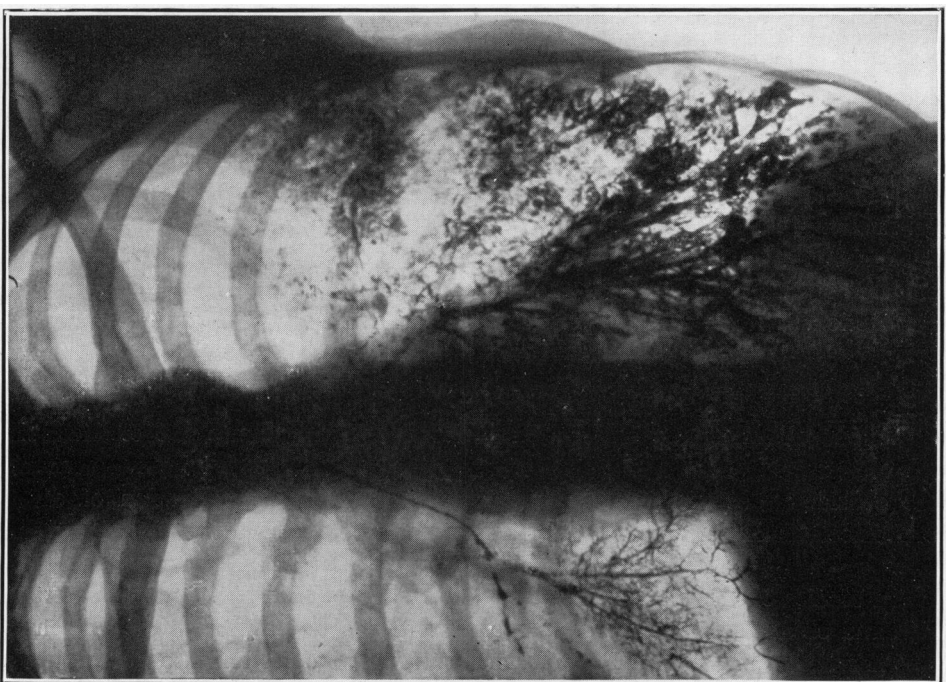
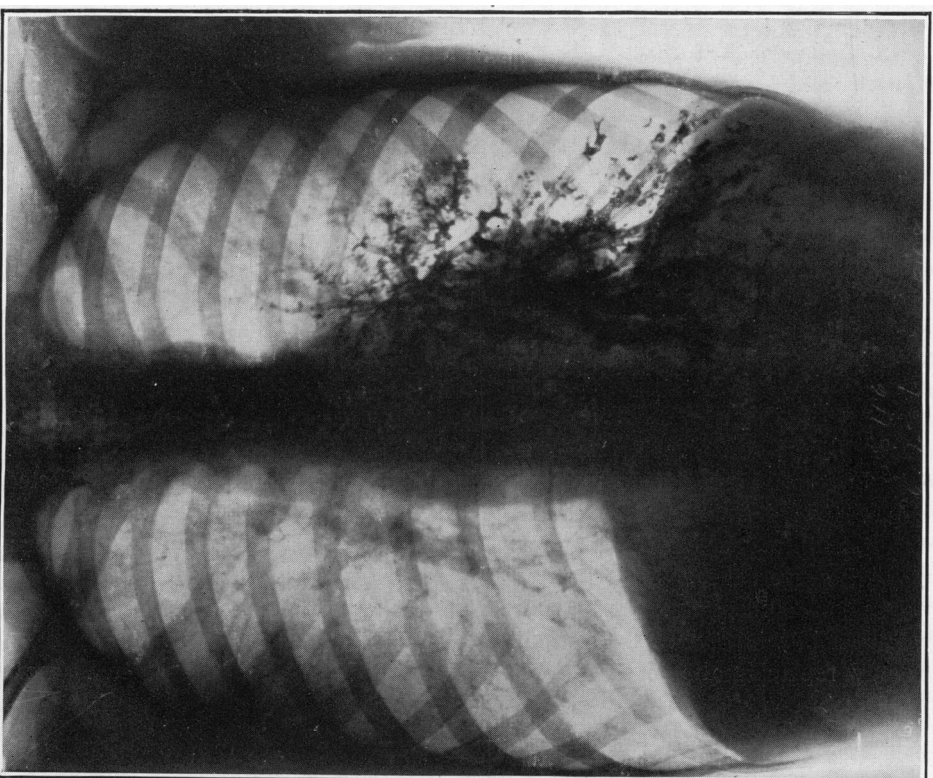


FIG. 7.



concepts. Firstly, that the shrinkage of the lung is occurring in a closed cavity, and consequently compensation for this shrinkage has to occur (i) by a decrease in volume of the closed cavity and (ii) by an increase in volume of the unaffected portion of the lung. Secondly, the "shrinking portion" of the lung consists of all the air-containing portion of the lung (bronchi and alveoli) distal to the obstruction.

Experimental studies on the relation between bronchiectasis and atelectasis

Much experimental work on the aetiology and pathogenesis of bronchiectasis has been done in recent years, some of it vitiated by failure to take into account the considerations outlined above. For instance, the demonstration of early bronchiectasis in experimental animals has been attempted from post-mortem studies and not by iodized oil injection on the living animal.

In studies on the relation between atelectasis produced by bronchial obstruction and bronchiectasis, the type of obstruction and its position in the bronchial tree has varied in the different experiments. These studies fall into two groups; those in which the main bronchus was permanently obstructed, and those in which the obstruction was finally distal to the main bronchus.

Studies in which the main bronchus was permanently obstructed

Adams and Escudero (1938), and Tannenbergs and Pinner (1942), obstructed the main bronchus by means of a lead shot or ligation of the bronchus, both methods producing an immobile obstruction.

In an obstructed system, air distal to the obstruction is absorbed by the pulmonary circulation and cannot be replaced from any outside source. As the air is absorbed, the pressure in that system drops. If the system were a perfectly rigid one, the pressure in it would drop until the same level of gaseous partial pressures was reached as existed in the pulmonary circulation, and then no more absorption of air would occur. The broncho-pulmonary system, however, is an elastic one; and as the air in an obstructed bronchus is absorbed and the pressure tends to fall, the elasticity of the bronchial walls allows of their coming together, and this shrinkage in volume maintains the intrabronchial pressure at a high level. This process goes on until all the air is absorbed and the bronchial and alveolar walls are in apposition. At the same time as the bronchial walls are collapsing, the mediastinum is moving over to the affected side, the diaphragm is rising, and the unaffected homolateral lung to a larger extent, and the contralateral lung to a lesser extent, are becoming emphysematous.

If, however, the vanishing air is replaced by bronchial secretion, then three things can happen, dependent on the volume of the replacing secretion. Either the bronchi will remain their usual size (in the unlikely event of the secretion exactly replacing the air), or the bronchi will be larger than normal (more

replacing secretion than vanishing air), or the bronchi will be slightly smaller (less replacing secretion than vanishing air).

Tannenberg and Pinner demonstrated in their uninfected animals with main bronchial obstruction that the bronchi distal to the obstruction were completely collapsed, with their walls in apposition. In infected cases they demonstrated bronchial dilatations filled with bronchial secretion distal to the obstruction.

Studies in which the main bronchus was not permanently obstructed

In peripheral bronchial obstruction, similar arguments apply up to a point, but there is a fundamental difference between this condition and main bronchial obstruction; the air in the larger bronchi is proximal to the obstruction and outside the atelectatic system, and consequently the bronchi will behave differently.

The air distal to the obstruction, i.e., in the bronchioli and alveoli, is absorbed; and with this absorption the alveoli and bronchioli contract until finally they are completely or almost completely airless. The bronchi, however, are proximal to the obstruction and are in communication with the outside air; thus they are free to take part in the compensatory measures. That the uninfected bronchus is highly elastic and is capable of complete obliteration with its walls in apposition has been shown by Tannenberg and Pinner's experiment; and, just as the bronchus is capable of complete obliteration, it is also capable of considerable expansion.

In this state the bronchi are dilated in order to occupy space left by the contracting lung. If the thorax is opened and the affected portion of lung or whole lung is removed, then the balance of tensions is upset and the dilated bronchi and the dilated alveoli, if still elastic, can assume their usual extra-thoracic size. If, however, the collapse has been long-standing or infection has led to destruction and fibrosis and has fixed the size of the bronchi, then the dilatation will still be demonstrable post-mortem. Likewise, if the dilatation is fixed by the presence of secretion inside the bronchus behind a permanent main bronchial obstruction, as in Tannenberg and Pinner's experiments, then this dilatation will still persist after removal from the thorax. Adams and Escudero (1938), by incompletely obstructing main bronchi and adding infection, produced a large flow of bronchial secretion. This thick secretion completed the obstruction and could not escape, and with the resultant atelectasis it was sucked down the bronchial tree and at length obstructed the finer bronchi. The state of affairs that then existed was peripheral bronchial obstruction with a patent main bronchus.

Lander and Davidson (1938a) introduced a mobile obstruction, in the form of viscid gum acacia, into the main bronchi of cats. This plug, as a result of the ensuing atelectasis, was sucked down the bronchial tree, split at each bronchial division, and obstructed every branch until finally the obstruction came to rest

in the finer bronchi. Iodized oil was then introduced, and radiography showed dilatation of the bronchi in the atelectatic lobes.

Respiratory changes in calibre of bronchi

Macklin (1929) reviewed the literature on calibre changes in normal bronchi which occurred with respiration. The balance of experimental evidence favoured the view that the bronchi became shorter and narrower with expiration and longer and broader with inspiration. Studies with iodized oil have, since this date, supported this view, though some observers report that the vertically disposed bronchi of the lower lobes in shortening with expiration may, at some stage of the expiratory phase, become broader (Heinbecker, 1927). All these changes, as pictured by bronchography with iodized oil, are not marked, as they show a change in diameter only; bronchoscopic methods, which record volume changes, show the respiratory changes in size of the bronchi more clearly (Ellis, 1936).

Lander and Davidson (1938b) showed that similar though more marked changes occurred with respiration in dilated bronchi. Their cases were selected from subjects whose bronchiectasis, although of long standing, showed few signs of infection. These findings have been challenged. Greenfield (1940) published five cases in which he stated that he was unable to demonstrate changes in calibre of dilated bronchi with respiration. Detailed examination of his published radiographs, however, does not bear out his conclusions. Dilatation is in fact demonstrated in the bronchi in which he stated it had not occurred. In three cases the bronchi are larger in inspiration, and in one case in expiration. In the one case in which no calibre change is visible, careful study of the height of the diaphragm, the angles formed by the rib crossings, and the relation of bronchi to rib crossings, suggests that there was very little change in lung volume between the "inspiratory" and "expiratory" bronchograms.

Further studies to demonstrate the elasticity of dilated bronchi were undertaken by Lander and Davidson (1938a). They showed that if air were introduced into the pleural cavity on the affected side in cases of atelectasis, thus compensating for the shrinkage of the lung, then bronchi which had been dilated assumed a more normal size. This change in calibre was, however, only temporary because, with the absorption of the pneumothorax, the bronchial dilatation will again be manifest if the atelectasis persists.

Tannenberg and Pinner (1942) denied that pneumothorax prevented the occurrence of bronchiectasis. This finding was based on their animals in whom collapse had been produced by main bronchial obstruction. In all their animals in which bronchiectasis was produced, infection had been introduced and the dilated bronchi were filled with secretion—a state of affairs which would not allow of regression of changes that had already occurred. The final and conclusive proof that dilated bronchi may retain their elasticity lies in the demonstration of the return to normal calibre of bronchi previously shown to be dilated.

This was first shown by Findlay (1935), and further cases have since been added by Jennings (1937), Lander and Davidson (1938a), Ogilvie (1941), Fleischner (1941), and Blades and Dugan (1944).

That these reversible cases do not represent true bronchiectasis has been suggested by many authors. Hinshaw and Schmidt (1944) talk of the "illusion of bronchiectasis which may result owing to the foreshortening and apparent widening of the larger bronchi in an atelectatic lobe." Blades and Dugan (1944) dismiss the possibility of reversible bronchial dilatation being true bronchiectasis on the grounds that bronchiectasis is a chronic infective process which is progressive and not reversible. Lisa and Rosenblatt (1943) reject the possibility of reversible bronchiectasis being an early stage of infected bronchiectasis by their adherence to the thesis that bronchial dilatations are the result of ulceration of bronchial wall and lung tissue and relining of the resulting cavities with bronchial epithelium. They reject these reversible cases with the statement "that the diagnosis is clinical (bronchogram) and not pathological. Possibly the bronchogram of a collapsed lobe gives the appearance of bronchiectasis without its actual occurrence." Kornblum (1944), in discussing reversible bronchiectasis, says: "If it can be established that the condition is definite clinical bronchiectasis, then our entire concept of the disease must be revised."

CONCLUSIONS

That there is an intimate relationship between bronchiectasis and atelectasis has been long recognized and commented on. Many theories have been formulated to explain this relationship, and there has been much argument as to the nature of the actual dilating force. Andrus (1937), in a detailed examination of all the physical forces, came to the following conclusions: "These forces (arising as a result of pulmonary atelectasis) provide much the most satisfactory explanation of bronchiectasis"; and "both infection and injury to the bronchial wall and an abnormal intensity of mechanical dilating stress are customarily necessary for the production of bronchial dilatation."

Lander and Davidson (1938a), as the result of experimental work carried out on animals and human subjects, came to the conclusion that infection was not necessary for the production of bronchial dilatation and that the dilating forces consequent on atelectasis were sufficient of themselves. The fact that most human cases had a coincidental infection was not denied. The theory that bronchial dilatation is one of the compensatory mechanisms consequent on pulmonary collapse was advanced and supported by experimental observation. Bronchial dilatation (bronchiectasis) and alveolar dilatation (emphysema) were thus regarded as part of the same process.

It is suggested that all cases of bronchiectasis are, at an early stage of their career, capable of reversion to normal. In the majority of cases in which

bronchiectasis is permanent, this reversion to normal is prevented by a failure of the lung to expand; in a minority of cases the bronchiectasis is maintained, in spite of re-expansion of the lung, by a permanent damage to the bronchial wall.

SUMMARY

Four cases of atelectasis with bronchiectasis are described. In all four cases re-aeration and re-expansion of the collapsed portion of the lung occurred. In three, this was accompanied by regression of the bronchiectasis with return to normal. In the fourth case, the bronchiectasis persisted. The reason for the persistence of the dilatation in the one case is discussed, and the suggestion is made that in this case infection had rendered the dilated bronchi incapable of returning to normal calibre; this may supply a link between reversible bronchiectasis and permanent dilatation. That this is not the usual way in which permanent dilatation is brought about is suggested by a detailed study of cases of bronchiectasis. In most of them persistent atelectasis with attendant crowding together of bronchi accompanies the bronchial dilatation. Damage by infection, as suggested in this case, however, may explain the dilatation that occasionally occurs in cases in which no crowding together of dilated bronchi is seen.

The experimental work on the aetiology of bronchiectasis is reviewed.

It is concluded that bronchiectasis occurs as a compensatory phenomenon when atelectasis takes place in a semi-rigid compartment, the thorax, and because in many cases of atelectasis the bronchi are obstructed peripherally and not centrally.

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