THE RELATIONSHIP BETWEEN ATELECTASIS AND BRONCHIECTASIS

WITH SPECIAL REFERENCE TO WHOOPING COUGH

By

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PREFACE.

Although bronchiectasis has been recognised as a clinical entity for well over a century, and was accurately described by the great Laennec[1] as long ago as 1826, the mode of origin of the condition remains highly controversial. My interest in this fascinating problem was quickly aroused when I joined the staff of Ruchill Sanatorium as Resident Medical Officer in October 1946, for it was then my lot to carry out numerous bronchographic investigations on patients admitted to the observation wards of the institution, and as the West of Scotland Thoracic Surgery Unit was functioning at that time in Ruchill Hospital, the fact that the accuracy of the findings was often checked in the operating theatre gave the work a somewhat dramatic significance.

Experience soon made it plain that bronchiectasis and pulmonary collapse frequently co-existed, and I was much impressed by an article written in 1938 by Lander and Davidson[2], which not only seemed to explain the association of the two conditions, but appeared to go far towards solving the riddle of the genesis of bronchiectasis. Lander and Davidson produced experimental evidence which suggested that pulmonary collapse is commonly caused by aspiration of sputum into the periphery of the bronchial tree, and that when collapse occurs in this manner, mechanical forces are set in motion which are often capable of producing dilatation of the bronchi in the collapsed portion of lung. They contended that bronchiectasis, in the vast majority of instances, arises in this way. Other workers in the same field, however, have come to very different conclusions. Adams and Escudero[3], for

example, who observed the effects of experimentally produced pulmonary collapse in dogs, decided that while collapse might aggravate bronchiectasis it could not cause it, and in 1942, Tannenberg and Pinner[4] who conducted similar experiments in rabbits, stated that pulmonary collapse led to diminution not increase in calibre of the bronchi in the affected area.

Nevertheless, the work of Lander and Davidson appeared so convincing, and their arguments so cogent, that it was interesting to consider the implications of their work. For example, if, as they maintained, pulmonary collapse is commonly caused by aspiration of sputum into the periphery of the bronchial tree, it would be logical to look for the frequent occurrence of the condition in respiratory diseases associated with the production of abundant, viscid secretion, above all in whooping cough, for as Erwin[5] says, "the tenacious nature of the sputum bespeaks collapse." Whooping cough commanded attention in another respect, in that it has long been regarded by clinicians as one of the main progenitors of bronchiectasis; Laennec[1] pointed out the connexion in 1826, and it was also noted by Andral[6] writing about the same time. The relationship has, however, apparently been inferred from case histories; in the rather extensive literature perused, no instance was discovered of bronchiectasis actually having been demonstrated to arise during an attack of whooping cough.

Clearly the work of Lander and Davidson was capable of supplying a scientific basis for the clinical deductions. Whooping cough might be expected to give rise to pulmonary collapse because of the aspiration of tenacious mucus, and the collapse in turn might produce bronchial dilatation.

In order to test this theory, one hundred and fifty consecutive whooping cough cases admitted to Ruchill Fever Hospital were carefully investigated.

This study proved to be interesting beyond expectations; not only was pulmonary collapse noted in a large number of cases, but bronchography revealed that when the condition was extensive, the bronchi contained in the collapsed portion of lung were apparently markedly dilated. Moreover, it was noted that if a collapsed portion of lung re-expanded, the apparent bronchial dilatation disappeared. This latter phenomenon has been noted by Fleischner,[7] Ogilvie,[8] Lander[9] and other authors, and has been termed by some "reversible bronchiectasis."

Fleischner[7] claimed that it was an early stage of true bronchiectasis, and Lander and Davidson[2] went so far as to claim that all cases of bronchiectasis associated with pulmonary collapse were potentially reversible "even after years."

On the other hand, workers such as Lisa and Rosenblatt[10] (1943) and Blades and Dugan[11] (1944), have denied that so-called "reversible bronchiectasis" can properly be regarded as bronchiectasis at all.

The discovery of such interesting and controversial material in the course of the whooping cough investigations, naturally further stimulated my interest in the factors responsible for the causation of bronchiectasis, and a critical review of the literature on the subject was undertaken. At the same time, investigations were carried out on cases of pulmonary collapse and bronchiectasis admitted to the observation wards of Ruchill Sanatorium or the wards of Ruchill Fever Hospital, and also on cases sent for bronchographic examination to the radiological

department from city chest clinics.

As a result of these studies, the conclusion was reached that pulmonary collapse may give rise to bronchial dilatation, which, in the course of time, is likely to become permanent.

A review of the literature, however, failed to reveal any method of distinguishing potentially "reversible" bronchiectasis from "true", classical or "irreversible" bronchiectasis, and as surgical removal of bronchiectatic lung tissue is now everyday practice, it was felt that it was highly desirable to supply this deficiency, in order to avert the possible excision of apparently permanently damaged lobes which might well return to normal if left to themselves. A test was eventually evolved which appeared to go some way towards satisfying this requirement.

The conception that bronchiectasis is at first purely a mechanical phenomenon arising as a result of abnormal intra-thoracic tensions produced by pulmonary collapse, suggested that certain therapeutic measures, if applied at a sufficiently early stage of the disease, might prevent the bronchial dilatation from becoming permanent. Clearly, the ideal treatment would be one which promoted prompt re-expansion of the collapsed portion of lung, but unfortunately measures directed towards this end, including attempted bronchoscopic aspiration of obstructing mucous plugs, have in practice proved disappointing, and I came to the conclusion that in cases in which re-expansion of the affected lung tissue could not speedily be brought about, the induction of an artificial pneumothorax of calculated extent might prove of value.

I should like to emphasise that I considered that this form of treatment was likely to be beneficial only

when carried out at that early stage of bronchiectasis when there was reason to believe the condition was potentially "reversible"; I was well aware that it had many times been unsuccessfully employed in old-standing cases of the disease.

During the course of the investigations on the causation of bronchiectasis certain findings were made with regard to the effects of pulmonary collapse which were apparently at variance with those of other observers. The dynamics of pulmonary collapse was therefore considered in some detail in order, if possible, to explain these discrepancies.

As the subject matter of this monograph is somewhat extensive, the order in which the work is presented in the following pages may briefly be indicated.

The thesis is divided into six chapters.

In the first, the methods employed in the whooping cough investigations are described, and the results of the study are set forth in the second. The causation of bronchiectasis is discussed in the third chapter, and the role played by pulmonary collapse in this connexion is particularly studied. Evidence brought to light in the whooping cough experiment, supplemented by material obtained from other sources, is considered in estimating the validity of the various theories which have been advanced from time to time. The fourth chapter comprises an investigation of the dynamics of pulmonary collapse, an attempt to discover a test for distinguishing between potentially "reversible" and permanent bronchiectasis, and a study on the efficacy of artificial pneumothorax as a therapeutic agent in potentially "reversible" bronchiectasis. The treatment of the different varieties of

bronchiectasis is discussed in the fifth chapter, and a brief review of the thesis is contained in the sixth.

The work embodied in this monograph was carried out between October 1946 and June 1948, during which period I was Resident Medical Officer to Ruchill Sanatorium, and later Senior Resident Medical Officer to Ruchill Fever Hospital, and I am happy to take this opportunity of expressing my appreciation for the help I received from so many quarters during its progress.

I should like above all to thank Mr. R. S. Barclay, Superintendent of Ruchill Sanatorium and Thoracic Surgeon to Mearnskirk Chest Unit, for the constant interest he displayed in the investigations throughout their course, for his ever ready encouragement, and for placing so freely at my disposal the benefit of his expert knowledge of diseases of the chest.

I am much indebted to Dr. T. Anderson, Senior Lecturer on Specific Fevers to Glasgow University, for the keen interest he took in the whooping cough investigations, and for the ready kindness with which he gave me the benefit of his great experience.

The study of the dynamics of pulmonary collapse was largely a matter of physics, and my thanks are due to Professor T. Alty, formerly of the Chair of Natural Philosophy, Glasgow University, for being good enough to read the proofs of this part of the work.

The late Dr. Fergus Henderson was good enough to give me his advice on problems of radiology, and I am happy to acknowledge the debt I owe him.

I should like to thank Dr. W. Elliott, Superintendent of Ruchill Fever Hospital, for his encouragement in pursuing these investigations, and for facilitating access to relevant material.

Dr. J. H. Lawson, Deputy Superintendent of Ruchill Hospital was good enough to read through the proofs of the work, and I have pleasure in recording my gratitude for many valuable suggestions.

In conclusion, I should like to thank the nursing staff of Ruchill Fever Hospital and Ruchill Sanatorium for their valued co-operation, and I am particularly grateful to the sister in charge of the radiological department for shouldering with such cheerfulness the additional burdens created by my frequent incursions into her territory.

CONTENTS.

VOLUME 1.

CHAPTER 1.

PLAN OF THE WHOOPING COUGH EXPERIMENT, MATERIALS AND METHODS.

It has been explained that there was reason to believe that whooping cough might be a frequent progenitor of pulmonary collapse, and that pulmonary collapse in its turn might give rise to bronchial dilatation. These suspicions were duly investigated, and the methods employed in this undertaking will now be discussed.

Incidence of Pulmonary Collapse in Whooping Cough.

The plan to discover the frequency with which pulmonary collapse occurs in whooping cough was simple. One hundred and fifty consecutive whooping cough cases admitted to Ruchill Hospital were x-rayed at weekly intervals until radiological and clinical recovery took place. If any indication presented itself for x-raying the case on any additional occasion, this was done.

The interval of one week was arbitrarily chosen. Tannenberg and Pinner[1] have shown that in dogs complete collapse of a lobe following experimental bronchial occlusion can take place in three hours, and no doubt re-expansion can occur with equal celerity. If it may be inferred that somewhat similar time factors apply to the human subject, under Utopian conditions three hourly radiograms would have been taken during this experiment. In every day life, however, scientific idealism has to make concessions to material limitations, and in any case, for all practical purposes, x-ray at weekly intervals was doubtless adequate; pulmonary collapse which clears up in

less than that time is interesting, but the likelihood of its having any consequences of moment is somewhat remote.

Since the object of the experiment was to gain a reasonably accurate picture of the frequency of pulmonary collapse in whooping cough, no selection was made from the cases admitted. That, of course, is not to deny that a selective element might have been present in the type of case sent to the hospital. It might be thought that as whooping cough is commonly nursed at home, only severe cases would have presented themselves. The hospital, however, serves a very poor quarter of the city, and owing to the often appalling home conditions, general practitioners are naturally desirous of giving any child, no matter how slightly ill, the benefit of hospital attention. The consequence was that most of the cases were not unduly grave, and they can be regarded as fair samples of whooping cough in the population.

Diagnosis of Whooping Cough.

Diagnosis of whooping cough was based upon the child having the typical spasms of coughing and whoop. Doubtful cases were discarded whether they had a suggestive blood picture or not. Culture of H. pertussis was not attempted.

Nomenclature.

Before passing to a discussion of the diagnosis of pulmonary collapse, it is advisable to clarify the question of nomenclature. From the introductory remarks made in the preface, and from the context in which the term has occurred, it will have been obvious that "pulmonary collapse" has signified the diminution in lung

volume which takes place when air is absorbed from the alveoli. The term "pulmonary collapse", however, is sometimes applied to the state of relaxation or compression of lung tissue resulting from the presence of air or other material in the pleural cavity.

In order to avoid confusion, many writers refer to pulmonary collapse brought about by absorption of alveolar air as "absorption collapse" or "massive absorption collapse", while others refer to it as "atelectasis". Strictly speaking, "atelectasis"should only be employed to indicate a congenital failure of lung tissue to expand, but it is now so widely accepted as meaning collapse of lung due to intrinsic causes, that it is somewhat pedantic to insist on the restriction.

In the following pages, "atelectasis, "absorption collapse", "pulmonary collapse", and "collapse of lung" are used as interchangeable terms. When the lung is diminished in volume as the result of the presence of air or other material in the pleural cavity, it is described as being relaxed or compressed as the case may be.

Diagnosis of Pulmonary Collapse.

The clinical and radiological signs indicative of pulmonary collapse, are so well known that it might appear superfluous to give an account of them here. Nevertheless, in a work in which the condition is to be so often discussed, it is felt that some description of the evidence upon which diagnosis was based is necessary.

The cardinal clinical features are impairment of the percussion note over the collapsed portion of lung,and displacement of neighbouring structures in the direction of the affected hemithorax.

When there is complete collapse of a lung, diagnosis based upon a consideration of the physical signs alone is generally easy. There is dullness over the atelectatic lung, and evidence of displacement of the heart and trachea towards the affected side of the chest. In the case of collapse of a lower lobe, the well known para-vertebral triangular area of dullness can be detected, and displacement of the heart may be observed; tracheal deviation is not present. When collapse of an upper lobe occurs there is impairment of the percussion note over the affected area and tracheal deviation may be noted; the heart is not displaced.

Breath sounds may or may not be heard over the collapsed lung tissue. If the collapse is due to obstruction of the main bronchus supplying the affected area, the respiratory murmur is absent, and fluid may be suspected. Aspiration results in a "dry tap". If, on the other hand, the collapse is caused by bronchiolar obstruction, the larger bronchi lying in the atelectatic zone are patent, and weak tubular breathing is usually heard. A few crepitations may also be detected, and the condition is liable to be mistaken for pneumonic consolidation.

In both varieties of collapse, even in the apparent absence of infection, pleural friction may occasionally be present over the affected area, presumably as a purely mechanical phenomenon.

It is important to remember that it is generally impossible to diagnose collapse of the right middle lobe by clinical methods alone, as there is no mediastinal displacement, and owing to compensatory emphysema of the right upper lobe the percussion note is not impaired.

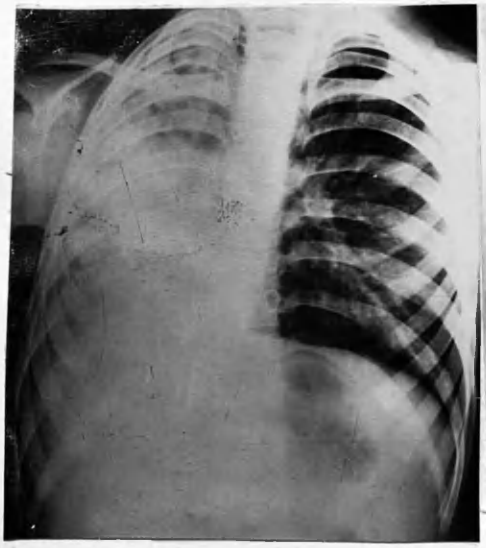
The same considerations apply to the corresponding lingular process of the left upper lobe. It is likewise impossible clinically to detect minor degrees of atelectasis in the upper and lower lobes, because emphysema of the surrounding sound pulmonary tissue is sufficient to redress the effects of the loss of lung volume, and consequently compensatory dislocation of neighbouring structures does not occur. The emphysema masks also the other physical signs of atelectasis.

Apart from the liability to overlook minor or even major degrees of atelectasis, clinical diagnosis is unsatisfactory in another respect; in certain circumstances it is particularly easy to mistake collapse for pneumonic consolidation. It has been noted that the physical signs of the two conditions may resemble one another, and in the febrile stage of whooping cough, for example, the difficulty of distinguishing atelectasis from broncho-pneumonia when displacement of the heart or trachea is too slight to be detected is obvious, while if broncho-pneumonia or bronchitis co-exist with atelectasis, the likelihood of confusion is still further increased.

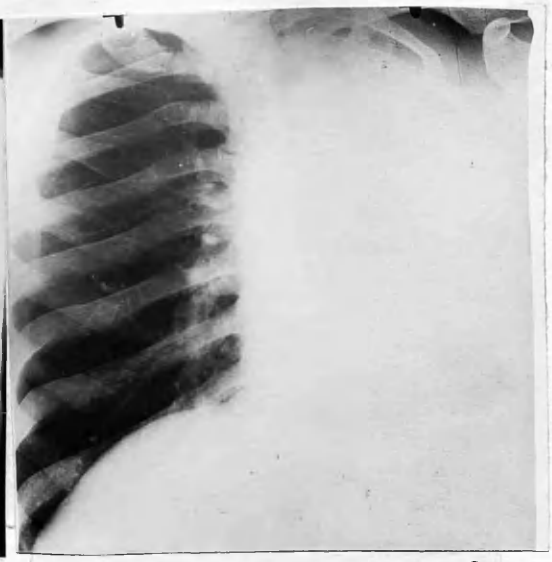
It was therefore decided in the whooping cough investigations to make radiological evidence the diagnostic criterion of pulmonary collapse, though the necessity for frequent and systematic clinical examinations was not overlooked.

The radiological features of pulmonary collapse will now be considered.

The cardinal signs are the homogeneous shadow with its often characteristic shape and position, and compensatory changes such as shift of the mediastinum and trachea to the affected side of the chest, and elevation of the



Case 1. Collapse of rt. lung. Marked mediastinal shift to right.



Case 2. Collapse of left lung. Marked mediastinal shift to left.

diaphragm. Twining[2] observed that peripheral to the collapsed area there is often emphysema of the lung, and suggested that this phenomenon is an ancillary aid to diagnosis. As Martin and Berridge[3] point out, however, a similar appearance can often be seen on full inspiration, and thus the sign cannot be relied upon.

When there is complete collapse of a lung, there is a homogeneous opacity in the affected side of the chest resembling a pleural effusion, but the mediastinal and tracheal shift to the affected side, and the isolateral elevation of the diaphragm make the diagnosis easy.

Case 1.

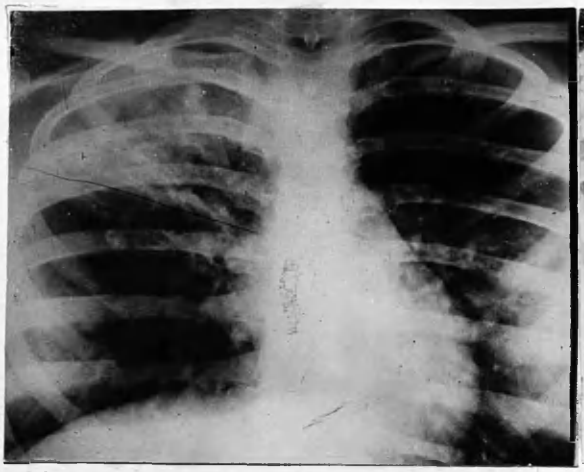
Female aged 5 years. Seen at a tuberculosis clinic on 4/2/46, and later admitted to Ruchill Sanatorium. Complete atelectasis of the right lung. History of whooping cough in 1944 with persistent cough thereafter. Clinically, tubular breathing, dullness on percussion over the affected lung field, and evidence of shift of the mediastinum to the affected side. The straight x-ray shown opposite reveals the features noted above.

Case 2.

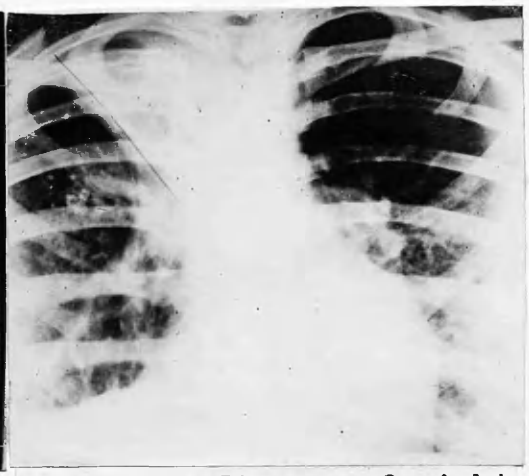
Male aged 35 years. Admitted to Ruchill Hospital on 6/9/47 with "pneumonia". Complete atelectasis of the left lung occurred on 14/10/47. Bronchoscopy on 20/11/47 revealed that the collapse was due to blockage of the left main bronchus by ulcerating tuberculous glands. Clinically, dull to percussion, and absent R.M. over the affected area, with mediastinal shift to the affected side. A radiogram is shown opposite.

In the case of collapse of the right upper lobe, the mediastinal shift is seen most clearly by the movement of the trachea to the affected side. The heart is little displaced, and the diaphragm unaffected.

The lower border of the lobe swings upwards and inwards, and in postero-anterior view is generally slightly concave. The lateral view has been likened to a gull in



Case 3. Tuberculosis right upper lobe.



Case 3. Collapse of right upper lobe. Upward swing of lower border.

flight, with the hilum representing the body of the bird.

Case 3.

Female aged 20 years, admitted to Ruchill Sanatorium on 21/1/46 with bilateral pulmonary tuberculosis. Atelectasis of the right upper lobe noted on routine radiographs. The photographs opposite show the collapse as evidenced by the upward swing of the lower border of the lobe. Clinically, tubular breathing and dullness on percussion over the affected area. Tracheal shift to the right.

Collapse of the left upper lobe, exclusive of its lingular process differs from collapse of the right in radiological appearance only in that it has often not the same sharp definition of its lower border.

Diagnosis of right middle lobe collapse is more difficult than in the case of the upper and lower lobes, as A. Lisle Punch[4] pointed out in an interesting article in 1940 which may be briefly summarised.

The middle lobe is wedge shaped, and lies between the lesser fissure and the lower part of the greater fissure. In a postero-anterior view in a radiogram, the area in which the middle lobe lies can be indicated by drawing a line from the midsternum, at the level of the fourth costal cartilage, horizontally outwards to the lateral margin of the thorax, and joining the outer end of this line to a point on the sixth rib in the midclavicular line. In the lateral radiogram, the middle lobe area can be marked out by drawing a line horizontally from the sternum, at the level of the fourth costal cartilage, backwards to approximately the centre of the radiogram, and joining the central end of this line to a point on the diaphragm about two inches behind the sternum. The wedge shaped area thus delineated, corresponds to the middle lobe.

It is therefore clear that an opacity in the middle lobe area, seen in a postero-anterior view may be either

in the middle lobe or in the lower part of the lower lobe. A lateral view is essential in order to demonstrate clearly in which lobe the opacity is situated.

If atelectasis takes place, the middle lobe collapses inwards and backwards. The greater fissure, which forms the posterior border of the middle lobe, remains stationary, but with the shrinkage of lung tissue the lesser fissure is shifted inwards, downwards, and backwards. In a postero-anterior radiogram, therefore, the atelectatic middle lobe is represented by a small triangular area close to the right border of the heart, lying well internal to the lateral border of the chest wall; it lies above, and at no point reaches down to the diaphragm.

In the lateral radiogram, the collapsed middle lobe appears as a somewhat oval area in the centre of the zone normally occupied.

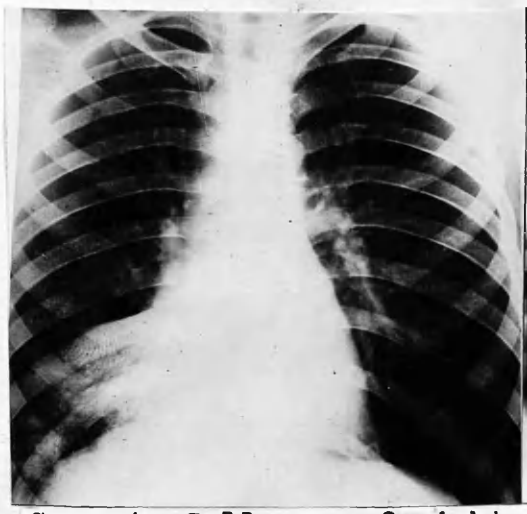
As the upper lobe enlarges by compensatory emphysema, and fills the space which has been left by the shrinkage of the middle lobe, no clinical signs may be apparent to give a clue as to what has happened. Shift of the mediastinum does not occur, and elevation of the diaphragm, if it takes place at all, cannot be detected in a straight x-ray. Diagnosis therefore rests on the typical shape and position of the homogeneous opacity seen in the radiogram.

Punch[4] apparently considered that postero-anterior and lateral films were adequate for diagnosis, but it may be mentioned that a lordotic view often excellently demonstrates any abnormality of the right middle lobe.

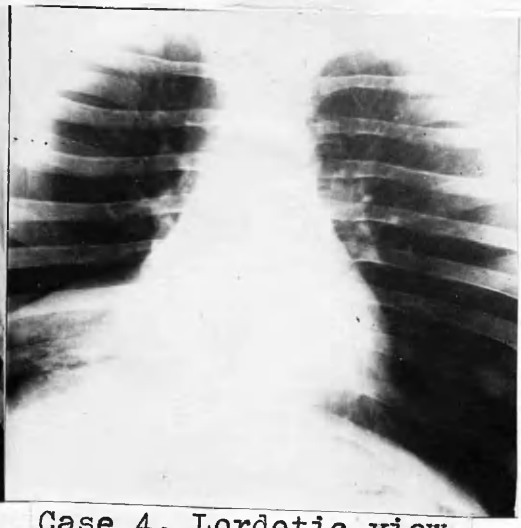
The condition is comparatively common, and two of many examples are shown.

Case 4.

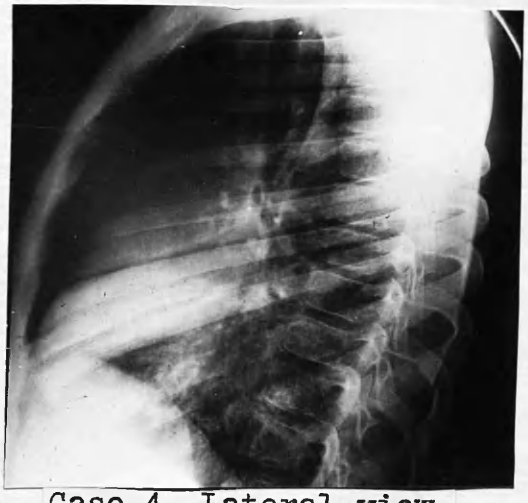
Male aged 12 years. Cough and pain in right side on



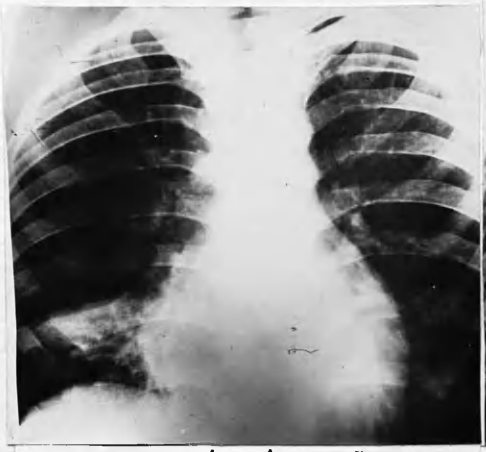
Case 4. Collapse of right middle lobe. P.A. view, 21/8/47.



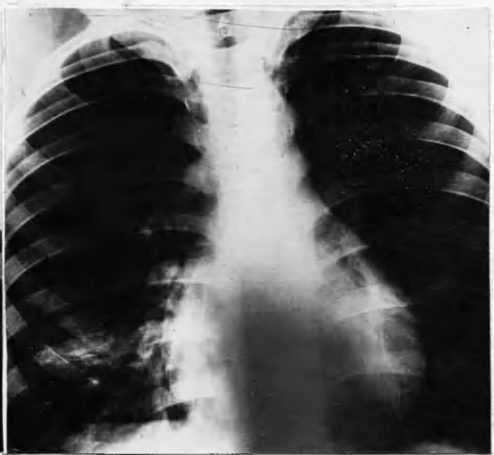
Case 4. Lordotic view, 21/8/47.



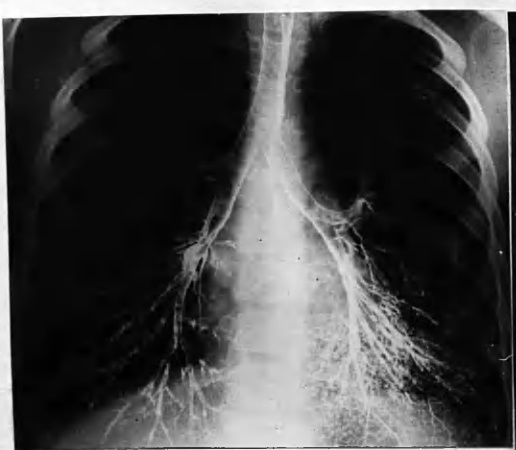
Case 4. Lateral view, 21/8/47.



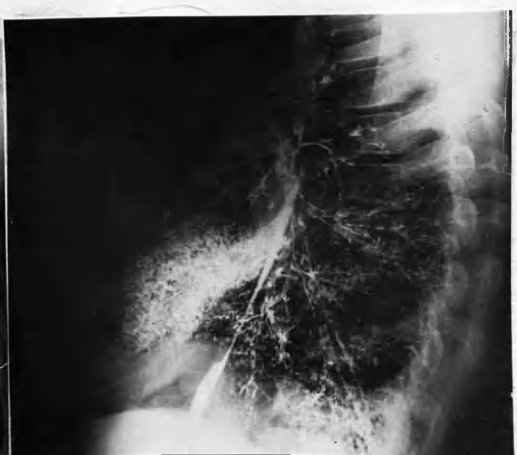
Case 5. 9/12/47. Collapse of rt. middle lobe.



Case 5. 12/12/47. Right middle lobe practically re-expanded.



Case 5. 12/12/47, P.A. bronchogram. No bronchial dilatation in rt. middle lobe.



Case 5. 12/12/47. Lat. bronchogram. Alveolar filling obscures rt. middle lobe bronchi in photograph, but in actual bronchogram it was plainly seen the bronchi were not dilated.

13/9/47. Admitted 20/8/47. X-ray on 21/8/47 showed collapse of the right middle lobe. The case is more fully discussed in a later chapter. Clinically, the only signs were occasional crepitations and slightly prolonged and tubular breathing over the affected lobe. Postero-anterior, lateral and lordotic radiograms are shown opposite.

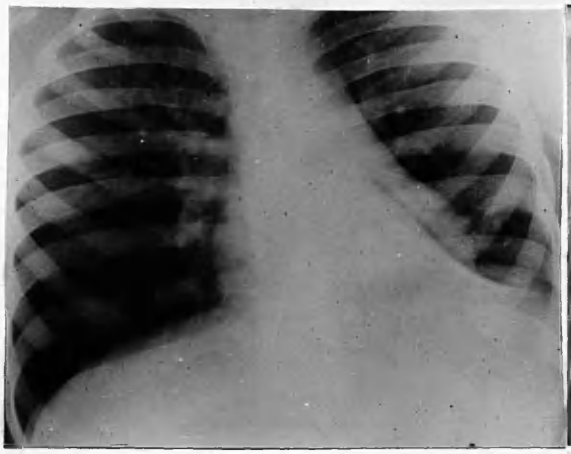
Case.5.

Male aged 16 years. "Pneumonia" right lung in May 1947, with pain in the right side, which recurred from time to time, accompanied by slight cough. When admitted to hospital on 8/12/47, the patient was symptomless. Clinically, slightly prolonged expiration with a tubular element heard over the right middle lobe area. P.N. and V.F. and V.R.were normal. X-ray on 9/12/47 showed collapse of the right middle lobe. Another film on 12/12/47 demonstrated that the lobe had re-expanded. Bronchography on the same day revealed a normal bronchial tree. The case is illustrated opposite.

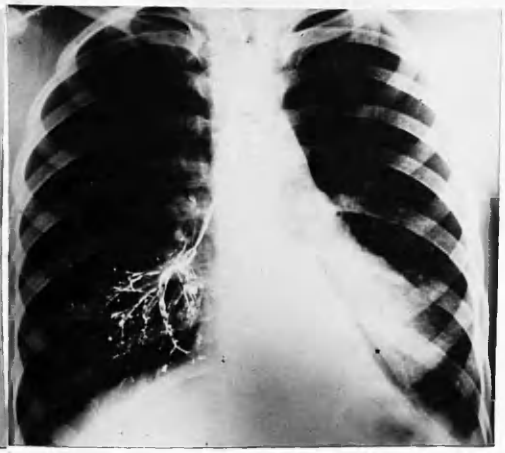
Diagnosis of collapse of the lingular process of the left upper lobe is based on the same considerations as apply to the right middle lobe. Although the lingula is very often involved in collapse in association with the left lower lobe, it is rather infrequently affected alone. No instance was discovered in the fairly large number of cases investigated.

Collapse of a lower lobe is indicated by a triangular or "jib-shaped" basal shadow. The heart in lower lobe collapse is displaced to the affected side, though, as will be maintained in a later chapter, in old standing cases this feature may not be much in evidence.

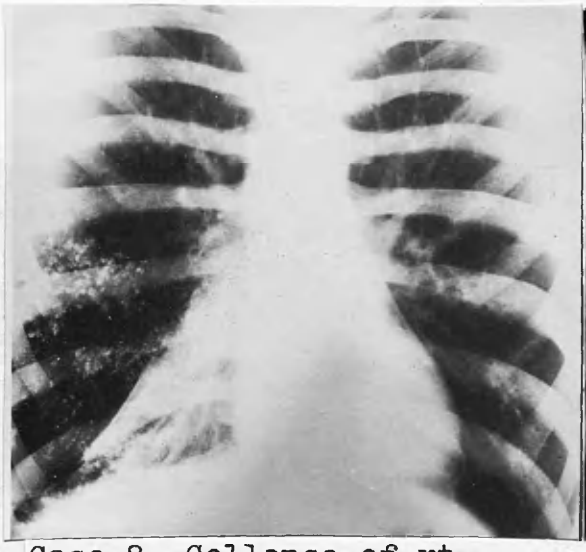
Lower lobe collapse is easy to diagnose radiologically, the only difficulty being that the oblique outer border of the triangle, when the left side is affected, lies behind the heart shadow in a postero-anterior view, and greater penetration than usual is necessary for its demonstration.



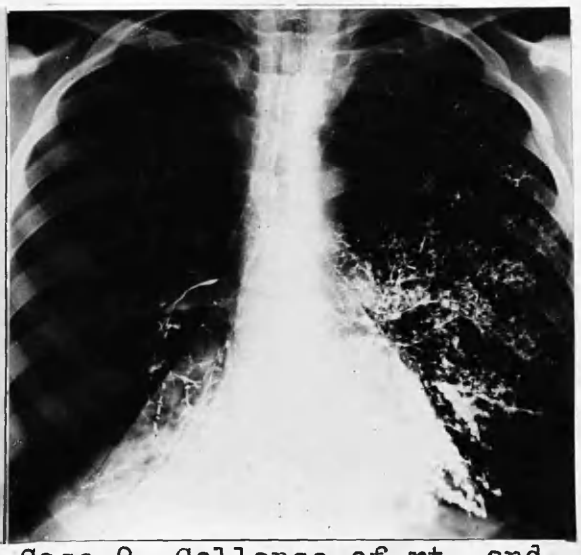
Case 6. Collapse of left lower lobe. "Jib shaped" shadow behind heart shadow.



Case 7. Collapse of left lower lobe. "Jib shaped" shadow behind the heart shadow.



Case 8. Collapse of rt. lower lobe. Typical triangular shadow. There is some old neohydriol in rt. lung.



Case 9. Collapse of rt. and left lower lobes, represented by triangular shadows. The bronchogram shows that the bronchi in the collapsed lobes are dilated.

Case 6.

Male aged 8 years. Admitted to Stobhill Hospital on 12/3/46, with complete collapse of the left lung and later transferred to Ruchill Sanatorium. The upper lobe re-expanded, and the radiogram opposite, taken on 23/3/46, shows only collapse of the left lower lobe. The case is more fully discussed later.

Case 7.

Female, aged 5 years. Admitted to Ruchill Sanatorium on 10/1/47. Radiology revealed collapse of the left lower lobe, and a film is shown opposite. The case is more fully discussed later.

Case 8.

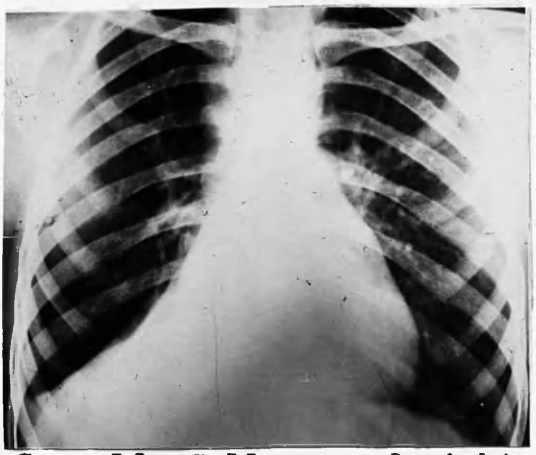
Male, aged 25 years. Admitted to Ruchill Sanatorium on 22/4/47 with collapse of the right lower lobe. Traces of old lipiodol are seen in the film shown opposite. The case is fully discussed later.

Case 9.

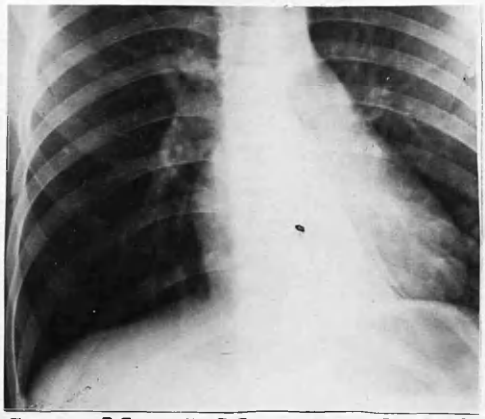
Male, aged 9 years. Seen as an out-patient on 12/5/47. History of cough and spit for months. The bronchogram reproduced opposite demonstrates atelectasis of the right lower lobe, and also of the left lower lobe.

More than one lobe may be collapsed at the same time. The commonest combinations are collapse of the left lower lobe and lingular process of the left upper lobe, collapse of the right lower and middle lobes, and collapse of the right and left lower lobes.

The previous case illustrates collapse of both lower lobes. The following two cases illustrate the typical appearances on the straight x-ray of right middle and right lower lobe collapse, and collapse of the lingula and left lower lobe. Lateral films of these two cases were not taken, but the diagnosis was confirmed by postero-anterior and lateral bronchograms. The advantages of bronchography for the accurate localisation of pulmonary collapse are discussed below.



Case 10. Collapse of right middle and lower lobes represented by triangular shadow.



Case 11. Collapse of left lower lobe and lingular process of left upper lobe. The typical triangular shadow can just be discerned behind the heart shadow.

Case 10.

Female, aged 6 years. Admitted to Ruchill Sanatorium on 12/5/47. Collapse of right middle and right lower lobes. A photograph of the straight x-ray is shown opposite.

Case 11.

Male, aged 14 years. Seen as an out-patient on 11/6/47. Collapse of the left lower lobe and lingular process of the left upper lobe. The radiogram illustrating the condition is shown opposite.

When a lobe is only partially collapsed it is, of course, obvious that mediastinal displacement and elevation of the diaphragm may not occur; it is not to be expected when only a few lobules are involved, that these signs will be present. In cases, therefore, in which pulmonary collapse is small in extent, and physical signs are absent, diagnosis rests solely on the appearance of the opacity in the radiogram, and the only sure interpretation of these shadows is long experience on the part of the radiologist. It was appreciated, therefore, that a high frequency of atelectasis in the cases of whooping cough investigated in this study might be ascribed to the zeal of an enthusiast finding out what no one else would be able to discover, particularly as there does not appear to be a similar study in the literature with which results could be compared. On this account, no radiological phenomenon was recorded as pulmonary collapse unless the diagnosis was confirmed by the routine report of the radiologist. Photographic prints of x-ray films showing small areas of pulmonary collapse are demonstrated in the course of a later section dealing with the classification of atelectasis, but it will readily be understood that as even the radiograms must be first class to ensure confident recognition, the technical difficulties involved in satisfactory reproduction are extreme.

The features of pulmonary collapse on the straight x-ray film have been discussed, and before turning to a consideration of the use of bronchography in demonstrating the condition, it may be of interest to mention that according to Erwin[5], on fluorescent screening the diaphragm is raised and restricted in movement, posteriorly in lower lobe collapse, and anteriorly in middle lobe collapse.

Bronchography is a most important aid in the diagnosis of atelectasis. Not only does it assist in defining the portion of lung involved, but it may throw valuable light on the cause of the condition.

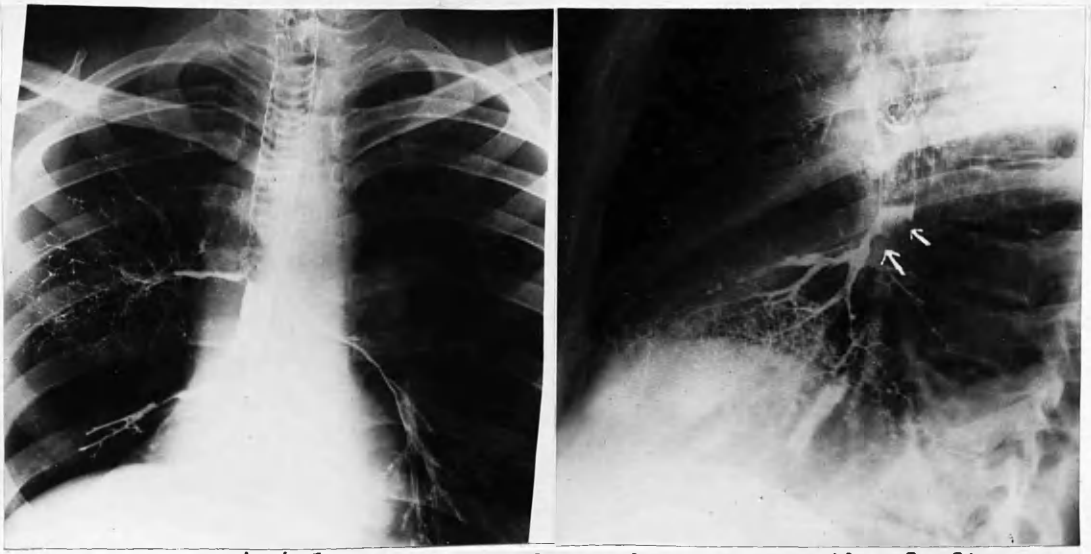
If the collapse is due to complete obstruction of the main bronchus of supply, the iodised oil will fail to enter the atelectatic area, while if the obstruction is not quite complete a trickle of oil may get through, and the narrowing of the bronchus will be demonstrated. In either event the portion of lung involved in the collapse will be indicated.

If, however, the atelectasis is due to occlusion of terminal bronchi or bronchioles, the medium will enter the larger bronchi lying in the collapsed portion of lung. These bronchi will be crowded together, and thus the atelectatic portion of lung will again be defined.

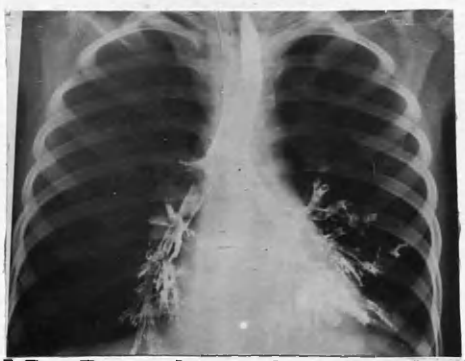
The following two cases illustrate the bronchographic appearances just described.

Case 12.

Male, aged 25 years, admitted to Ruchill Sanatorium on 7/1/46. In July 1942, he had complained of pain and numbness in the right side and arm, and an irritating, productive cough. These symptoms appeared intermittently for months, and in April 1945 there was a febrile episode. In June, 1945, slight haemoptysis took place, and recurred from time to time in the following months. Straight x-ray on admission revealed atelectasis of the right lower lobe, and bronchography on 13/1/46 showed failure of the



Case 12. 13/1/46. The P.A. bronchogram on the left shows collapse of rt. lower lobe, and failure of the radio-opaque medium to enter the rt. lower lobe bronchus. The lateral bronchogram on the right shows complete failure of the radio-opaque medium to enter the rt. lower lobe bronchus, and also a lobulated filling defect of the rt. middle lobe bronchus. The medium has entered the rt. middle lobe, however, and the partial obstruction of its bronchus was not sufficient to cause collapse.



Case 13. P.A. bronchogram 13/5/47, showing collapse of left lower lobe. The contained bronchi are patent up to their extremities, crowded together, and markedly dilated. Contrast appearances with those of case 12 in which the collapse was due not to bronchiolar obstruction, but to obstruction of the main bronchus of the lobe.

medium to enter the right lower lobe bronchus, and also a filling defect of the right middle lobe bronchus. Bronchoscopy on 21/1/46 demonstrated a pedundulated adenoma projecting into the right lower lobe bronchus, and encroaching on the lumen of the right middle lobe bronchus. The patient first came under the care of the author in October 1946. On clinical examination there was a triangular area of dullness at the right base, with diminished V.F. and V.R., and absent breath sounds. The bronchograms shown opposite beautifully display the lobulated filling defect due to the adenoma.

Case 13.

Male, aged 5 years. Became ill on 26/3/47, and later developed whooping cough. He was admitted to Ruchill Fever Hospital on 9/4/47. Physical examination revealed scattered crepitations and rhonchi in both lung fields, and a triangular area of dullness at the left base over which weak tubular breathing and medium moist crepitations could be heard. The apex beat was in the left mid-clavicular line. A provisional diagnosis of whooping cough, broncho-pneumonia, and collapse of the left lower lobe was made. A radiogram taken on 18/4/47 showed broncho-pneumonic infiltration in both lungs, and extensive collapse of the left lower lobe. The collapse persisted. A bronchogram taken on 13/5/47 is shown opposite. It will be observed that iodised oil has freely entered the left lower lobe, and outlined its larger bronchi. Clearly the collapse has not been brought about by obstruction of the main left lower lobe bronchus or its larger branches; it must be the bronchioles or finer bronchi of the lobe which are occluded. The bronchi of the left lower lobe are seen to be crowded together, thus making it obvious that the lobe is atelectatic. It may be noted here that the bronchi contained in the left lower lobe are also markedly dilated; the significance of this fact will be considered in a later chapter when the case is fully discussed. The differences between the clinical findings in this case and the previous one are worth emphasising. The absence of breath sounds over the collapsed lobe in the previous case suggested that the cause of the atelectasis was obstruction of the main bronchus of the lobe, while the presence of weak tubular breathing over the collapsed lobe in this case suggested that the cause of the atelectasis was obstruction of the bronchioles or finer bronchi. In both instances the clinical indications proved reliable guides.

Differential Diagnosis of Pulmonary Collapse.

Collapse of an upper lobe in a child may be mistaken for epituberculosis. Prosoroff[6], Morlock and Pinchin[7], Erwin[5] and others have reported cases of upper lobe collapse in children due to pressure of an enlarged gland on its main bronchus. Canalisation by bronchoscopy cleared up the condition. This is not the place to inquire in to the nature of epituberculosis, but if it is accepted that the phenomenon is an allergic response to a focus of infection characterised by an outpouring of fluid into the pulmonary alveoli, the radiological criteria of collapse will be absent.

As has been mentioned, collapse of a lobe may be mistaken for pneumonia or pleural effusion, and it may also be confused with pleural thickening, but a consideration of the diagnostic criteria for atelectasis already given, eliminates error without undue difficulty.

Classification of Pulmonary Collapse According to Extent.

For many years, the unit of collapse was the lobe, but comparatively recent work in the field of bronchoscopy, and increased accuracy in the interpretation of radiological phenomena, has led to the adoption of the lobule as the unit. Brown[8] has described cases in which x-ray suggested lobular collapse, and bronchoscopic removal of plugs of sputum from the lesser bronchi resulted in clearing of the opacities.

Erwin[5] suggested the term "sector collapse" for atelectasis which involves a main subdivision of a lobe, but admitted that a clear cut collapse of this nature is unusual.

In the whooping cough investigations, pulmonary

collapse was often encountered, but the amount of the lung tissue involved varied from case to case. It was therefore necessary to find some method of conveying to the reader, in words or figures, an idea of the severity of the atelectasis noted. It has been stated that the lobule is now looked on as the unit, but it is not yet possible to indicate the extent of a collapse by stating the number of lobules affected. Only an approximate idea can be given, and for this purpose the lobe has been adopted as the unit, and atelectasis classified as marked, moderate, or slight in extent.

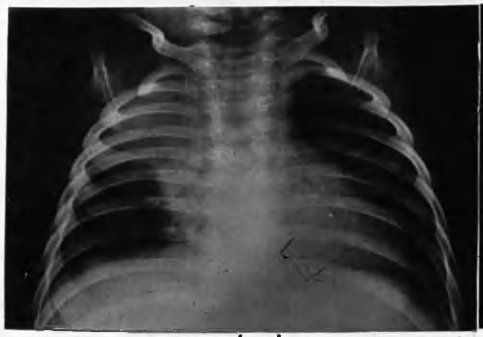
This classification is, of course, purely arbitrary, and adopted merely for convenience in description, and in analysis of the findings of the experiment.

Marked collapse means that at least one lobe is completely or almost completely atelectatic, moderate collapse signifies that a considerable portion of the lobe is involved, and slight collapse indicates that a small but definite degree of lobar atelectasis is radiologically apparent.

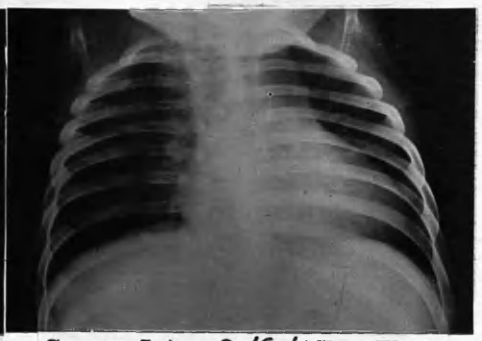
It should be noted that although the last mentioned degree of collapse is small taking the lobe as the unit, it is considerable if the lobule is regarded as the unit.

The following examples, all from the whooping cough series, are representative of the different classes.

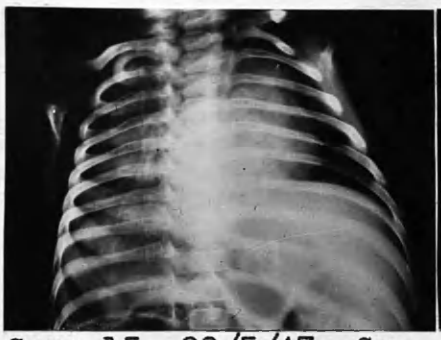
In giving examples of slight and moderate collapse, cases involving the left lower lobe, although it was the one most frequently affected, have been avoided, owing to the difficulty of reproducing satisfactorily the typical shadow behind the heart.



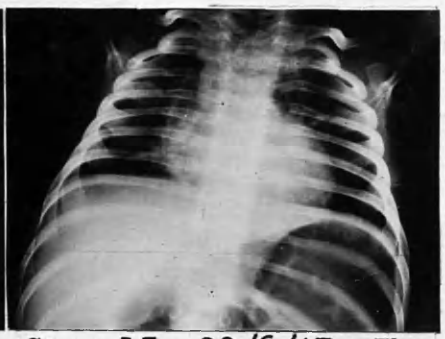
Case 14. 21/2/47. Marked collapse rt. upper lobe.



Case 14. 9/6/47. The collapsed rt. upper lobe has re-expanded.



Case 15. 28/5/47. Complete collapse rt. lung and collapse left upper lobe. As the film was "thin" the shadow of the collapsed rt. lung does not reproduce well, and owing to obliquity of the patient's position when x-rayed, mediastinal shift to the right has been masked.



Case 15. 20/6/47. The collapsed areas have re-expanded.

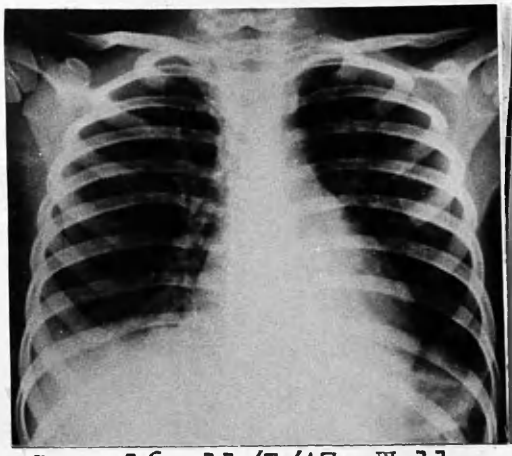
Marked Pulmonary Collapse.

Case 14.

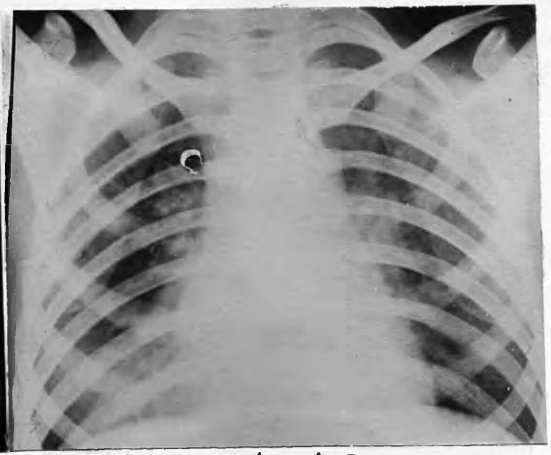
Male aged 10 months. Illness first noted on 25/1/47. Admitted to hospital with whooping cough on 7/2/47. Mantoux negative. Chest clear radiologically; scattered crepitations in both lung fields on clinical examination. X-ray on 21/2/47 revealed collapse of the right upper lobe. Clinically, some impairment of the P.N., and tubular breathing over the affected area. By 5/3/47, the atelectasis was beginning to diminish in extent, but did not clear completely until 9/6/47. Mantoux on 15/6/47 negative. Owing to the tender age of the child, bronchography was not carried out. Radiograms are shown opposite.

Case 15.

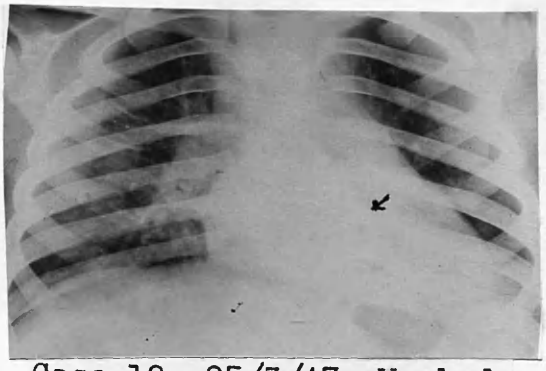
Male aged 11 months. Mantoux negative. Illness noted 14/3/47, and admitted to hospital on the same day with whooping cough. On 26/5/47, the patient became extremely distressed, with rapid respirations and cyanosis. There was dullness on percussion over the right lung, absent R.M., and mediastinal shift to the right. Aspiration resulted in a "dry tap". Temperature 100.F. On 27/5/47, the clinical signs were the same except that weak tubular breathing could now be heard over the affected area. Atelectasis of the right lung was diagnosed. It was concluded that a plug of mucus had blocked the main bronchus of the right lung. The replacement of absent breath sounds by weak tubular breathing on 27/5/47 was accounted for by postulating that the plug of mucus had been drawn to the periphery of the bronchial tree, splitting at the branches until it had finally blocked the finer bronchi or bronchioles, thus leaving the larger bronchi in the atelectatic area patent. Bronchography was not carried out owing to the feeble condition of the patient. X-ray on 28/5/47, demonstrated complete atelectasis of the right lung, and extensive atelectasis of the left upper lobe. The latter condition had been missed clinically. The routine weekly radiograms of the patient had been clear up to the time the extensive collapse was demonstrated. The condition resolved by 20/6/47 when the radiogram was once more clear. Mantoux on 22/6/47 negative. X-rays are shown opposite.



Case 16. 11/3/47. Well marked collapse of both lower lobes. The triangular shadow can be seen in the rt. lung, but it is difficult to see it in the left in the photograph as it lies behind the heart.



Case 17. 25/12/46. Marked collapse involving the rt. middle lobe and the ant. basal segment of the rt. lower lobe.



Case 18. 25/3/47. Marked collapse left lower lobe. The denser triangular shadow of the collapsed lobe can just be seen in the photograph lying in the heart shadow. The outer border of the triangle is indicated by an arrow.

Case 16.

Female, aged 6 years. Illness noted on 26/1/47, and admitted to hospital with whooping cough on 7/2/47. Mantoux positive. Radiogram on 11/3/47 showed well marked atelectasis in both lower lobes. The photograph opposite shows the collapse on the right, but the shadow behind the heart is not well brought out. The case is fully discussed in a later chapter.

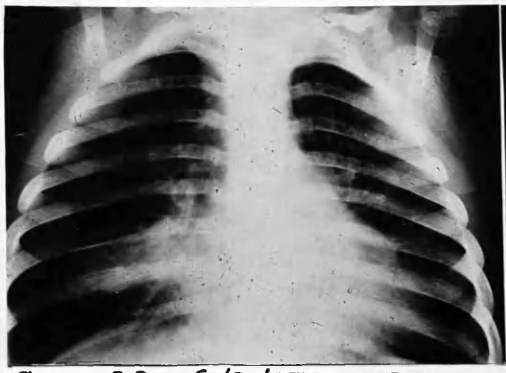
Case 17.

Female, aged 6 years. Mantoux positive. Illness first noted on 12/11/46. Admitted to hospital with whooping cough on 16/11/46. Radiological evidence of slight atelectasis involving the right middle lobe on 4/12/46. The atelectasis later became more extensive, and involved not only the right middle lobe, but the anterior basal segment of the right lower lobe. A radiogram taken on 25/12/46 is shown opposite. The case is fully discussed later and illustrated with postero-anterior and lateral bronchograms.

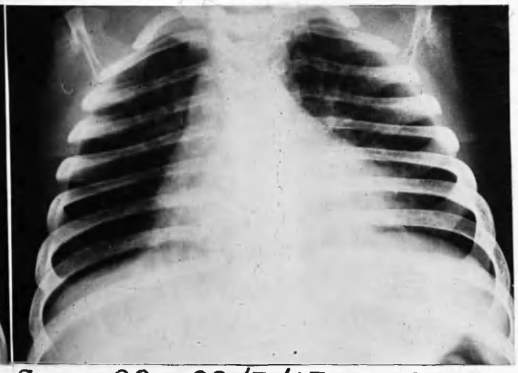
Case 18.

Male, aged 3 years. Mantoux negative. Illness noted on 12/12/46, and admitted to hospital with whooping cough on 16/12/46. A radiogram on 29/1/47 revealed slight atelectasis at the right base. By 4/2/47, this was beginning to clear, but it was noted that atelectasis had developed in the left lower lobe. The right lower lobe cleared up in a further fortnight, but the atelectasis of the left lower lobe became markedly worse. A radiogram taken on 25/3/47 reproduced opposite, showed extensive collapse. The case is fully discussed later, and bronchograms shown. Mantoux negative, 25/6/47.

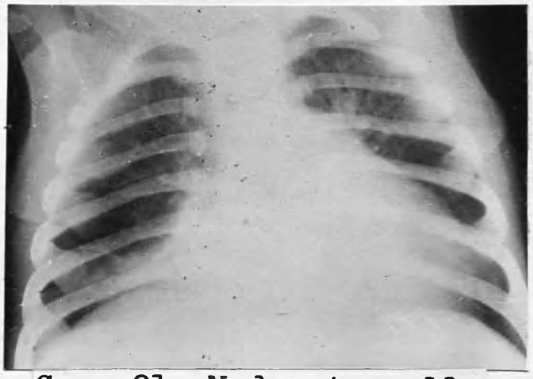
Case 13 was another example of marked pulmonary collapse occurring in the whooping cough series. Brief notes on this case appear on page 13, and it is more fully discussed in a later chapter. A bronchogram clearly indicating the extent of the collapse is shown opposite page 13.



Case 19. 6/2/47. Moderate collapse of rt. middle and left lower lobes. The left lower lobe collapse cannot be seen in the photograph.



Case 20. 28/3/47. Moderate collapse rt. and left lower lobes. Only the rt. lower lobe collapse can be seen in the photo-graph.



Case 21. Moderate collapse right lower lobe.

Moderate pulmonary collapse.

Case 19.

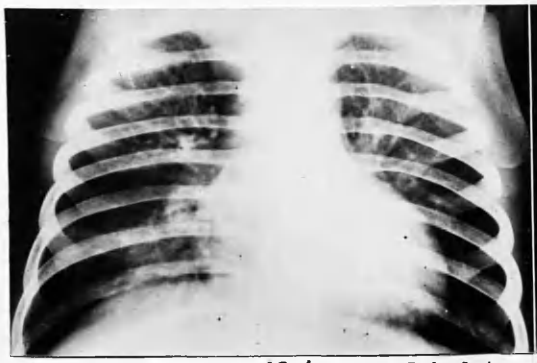
Female, aged 1½ years. Illness noted on 20/1/47, and the patient admitted to hospital with whooping cough on 23/1/47. Mantoux negative. X-ray on 6/2/47 revealed a moderate degree of atelectasis in the right middle and left lower lobes. Clinically, impairment of the P.N., and crepitations at the left base. The reproduction opposite demonstrates the right middle lobe, but not the left lower lobe collapse. The condition cleared up in 7 weeks. Mantoux negative on dismissal.

Case 20.

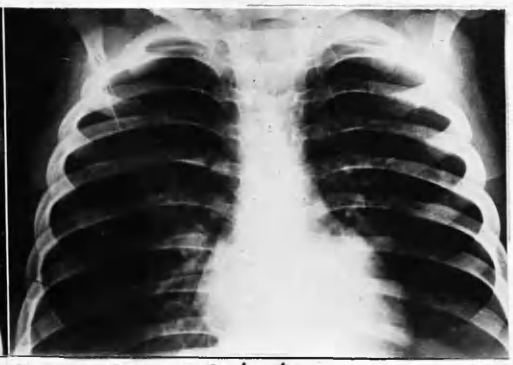
Male, aged 1 year. Illness noted on 20/2/47, and the patient admitted to hospital with whooping cough on 28/2/47. Mantoux negative. X-ray on 28/3/47 revealed a moderate degree of collapse in the right and left lower lobes. Clinically, impairment of the P.N., prolongation of expiration, and medium crepitations at both bases. The chest was clear again in 8 weeks. The reproduction opposite shows only the right lower lobe collapse. Mantoux negative on dismissal.

Case 21.

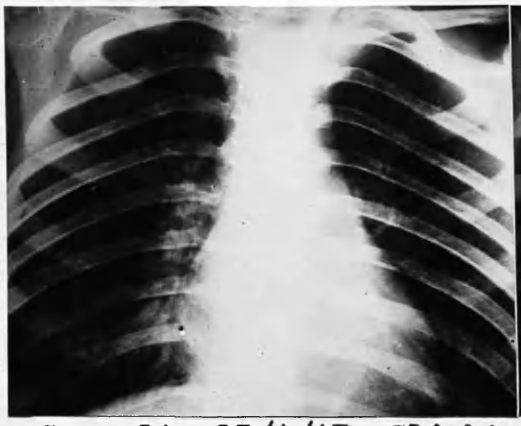
Female, aged 2 years. Illness noted on 5/3/47, and admitted to hospital on 10/3/47 with whooping cough. Mantoux negative. A radiogram on 12/4/47 demonstrated a moderate degree of collapse of the right lower lobe. Clinically, expiration was prolonged over the affected area, and medium crepitations could be heard. The atelectasis resolved in 4 weeks. Mantoux negative on dismissal. X-ray shown opposite.



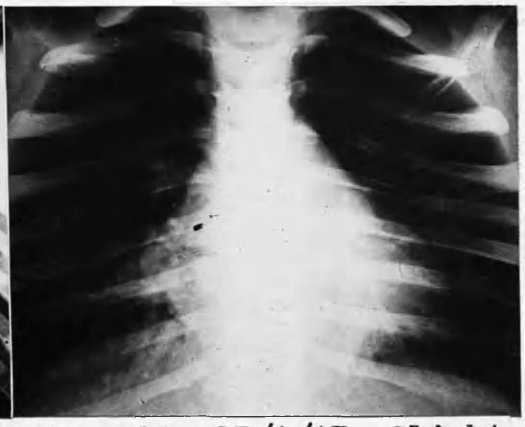
Case 22. 14/6/47. Slight degree of collapse rt. lower lobe.



Case 23. 19/4/47. Slight degree of collapse rt. lower lobe. Difficult to reproduce satisfactorily.



Case 24. 25/4/47. Slight degree of collapse rt. middle lobe. P.A. view.



Case 24. 25/4/47. Slight degree of collapse rt. middle lobe. Lordotic view.

Slight pulmonary collapse.

Case 22.

Female, aged 14 months. Illness noted on 22/5/47, and the patient admitted to hospital with whooping cough on 27/5/47. Slight atelectasis of the right lower lobe demonstrated radiologically on 14/6/47. Clinically, a few crepitations at the right base. The condition cleared up in 3 weeks. Mantoux negative on admission and dismissal. X-ray opposite.

Case 23.

Male, aged 16 months. Illness noted on 12/3/47, and the patient admitted to hospital with whooping cough on 21/3/47. Slight atelectasis of the right lower lobe radiologically demonstrated on 19/4/47. Clinically, a few crepitations at the right base. The condition cleared up in 3 weeks. Mantoux negative on admission and dismissal. X-ray opposite.

Case 24.

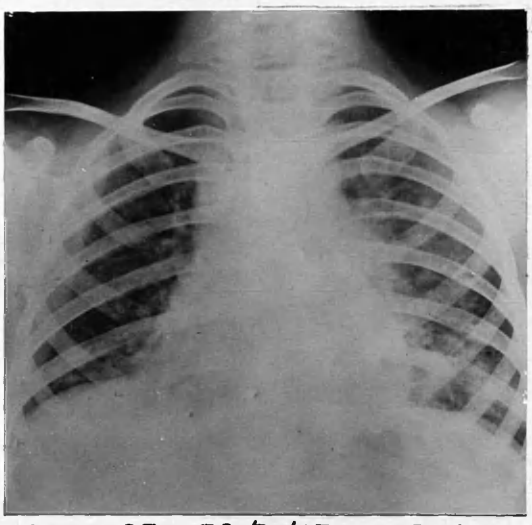
Male, aged 4 years. Illness noted 22/2/47, and the patient admitted to hospital with whooping cough on 4/3/47. A radiogram taken on 25/4/47 demonstrated slight collapse of the right middle lobe. No abnormal physical signs could be detected. The condition cleared up in 6 weeks. Mantoux negative on admission and dismissal. X-ray opposite.

Two examples of multilobular collapse were noted in the series, and thus this condition must be briefly touched upon.

The radiographic appearance is one of soft mottling, and there may or may not be mediastinal shift to the affected side, depending on the extent of the condition. It is frequently seen after haemoptysis in cases of tuberculosis.

Clinically, the signs are suggestive of broncho-pneumonia, and thus the condition is well night impossible to diagnose in whooping cough without recourse to radiology.

The condition is illustrated by the following case.



Case 25. 30/1/47. Multi-lobular collapse both lungs, more extensive in left lung. The appearance is not well brought out in the photograph. There is also some broncho-pneumonic infiltration in both lungs, and a moderate degree of collapse of the rt. lower lobe.

Case 25.

Female, aged 6 years. Illness noted on 9/1/47, and the patient admitted to hospital on 23/1/47. Mantoux positive. On clinical examination, crepitations could be heard throughout both lungs. The R.M. was harsh vesicular with a tubular element at the bases. The temperature was elevated to 102. F. on admission, but settled in a few days on the administration of sulphadiazine. A radiogram on 30/1/47 revealed broncho-pneumonic infiltration in both lungs. There was also bilateral multilobular collapse more marked on the left side, and a more extensive area of collapse at the right base.

The lungs were clinically and radiologically clear a month after admission. Radiogram shown opposite.

Technique of Bronchography.

Sicard and Forestier[9] in 1922 employed iodised oil for outlining the bronchial tree, and since that time various methods have been advocated for securing the most satisfactory results. Bronchography was carried out so frequently in the course of this study, and was of such vital importance, that it may be of interest to describe the technique adopted.

The operation may be carried out under local or general anaesthesia.

It is obvious that local anaesthesia has many advantages - there is less danger, an anaesthetist is not required, the patient is less inconvenienced, and the routine of a busy radiological department is less disturbed.

The drawback of this method is that considerable co-operation is required from the patient, and this may not be forthcoming in the very young or nervous, particularly if the technique adopted is unpleasant and alarming. Penetration of the crico-thyroid membrane, for example, is scarcely calculated to induce that state of mental

repose in the patient so helpful in securing a smooth performance of the operation. Few subjects, too would welcome the frequent repetitions of the procedure which are necessary in pursuing certain investigations. There is also an unpleasant element of danger in this method which is undesirable if bronchography is a routine. For all these reasons, this procedure was eschewed.

The technique of guiding a catheter with the assistance of fluoroscopy down the trachea and into the bronchus supplying the affected lobe has its advocates; it is claimed that in this way the oil can be directed into the area in which interest is centred. From a practical point of view, there appears to be little advantage in this, for in the vast majority of cases, it is desired not merely to outline the bronchi of the suspected lobe, but to compare their appearance with that of the other bronchi of the lung, and estimate their relationship to the bronchial tree as a whole. Moreover, it is seldom that there is no interest in the condition of the other bronchi of the same lung, or even those of the contralateral one. Above all, the procedure is time consuming, and requires a very placid type of patient.

The method adopted was the simplest of all, and the results were eminently satisfactory. It was employed in children as young as four years of age, and was ideal for out-patients referred from clinics to the hospital for investigation.

With a view to minimising the inconvenience of sickness, the procedure, if the subject were an in-patient, was carried out not sooner than three hours after a light meal. This precaution was not possible with out-patients, but vomiting was a rarity.

The patient, even if a child, was shown the materials about to be used, and the incidents and objects of the proceedings fully explained.

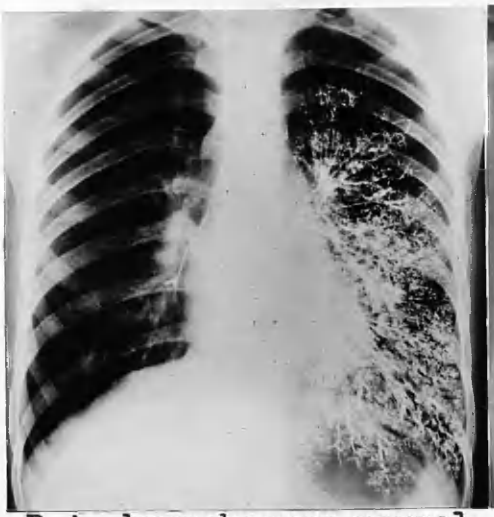
The subject then sat in a chair with the head tilted back, and supported by a nurse. The tip of the tongue, covered by a gauze swab, was gripped, and held firmly forward. A one c.c. syringe was then introduced into the mouth, and one cubic centimetre of 2% Anethaine slowly trickled over the back of the tongue. This procedure was repeated twice, so that in all 3 c.c. of the local anaesthetic were given.

A chair was now placed alongside that of the patient on the same side as the affected lung, and on it he rested his hand, so that the body was tilted to that side. The iodised oil was then trickled over the back of the tongue in exactly the same manner as the Anethaine, except that here a 20 c.c. syringe with a piece of rubber tubing attached to the nozzle was used. The tubing was employed merely because a 20 c.c. syringe has too large a diameter to enter the mouth conveniently.

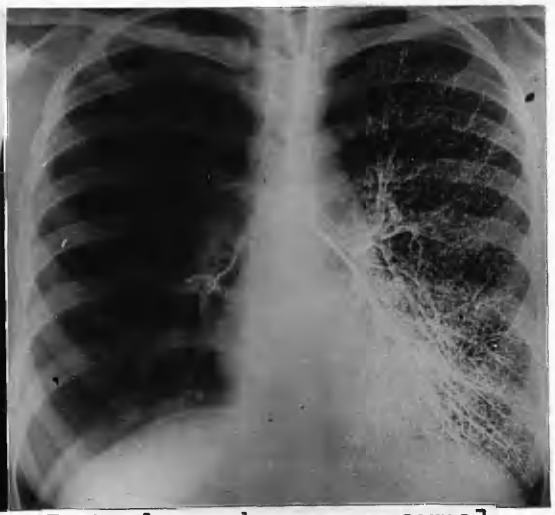
The patient, still tilted towards the affected side, was then picked up in the arms of the operator, and held so that each broncho-pulmonary segment in turn was in a dependent position and filled with oil.

This method was ideal for children, but in the case of heavy adults, some strength on the part of the operator was demanded. The adult could assist considerably, however, by helping to support himself with one hand on the floor while the operator manoeuvred him about in filling the lobes.

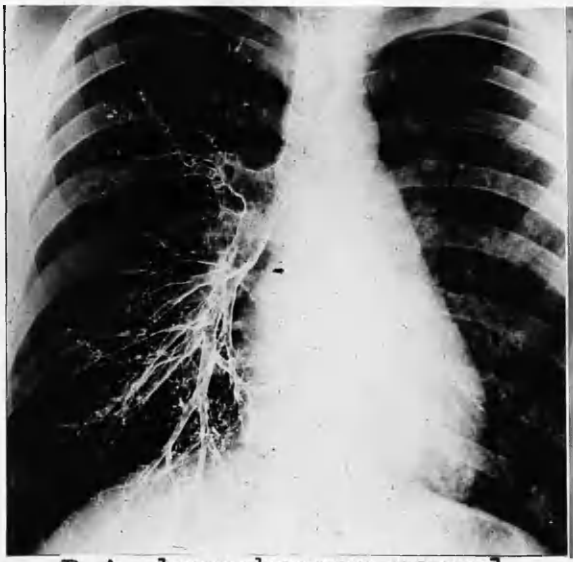
The procedure was carried out much more rapidly than is usually advised; the oil was trickled over the back of



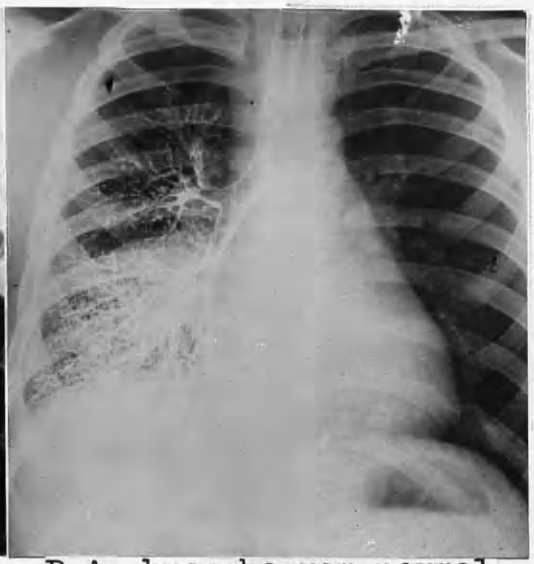
P.A. bronchogram normal left lung.



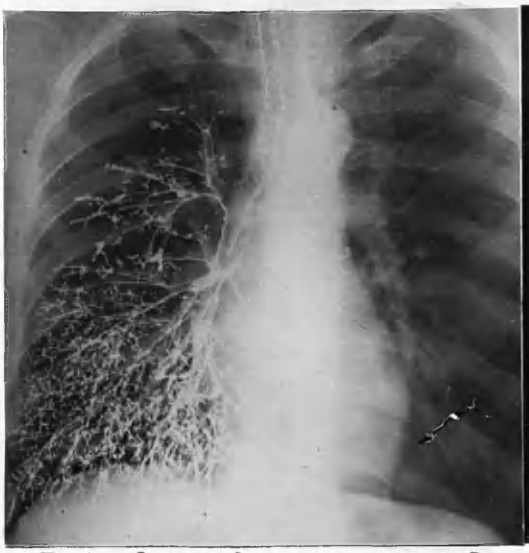
P.A. bronchogram normal left lung.



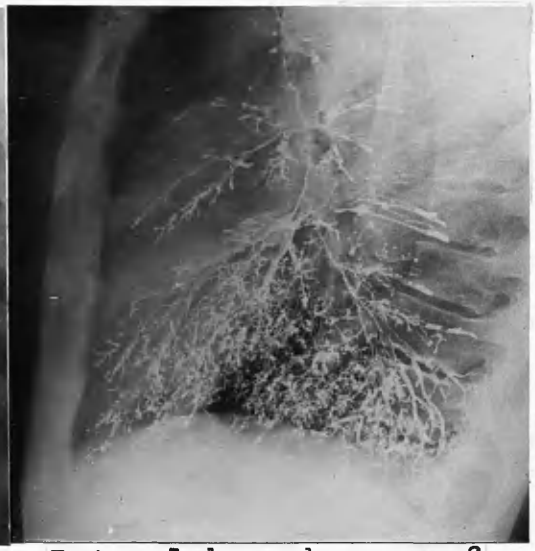
P.A. bronchogram normal right lung.



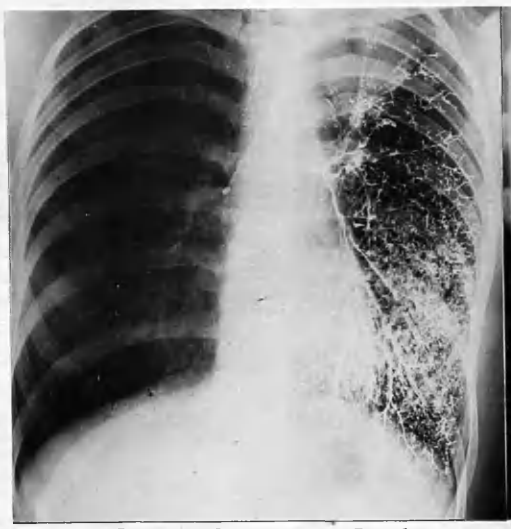
P.A. bronchogram normal right lung.



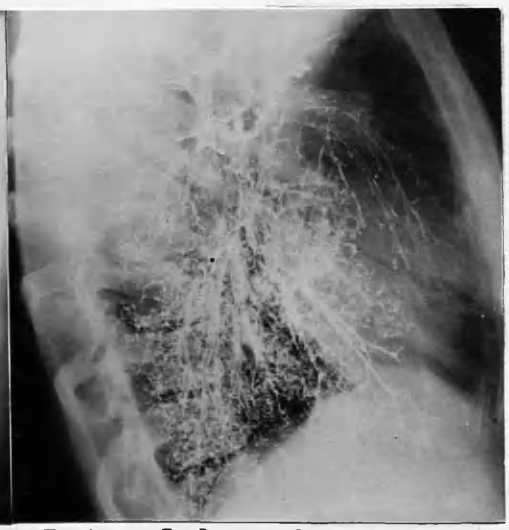
P.A. bronchogram normal right lung.



Lateral bronchogram of same case.



P.A. bronchogram left lung in which pleural thickening at apex simulated collapse. The bronchi are normal.



Lateral bronchogram of same case.

the tongue in less than two minutes.

Over one hundred bronchographies were performed by this method, and only one case of sensitivity to the anaesthetic was noted. Towards the end of the series, a boy of six years of age went into convulsions from which he recovered in thirty minutes. It was concluded that the solution used had become concentrated by evaporation, as it was supplied by a ward in which bronchography was an investigation of comparative rarity. Information that untoward reactions had been noted in two other hospitals about the same time, however, prompted a reduction in strength of the solution from 2% to 1%.

It may fairly be claimed that the method used was most satisfactory and gave excellent results. A few examples are shown opposite.

As has been mentioned, it was found possible to carry out bronchography in children as young as four years of age using local anaesthesia. For children younger than that, general anaesthesia was employed, and, of course, this was also required for older children who did not respond to the blandishments of the operator.

Chloroform was used by the anaesthetist.

The procedure followed was much the same as for bronchography under local anaesthesia.

The child was held up in a reclining position on the x-ray table, and tilted to the side of the lung about to be done. The tongue was pulled forward by sharp pointed tongue forceps, and the oil was trickled into the throat as before. The child was then picked up, and manoeuvred in the manner described.

At one time a different technique was used, in that the anaesthetist passed an intratracheal tube into which a

thin catheter leading from the syringe was then introduced. Excellent results were obtained by this method, but as the simpler technique was just as effective, the original method was retained.

The degree of filling to be aimed at apparently varies with different authors. Ogilvie[10], for example, prefers a proportion of the medium to enter the alveoli; in fact he says he is suspicious of atelectasis if this is not seen. There seems little to commend this point of view. If, in a bronchogram, there is much alveolar filling, the features are obscured, and since the lipiodol remains in the alveoli for many weeks or months, subsequent bronchograms are very difficult to interpret. Besides, some degree of alveolar filling does not exclude atelectasis, because as Ogilvie[10] himself points out in another connexion, in a collapsed lobe there are often emphysematous air bearing patches of lung interleaved with atelectatic lung tissue.

In the bronchograms carried out during the course of this work, it was endeavoured, therefore, to outline completely the bronchial tree, and yet avoid alveolar filling as far as possible. By following this course, it was found that the opaque medium generally disappeared from the lung in a few hours or days, and the definition of subsequent bronchograms was consequently unimpaired.

Where bronchiectasis is suspected, it is always desirable to perform bronchography on both lungs, and lateral views are essential for accurate diagnosis. It is best to visualise the bronchial tree of the presumed sound or least affected lung first, as iodised oil clears more quickly from normal than from ectatic bronchi; the lateral view of the contralateral bronchial tree to be subsequently visualised is then less likely to be obscured.

The quantity of iodised oil used for one lung varied from 5 c.c. for a child one year old to 20 c.c. for a very large adult.

Bronchography was employed in this experiment with great, but as the event proved, not undue freedom. Children under one year of age were not done, since it was considered that the risk of a general anaesthetic and the strain of the operation were out of proportion to any benefit the child was likely to receive, no matter how interesting the scientific aspect might be. Again, if a child were seriously ill, the investigation was not carried out.

Apart from these considerations, bronchography was employed at all stages of the disease; whooping cough per se was not regarded as any bar to the procedure, and no attempt was made to wait until the whoop had disappeared. If atelectasis was diagnosed, bronchography was carried out.

Neohydriol was the proprietory preparation used, and in all the bronchographies performed there was not an instance of any reaction to the preparation. Preliminary testing for liability to iodism was not carried out.

Having described the methods employed for diagnosing pulmonary collapse, and the technique adopted for outlining the bronchial tree, it is proposed to set forth the findings of the whooping cough investigations in the next chapter.

INCIDENCE OF ATELECTASIS IN 150 WHOOPING COUGH CASES WITH AGE DISTRIBUTION

AGE	CASES	CASES WITH ATELECTASIS	
		NUMBER	PERCENTAGE
1 ~	44	9	20
1 +	30	14	47
2 +	19	11	58
3 +	17	9	53
4 +	13	8	62
5 +	12	8	67
6 +	11	6	55
7 +	3	~	~
8 +	1	~	~
TOTAL	150	65	43

CHAPTER 2.

RESULTS OF AN INVESTIGATION

OF ONE HUNDRED AND FIFTY WHOOPING COUGH CASES.

The investigation of the incidence and effects of atelectasis in whooping cough proved to be an interesting study of one of the specific fevers, and it is therefore convenient to set forth the results in compact form. This has the further advantage that the systematic marshalling of the carefully recorded observations provides important material to which easy reference can be made when the various theories relative to the causation of bronchiectasis come to be discussed.

Incidence of Atelectasis in Whooping Cough.

The table opposite shows the incidence of atelectasis in the total number of whooping cough cases, and in the different age groups.

The numbers are, of course, relatively small, but when it is seen that out of 150 cases, atelectasis was demonstrated in 65, it can be presumed with some confidence that the condition is a common complication of whooping cough.

It would obviously be ridiculous to attach any statistical significance to the relative incidence in age groups, but the figures suggest that atelectasis is less common in whooping cough patients aged under one year, than in older children. It would be wrong to assume, however, that because collapse seems less frequent in this group it is

also less severe when it does occur. In point of fact, the most extensive collapse in the series occurred in a child of 11 months; there was atelectasis of the right lung and left upper lobe. A reproduction of the radiograph has already been shown. (Case 15, p.16).

Lobar Incidence of Atelectasis in Whooping Cough.

The incidence of atelectasis in the different lobes is interesting.

Some cases had more than one lobe involved, the lobes either showing atelectasis at the same time, or one becoming affected at a later date than the other.

Thus although atelectasis occurred in only 65 patients, more than 65 lobes were involved.

In two cases the atelectasis was multilobular in distribution, and these are excluded from the analysis. In the remaining 63 cases, 85 lobes showed collapse of greater or lesser extent.

The following table illustrates the number of times a particular lobe was affected, whether by itself,or in combination with other lobes. The lingular process of the left upper lobe is regarded as a separate lobe. Thus, unless the contrary is clearly indicated, "left upper lobe" does not include the lingular process.

LOBE.	NO. OF TIMES INVOLVED.	%.
Left Lower	43	50.5
Right Lower	31	36.5
Right Middle	8	9.4
Right Upper	2	2.4
Left Upper	1	1.2
Lingula	-	-
	85	100.0

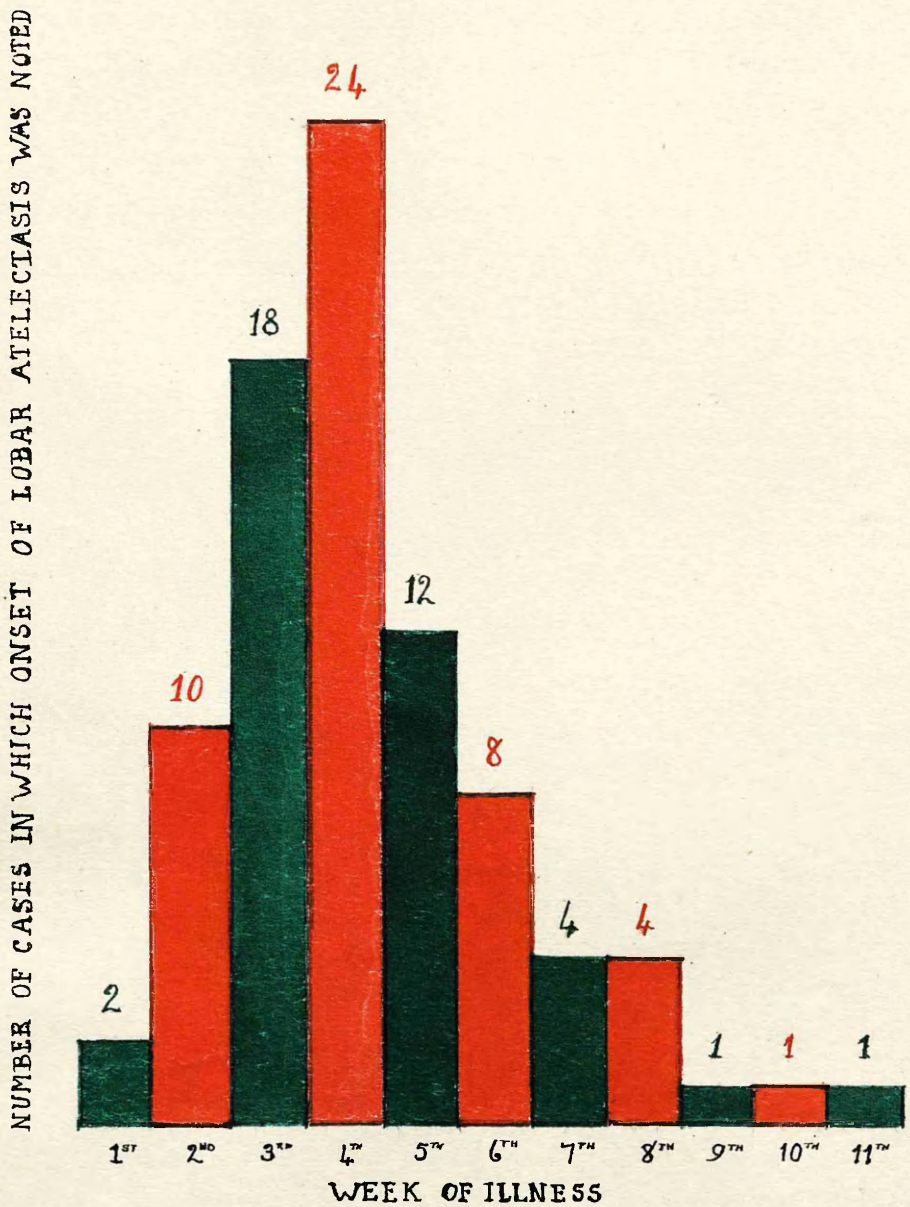
The next set of figures indicates how often the lobes were affected singly, and how often in combination with others.

LOBE OR LOBES.	NO. OF TIMES INVOLVED.
Left Lower Alone	25
Right Lower Alone	15
Left Lower and Right Lower	14
Left Lower and Right Middle	4
Right Middle Alone	2
Right Upper Alone	1
Right Middle and Right Lower	1
Right Lung and Left Upper	1
Total Cases	63

The striking feature which emerges from a consideration of the figures is the frequency with which the lower lobes were involved as compared with the others, and the predominance of the left over the right.

The significance of these findings in relation to the causation of bronchiectasis will be brought out in the next chapter.

TIME OF ONSET OF ATELECTASIS



Extent of Atelectasis in Whooping Cough.

Some indication of the extent of the atelectasis seen in the different cases is required, and the classification into three groups - slight, moderate, and marked, has been explained in the previous chapter.

Excluding the two cases of multilobular collapse, the remaining 63 cases provided 85 instances of lobar atelectasis. Of these, 60 were slight, 15 moderate, and 10 severe; in percentages. 70.6 per cent slight, 17.6 per cent moderate, and 11.8 per cent severe.

Time of Onset of Atelectasis in Whooping Cough.

Information regarding the time of onset of atelectasis in the whooping cough cases can best be given in graphic form, and this has been done on the page opposite. Sixty three cases of whooping cough provided eighty five instances of lobar atelectasis of varying extent. The graph indicates the frequency distribution in the different weeks of illness of the onset of atelectasis in these lobes.

It will be seen that atelectasis occurred with increasing frequency until the fourth week of illness, when it reached its peak, and thereafter the incidence declined.

It is worthy of note that a case occurred as late as eleven weeks after the onset of whooping cough, and there is no reason to suppose that in patients in whom respiratory catarrh persists because of supervening infection, neglect, or other cause, atelectasis may not take place at a later date still.

It may be that in some of the cases in the series, collapse occurred after the patient had been dismissed from hospital. The criteria for dismissal were six weeks

absence of atelectasis or other abnormality on radiological examination, disappearance of the whoop, and clinical cure.

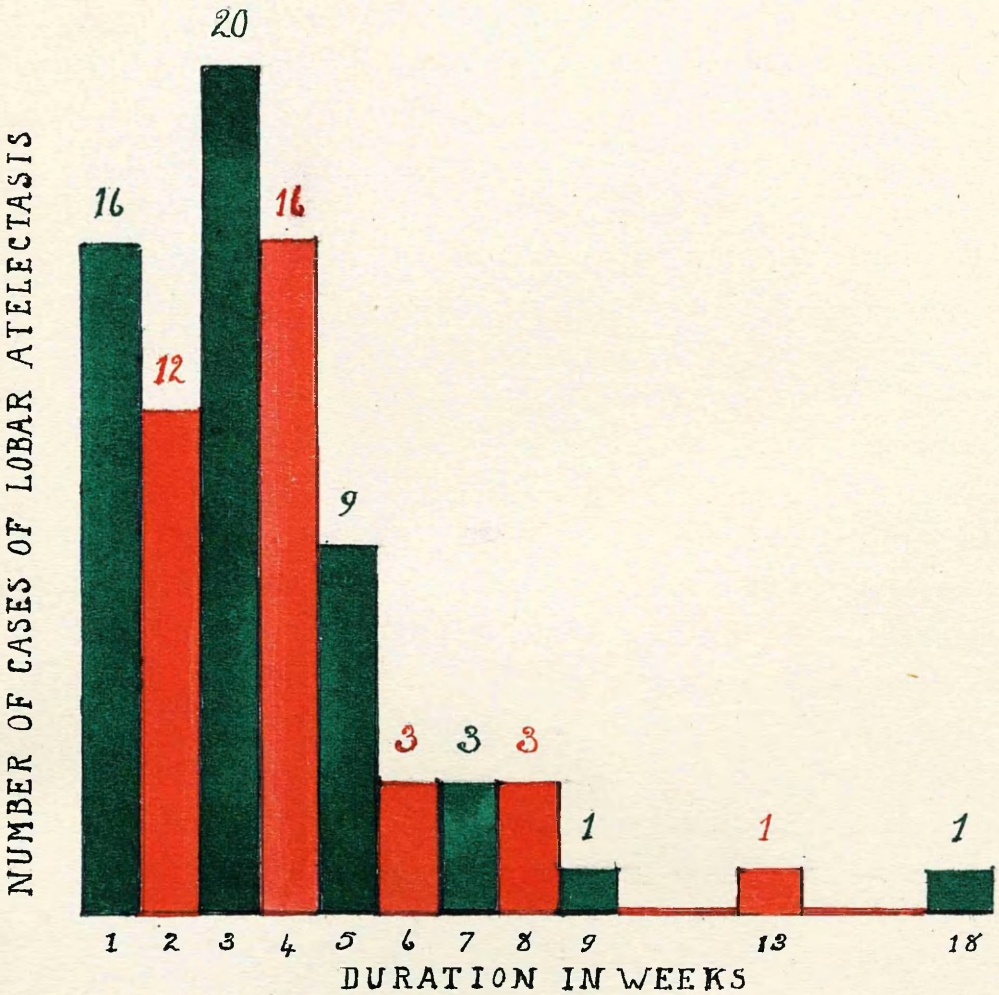
The general impression is that whooping cough leaves a "weakness in the chest", or in medical jargon, "a predisposition to respiratory disease". If this is so, a supervening infection after departure from hospital, may, in a proportion of the cases studied, have caused atelectasis indirectly attributable to whooping cough. As only some were subsequently investigated as out-patients, and no such supervision was exercised on the others, a categorical statement on this point cannot be made.

The "weakness in the chest", however, with which the laity is familiar, generally means a persistent cough with a liability to attacks of "pneumonia". This type of picture, much more than one of clubbed fingers, stinking breath, and copious sputum, is associated in the mind of the modern chest physician with bronchiectasis. The suggestion, which will be amplified later, is made here, that much of the respiratory trouble following whooping cough is due, not to a hypothetical lowered vitality of the lung paving the way for respiratory infection, but to exacerbations of a bronchiectasis arising during the course of the disease.

It is therefore considered somewhat unlikely that collapse took place after dismissal in the carefully investigated patients of the series.

This is supported by the fact that neither atelectasis nor bronchiectasis was detected in any of the numerous cases subsequently examined as out-patients for one reason or another.

DURATION OF ATELECTASIS



Duration of Atelectasis in Whooping Cough.

The duration in weeks of atelectasis in the eighty five affected lobes is illustrated by the graph opposite.

It will be seen that comparatively few collapses lasted longer than five weeks. In general, it could be said that atelectasis of slight extent cleared up more quickly than that of marked degree, but there were exceptions to the rule.

In a case previously described (Case 15,p.16) for example, where there was collapse of the right lung and extensive collapse of the left upper lobe, the whole condition cleared up in four weeks. In the other five cases showing extensive atelectasis, recovery was slower.

One of these patients (Case 14, p.16) had extensive collapse of the right upper lobe. There was considerable resolution in a fortnight, but the condition did not completely disappear until a further three months had elapsed.

Another (Case 16, p.137) had extensive collapse of the right lower lobe, and also the left lower lobe; the condition on the right side took 8 weeks to clear up, and that on the left, 9 weeks.

The remaining three cases (Case 17, p.244; Case 18, p.246; Case 13, p.249.) showed return to normal in 8, 13, and 18 weeks respectively, but as an artificial pneumothorax was induced and maintained in each of them, it cannot be asserted that this was the natural duration of the collapse. According to the individual's judgement of the effect of artificial pneumothorax, it could be considered that re-expansion was hastened, delayed, or neither one nor the other. Evidence will later be adduced, however, which tends to show that the institution of an artificial pneumothorax at least does not delay re-expansion of collapsed lung tissue.

Elevation of Temperature in Relation to the Onset of Atelectasis in Whooping Cough.

When this study was embarked upon, it was believed that a sharp elevation of temperature might prove a valuable indication that atelectasis had occurred, as this phenomenon has often been observed in cases of post-operative collapse of lung.

This expectation was not, however, realised in practice, for analysis of the cases failed to show any correlation between rise of temperature and the occurrence of atelectasis; isolated temperature elevations were noted in cases in which collapse did not take place, while in some cases of extensive atelectasis, the temperature was not elevated at all.

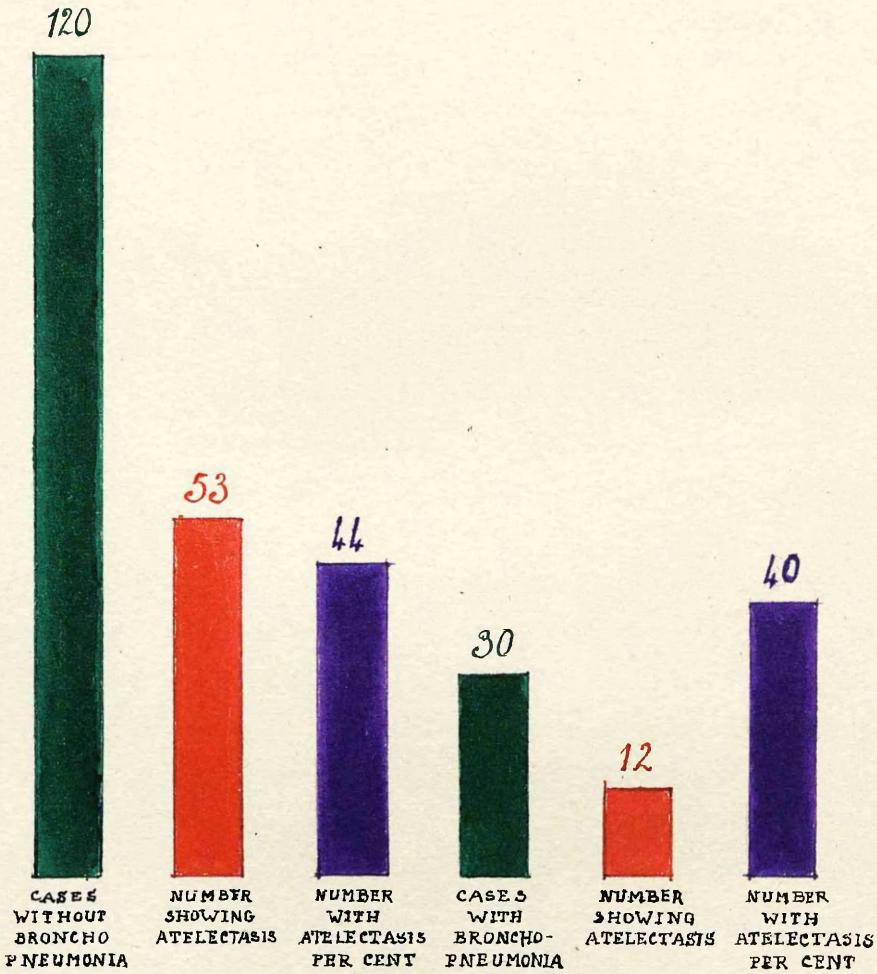
It was therefore concluded that a rise of temperature in whooping cough cases is not a satisfactory indication that atelectasis has occurred.

It is interesting to note that Gray has pointed out that while pulmonary collapse following abdominal operations is often accompanied by an elevation of temperature this is usually not the case when pulmonary collapse follows operation in the thorax.

Pain in Relation to the Onset of Atelectasis in Whooping Cough.

Sometimes the onset of atelectasis is accompanied by pain over the affected area. This was noted by a few patients encountered in chest observation ward of the Sanatorium, and in these cases a pleural rub was sometimes heard over the affected area. As has been mentioned previously, the pleuritic friction is probably purely a mechanical phenomenon. None of the children in the

INCIDENCE OF BRONCHO-PNEUMONIA IN WHOOPING COUGH AND ITS RELATION TO ATELECTASIS



whooping cough series made any complaint of pain. Many, of course, were in any case too young to do so. Pleural friction was not noted.

Incidence of Broncho-pneumonia
and its Relation to Atelectasis.

Broncho-pneumonia, as is well known, is the most serious complication of whooping cough, as it is the one responsible for most of the deaths which occur in the disease. It is well to remember, however, that bronchiectasis is undoubtedly responsible for a much higher mortality rate than is generally supposed, and it is probably safe to say that no one as yet can even hazard a guess at the approximate figures. No radiological study of whooping cough with a follow up of those cases which have developed bronchiectasis, has apparently appeared in the literature. Even when such a study has been carried out, the present mortality will never be known, as no observer, having noted bronchiectasis in its earlier stages, would be so foolish as to allow it to progress beyond a stage suitable for surgical intervention.

In this study the main concern with broncho-pneumonia was its influence on the incidence of atelectasis, as it was believed atelectasis might give rise to bronchiectasis, and special care was taken to find out if broncho-pneumonia produced bronchiectasis in cases of whooping cough by predisposing to atelectasis or in any other way.

The diagnostic criterion for broncho-pneumonia was radiological evidence, supported, of course, by clinical findings.

Diagnosis on clinical grounds alone was considered unsatisfactory. An elevated temperature, crepitations in

the chest, patchy dullness, and areas of tubular breathing, could indicate little else than broncho-pneumonia, but where there is only an elevated temperature and numerous crepitations, it is difficult in an infant to say whether the condition is broncho-pneumonia or bronchitis; and a combination of atelectasis and broncho-pneumonia is almost impossible to diagnose without the assistance of radiology.

In the series under investigation, broncho-pneumonia was detected in 30 cases out of 150 i.e. 30 of the whooping cough patients had broncho-pneumonia as a complication. Seven of the 30 cases developed the condition after admission to hospital.

Atelectasis occurred in 12 of the 30 cases with broncho-pneumonia, a 40 per cent incidence.

One hundred and twenty cases did not show evidence of broncho-pneumonia, and of these 53 developed atelectasis, that is to say a 44 per cent incidence.

It appears, therefore, that broncho-pneumonia occurring in whooping cough, has little influence on the frequency of atelectasis. If it is believed that atelectasis is due mainly to the aspiration of sputum, this finding is readily credible. The abundant, tenacious sputum of whooping cough seems eminently suitable for producing collapse, and any additional sputum there may be from an associated broncho-pneumonia does not appear likely to add greatly to the risk.

Broncho-pneumonia, of course, can per se give rise to atelectasis; how often it does so is problematical, as there does not appear to be a radiological investigation on the subject in the literature. Some cases are shown in the next chapter. Bronchial dilatation was never demonstrated in whooping cough cases complicated by broncho-pneumonia except in atelectatic areas.

Incidence of Hilar Adenitis
and its Relation to Atelectasis.

Enlargement of hilar glands was demonstrated radiologically in 28 of the 150 whooping cough cases, that is to say, in 18.7%. This was a finding of some importance, for it was obvious that atelectasis might result from glandular pressure on a bronchus, and great care was therefore taken throughout the investigations to distinguish atelectasis due to glandular pressure on a main bronchus from atelectasis due to occlusion of bronchioles or finer bronchi. The diagnostic criteria relied upon were those described in the previous chapter - that is, in the case of obstruction of a main bronchus by pressure from enlarged glands, absent breath sounds over the collapsed area and failure or partial failure of iodised oil to pass the obstruction, and in the case of atelectasis due to occlusion of bronchioles or finer bronchi, weak tubular breathing over the collapsed portion of lung and free entry of iodised oil into the larger bronchi lying in the atelectatic area.

The appearances on the straight x-ray were, of course, often sufficient by themselves to eliminate pressure of hilar glands on a main bronchus as the cause of the collapse. The mere absence of demonstrable glandular enlargement was naturally not sufficient to do so, but since the only bronchi likely to be compressed by this agency supply a large portion of lung, the resultant collapse would of necessity be correspondingly considerable; enlarged hilar glands could scarcely be held responsible for small, peripheral areas of atelectasis. It was therefore only when collapse was fairly extensive that it was necessary to invoke the aid of bronchography.

It may be mentioned that in two instances of extensive collapse occurring in children under one year of age, the elimination of hilar glands as a causative factor had to be made from the clinical findings alone, as it was considered that the patients were too young to warrant the employment of bronchography.

In the few cases in which even the bronchographic appearances leave doubt as to whether atelectasis is due to compression of the main bronchus supplying the collapsed area, bronchoscopy clinches the diagnosis, but its assistance was not required in the whooping cough series.

In the whooping cough cases investigated, although atelectasis was so often demonstrated, no instance was discovered in which the cause was pressure of enlarged hilar glands on a bronchus. The right middle lobe was regarded as being particularly vulnerable in this respect, because of the close relation of the right inferior tracheo-bronchial lymph glands to its bronchus, and although it escaped in the cases of the series, no doubt right middle lobe collapse does occasionally occur from this cause.

Bronchial Dilatation in Atelectatic Portions of Lung.

In view of the fact that the association between atelectasis and bronchiectasis has aroused increasing interest in recent years, it was decided to carry out bronchographic investigation of whooping cough cases which showed evidence of atelectasis of marked or moderate degree, and also of cases in which atelectasis was slight in extent, provided the condition persisted for three weeks or longer . Bronchography was not performed in children under one year of age, however, as it was considered that the risk was

here too great to warrant the procedure.

Six patients in whom pulmonary collapse of marked extent occurred were encountered in the series. Two of them (Cases 14 and 15, p. 16) were under one year of age, and consequently bronchography was not performed. The procedure was carried out in the four other cases, and in every instance well marked dilatation of the bronchi in the collapsed portion of lung was demonstrated. In one of these four cases there was extensive collapse of both lower lobes with dilatation of the contained bronchi. Re-expansion of the right lower lobe took place after eight weeks, and bronchography subsequently showed that the associated bronchial dilatation had disappeared. After nine weeks the left lower lobe re-expanded, and it was again demonstrated that the contained bronchi had resumed their normal calibre. This case is fully discussed and illustrated in the next chapter (Case 16, p. 137.).

In the remaining three of the four cases in which bronchial dilatation was demonstrated, the lung affected by the pulmonary collapse was relaxed by means of an artificial pneumothorax for reasons which will appear later. In one of these three cases, the collapse involved the right middle lobe and an adjacent portion of the right lower lobe; in the remaining two cases the left lower lobe was affected. Re-expansion of the collapsed lung tissue took place in eight weeks in the first of these cases, in thirteen weeks in the second, and in eighteen weeks in the third. The bronchial dilatation disappeared in every instance with re-expansion of the collapsed lung tissue. These cases are fully discussed and illustrated in the fourth chapter of this work. (Case 17, p. 244; Case 18, p. 246; Case 13, p. 249.).

Fifteen of the cases in the whooping cough series showed atelectasis of moderate extent, but the bronchi in the collapsed portions of lung were only visualised in ten of them. In the five cases in which bronchography was not carried out, either the atelectasis had disappeared before the examination could be performed, or the patient was too young, or permission was refused.

Of the ten cases bronchographically investigated, bronchial dilatation was detected in four. It was, however, very slight in extent. Re-expansion of the collapsed lung tissue in these four cases occurred in approximately one month, and subsequent bronchography revealed that in each of them the bronchial dilatation had disappeared.

Bronchography was also carried out in ten cases in which there was atelectasis of slight extent, but bronchial dilatation was not seen.

For various reasons bronchographic examinations were performed on twenty five of the whooping cough patients in which pulmonary collapse was not present. In some of these atelectasis had previously been detected and had cleared up, while others had suffered from the complication of broncho-pneumonia. Bronchial dilatation was never demonstrated.

In the whooping cough series, therefore, marked bronchial dilatation was demonstrated in four cases of marked pulmonary collapse, and slight bronchial dilatation in four cases in which pulmonary collapse was moderate in extent; it was only present in the atelectatic area, and disappeared when the collapsed portion of lung re-expanded. Bronchial dilatation unassociated with pulmonary collapse was never observed.

Comment.

Regarded as a study in whooping cough, the investigations brought to light matter of great interest. The high incidence of pulmonary collapse observed appears at first sight to be somewhat surprising in view of the fact that its presumptive frequent occurrence in whooping cough has apparently received little recognition in the literature. Harries and Mitman,[2] for example, whose excellent text book on the specific fevers is a standard work, do not even mention pulmonary collapse as a complication of the disease. Yet although no hard and fast conclusions on the incidence of atelectasis in whooping cough can be drawn from a study of a mere one hundred and fifty cases, it can scarcely be doubted that it occurs with considerable frequency. It is suggested that this fact has been generally overlooked because the only method of establishing it, that of repeated radiological examination, has been seldom employed. A survey of the literature failed to reveal a radiological investigation of whooping cough on the lines of the one carried out in this work. There is therefore no reason to suppose that the incidence of pulmonary collapse recorded in the cases examined is not fairly representative of the frequency with which the condition usually occurs in whooping cough. It is worth noting that in the majority of instances in this study, the pulmonary collapse observed was small in extent and comparatively ephemeral in duration, and could not have been diagnosed from the physical signs alone. Even in the cases in which the atelectasis was more extensive, enough has been said in the previous chapter to indicate that in the absence of radiological evidence an observer not specifically

searching for the condition might well have confused it with pneumonic consolidation or overlooked it altogether.

The demonstration of what was apparently marked bronchial dilatation in areas of extensive pulmonary collapse was of particular interest. Authorities on bronchiectasis are unanimous on the point that the condition may have its origin in an attack of whooping cough, but so far as can be ascertained from a review of the literature, this opinion is based on the fact that patients who had no respiratory trouble before suffering from whooping cough had a persistent productive cough thereafter, which bronchography eventually revealed to be a symptom of bronchiectasis; no instance was encountered of the bronchial dilatation actually having been shown to arise during the course of the whooping cough.

The demonstration in this study of bronchial dilatation in collapsed portions of lung obviously suggested a mechanism by which bronchiectasis could be produced in whooping cough, and seemed to give great significance to the finding that atelectasis in whooping cough is of frequent occurrence. Yet there is evidence which prevents a too ready acceptance of these inferences. Cases have been recorded in which bronchography indicated bronchial dilatation in areas of pulmonary collapse, and in which re-expansion of the collapsed lung tissue subsequently took place with disappearance of the bronchial dilatation. These are examples of so called "reversible bronchiectasis." It has already been explained that interpretations of these radiological phenomena differ widely. Some writers believe that the radiological appearances of bronchial dilatation are illusory, and others maintain that while bronchial dilatation exists,

it is not "true" bronchiectasis. On the other hand it has been claimed that potentially "reversible" bronchiectasis represents an early stage of "true" or permanent bronchiectasis. This latter view was the one adopted by the author.

When bronchial dilatation was noted in collapsed areas of lung the problem arose how best to use this rather precious material. It was expected that in all probability some cases of bronchial dilatation would clear up, while others would become indubitable examples of permanent, "true" bronchiectasis, though no means would be available of predicting, when the dilatation was first diagnosed, what the event would be. It is significant that in such circumstances none of the authorities who claim that "reversible" bronchiectasis is not "true" bronchiectasis, suggest any method of distinguishing the conditions; they can only advise with Mr. Asquith - "Wait and see."

It was tempting to act on that maxim for two reasons: firstly, because if dilated bronchi in some instances reverted to their normal calibre in the exact circumstances in which others did not it would be irrefutable proof that bronchiectasis is, for a time at least, a "reversible" condition; secondly, an estimate could be made of the frequency of permanent bronchiectasis in whooping cough, and hence completeness would be given to the study of this disease.

On the other hand, there were grounds for believing that the induction and maintenance of an artificial pneumothorax in cases of bronchial dilatation seen in a recent atelectasis would be likely to prevent the ectasia from becoming permanent, and it is not every day that

atelectasis is encountered the onset of which is known for certain.

Apart from the possible benefit to the patient, it was decided that on the whole it would be more valuable to study the effects of artificial pneumothorax because not only would some idea of the benefits, if any, of its employment in early atelectatic bronchiectasis be gained, (assuming for the moment that the condition is bronchiectasis), but points of great theoretical interest with regard to the relation between atelectasis and bronchiectasis would be clarified. Artificial pneumothorax was therefore instituted in three cases of extensive pulmonary collapse apparently associated with marked bronchial dilatation, and it has already been noted that in all these cases the bronchi reverted to their normal calibre when the atelectatic portion of lung re-expanded. Whether this satisfactory conclusion would have resulted in each of these instances had this measure not been adopted is a question which will later be considered. It is sufficient here to add that as the fourth case of extensive pulmonary collapse in which bronchography apparently demonstrated marked bronchial dilatation eventually completely recovered without interference in the way of induction of an artificial pneumothorax, and a similar event was noted in the remaining four much less striking instances of apparent bronchial dilatation which were brought to light, no example of permanent bronchiectasis resulting from the effects of pulmonary collapse was noted in the series.

It was therefore obvious that the importance of the whooping cough study depended largely on the nature of "reversible bronchiectasis." If it were an early stage of "true" , permanent bronchiectasis then the high

incidence of pulmonary collapse demonstrated in whooping cough would satisfactorily explain the association of bronchiectasis with the latter disease. If, on the other hand, "reversible bronchiectasis" was an entity unrelated to permanent bronchiectasis, then the significance of the frequent occurrence of pulmonary collapse in whooping cough would be somewhat more obscure.

In order to clarify this point, the causation of bronchiectasis was studied, effort being especially directed towards discovering the role, if any, played by atelectasis. Observations recorded in the whooping cough investigations, taken in conjunction with evidence derived from other sources, appeared to throw light on this interesting subject which will be discussed in the following chapter.

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CONTENTS.

VOLUME 2.

CONTENTS (contd.).

CONTENTS (contd.).

CHAPTER 3.

BRONCHIECTASIS.

In the previous chapter, it was seen in the series of whooping cough cases studied, that atelectasis occurred in a considerable number. This was a finding of great interest, and led inevitably to an extremely difficult problem - an assessment of its significance. The association of atelectasis with bronchiectasis has long been known, but a review of the literature on the subject made it plain that the precise relationship of the conditions was still much in dispute. The issue raised by the findings of the whooping cough experiment was whether atelectasis can cause bronchiectasis, but it was plain that no attempt could be made to answer this question without considering the other factors which have been implicated.

In this chapter a survey of the literature on bronchiectasis is attempted. At first sight, in view of the numerous productions which accumulated down the years, this may seem to demand a monumental work, but closer examination reveals that considerable compression is not only possible, but advantageous. Much that has been written on bronchiectasis, while interesting, is essentially repetitive, adding nothing to fundamental conceptions of the disease, and much is of historical rather than of practical importance. To adapt Voltaire's aphorism on Shakespeare, it may be said of the literature on bronchiectasis, that "there are some pearls on the mountainous dunghill."

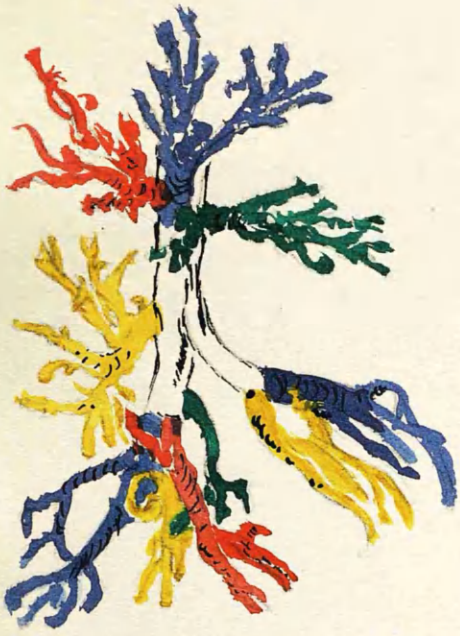
Some theories advanced on the causation of the disease are therefore dealt with briefly; the roles of

infection and atelectasis receive detailed treatment, and the discussion is illustrated by cases encountered in the chest unit, as well as in the whooping cough and other fever wards.

As bronchiectasis is a subject with many aspects, it is proposed, for convenience, to divide the chapter into several sections. In the first section relevant features of the anatomy and physiology of the bronchial tree are outlined, in the second section the pathology and bacteriology of bronchiectasis are considered, in the third section generally accepted classifications of bronchiectasis are dealt with, and in the fourth section the causation of bronchiectasis is discussed in detail.

LAT VIEW.

MEDIAL VIEW.



LAT VIEW.

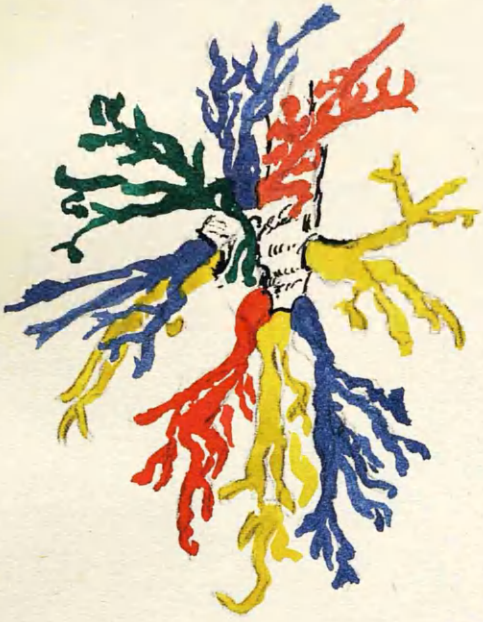
P.A. VIEW.

BRONCHO-PULMONARY SEGMENTS AND BRONCHIAL TREE. RIGHT LUNG. [AFTER BROCK].

UPPER LOBE -	APICAL SEGMENT AND BRONCHI	- BLUE
	SUBAPICAL SEGMENT AND BRONCHI	- RED
	PECTORAL SEGMENT AND BRONCHI	- GREEN
MIDDLE LOBE -	MEDIAL SEGMENT AND BRONCHI	- BLUE
	LATERAL SEGMENT AND BRONCHI	- YELLOW
LOWER LOBE -	APICAL SEGMENT AND BRONCHI	- YELLOW
	ANT. BASAL SEGMENT AND BRONCHI	- RED
	MIDDLE BASAL SEGMENT AND BRONCHI	- YELLOW
	POST. BASAL SEGMENT AND BRONCHI	- BLUE
	CARDIAC SEGMENT AND BRONCHI	- GREEN

LAT VIEW.

MEDIAL VIEW.



LAT VIEW.

P.A. VIEW.

BRONCHO-PULMONARY SEGMENTS AND BRONCHIAL TREE. LEFT LUNG. [AFTER BROCK].

UPPER LOBE –	APICAL SEGMENT AND BRONCHI	– BLUE
	SUBAPICAL SEGMENT AND BRONCHI	– RED
	PECTORAL SEGMENT AND BRONCHI	– GREEN
LINGULA –	UPPER SEGMENT AND BRONCHI	– BLUE
	LOWER SEGMENT AND BRONCHI	– YELLOW
LOWER LOBE –	APICAL SEGMENT AND BRONCHI	– YELLOW
	ANT. BASAL SEGMENT AND BRONCHI	– RED
	MIDDLE BASAL SEGMENT AND BRONCHI	– YELLOW
	POST. BASAL SEGMENT AND BRONCHI	– BLUE

SECTION 1.

ANATOMY AND PHYSIOLOGY.

Anatomy of the Bronchial Tree.

It is unnecessary in an essay dealing primarily with the causation of bronchiectasis to discuss in detail the gross anatomy of the bronchial tree, but it may be mentioned in passing that studies on broncho-pulmonary segments by observers such as Nelson[1,2], Foster-Carter[3], Appleton[4], Lodge[5] and especially Brock[6,7] are extremely important contributions to the literature on this subject, and have made possible recent advances in the surgical treatment of the disease. In the light of their work the segment of lung and not the lobe may now be used in describing the distribution of bronchiectasis, and, indeed, must be used, if surgery is contemplated. In the pages which follow, however, unless where otherwise essential, the lobe has been taken as the unit, as not only does this facilitate comparison with the findings of less recent writers, but, in any case, the nomenclature of the broncho-pulmonary segments is not yet standardised, and is apt to be confusing. Nevertheless, as it will be occasionally convenient in the course of the work to refer to broncho-pulmonary segments, their distribution is indicated in the diagrams shown opposite. The nomenclature adopted is that suggested by Brock[7].

Some features of the structure of the bronchi require to be noted. According to Macklin[8], the bronchi are to be regarded as tubes of muscular and elastic tissue ("myo-elastic tissue"), which are enclosed in a sheath of collagen and elastica, stiffened by cartilaginous plates,

and lined by a continuous sheet of epithelium. He regards the arrangement of these elements as being of great importance. Internally, beneath the mucosa, is a layer of branched elastic fibres, which is succeeded by flat branching muscular bundles. The elastic fibres run parallel to the muscle strands. Macklin[8] describes the arrangement as "a branched, tubular net." This association of muscle and elastic tissue becomes even more intimate as the finer bronchial tubes are reached, and the tunica propria disappears as a separate layer. The muscle of the bronchi, although the direction of the fibres varies from point to point, is neither circular nor longitudinal, but rather oblique.

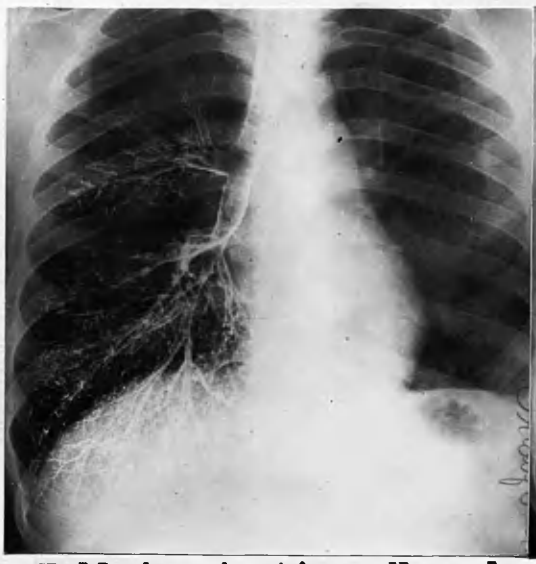
Miller[9] describes the structure as a "geodesic network", and his observations on the bronchial blood supply show clearly how liable is the internal capillary system to haemorrhage in destructive lesions of the tubes.

Bronchial Movements During Respiration.

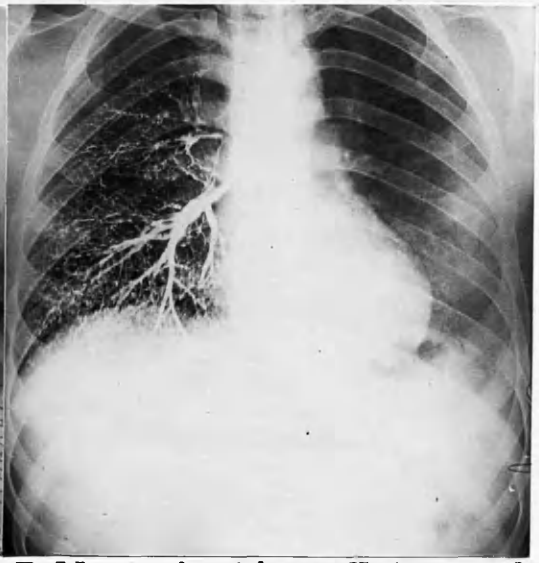
The introduction of bronchography and bronchoscopy has made it possible to study movements of the bronchi during respiration.

By means of bronchoscopy, Jackson,[10] in 1917, observed in a child a difference in length of 2 cms. in the right main bronchus between maximum inspiration and maximum expiration, and in 1920, Bullowa and Gottlieb[11] demonstrated calibre changes by the use of radio-opaque oil.

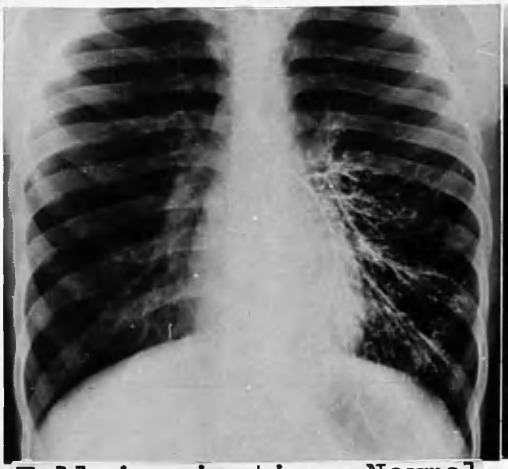
The findings of different workers varied, however, and in 1929 Macklin[8] reviewed the literature on the subject. He found that the balance of experimental evidence favoured the view that the bronchi become longer and wider on inspiration, and shorter and narrower on expiration.



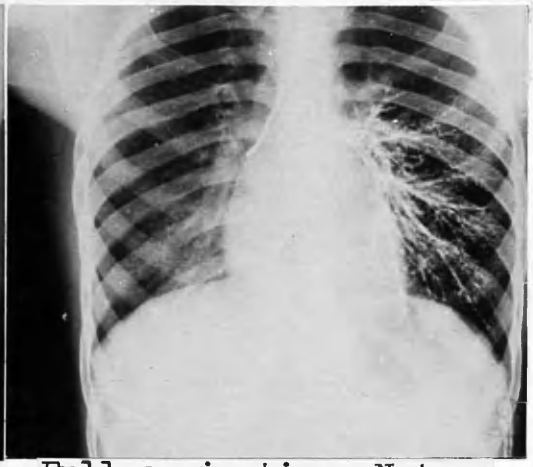
Full inspiration. Normal right bronchial tree visualised.



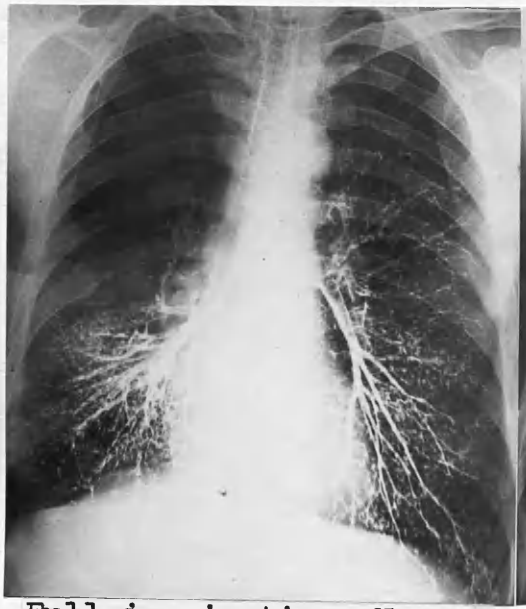
Full expiration. Note good respiratory excursion. Even in the photograph slight diminution in bronchial calibre can be observed.



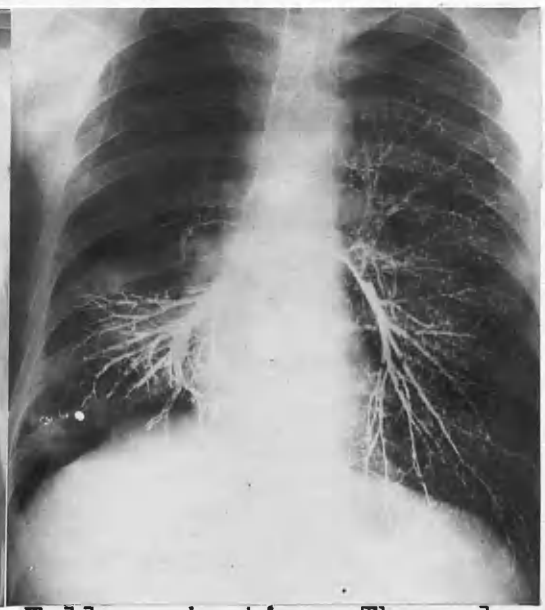
Full inspiration. Normal left bronchial tree visualised.



Full expiration. Note good respiratory excursion evidenced by elevation of diaphragm and increased markings in rt. lung.



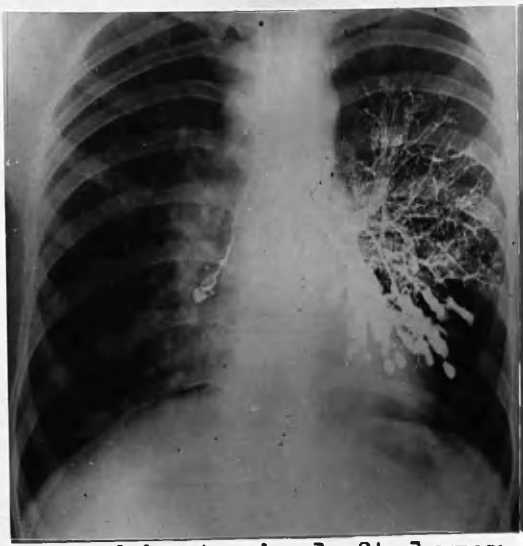
Full inspiration. Normal left bronchial tree visualised, and some radio-opaque medium also present in normal right bronchial tree.



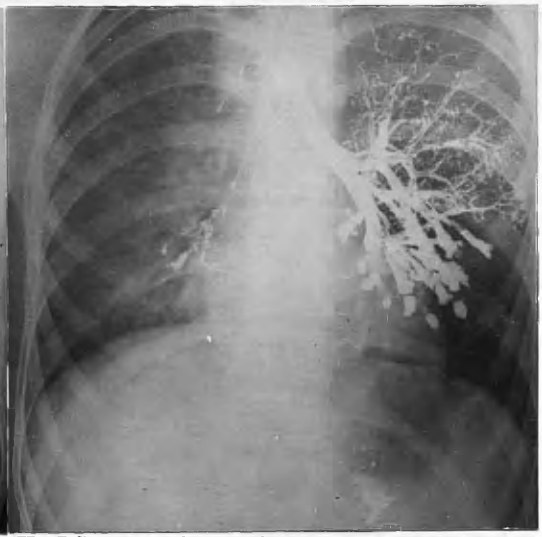
Full expiration. The calibre of the bronchi visualised in the rt. lung seems actually to have increased, but note that only the proximal parts are now visualised.

During the course of the present work, many patients were referred from clinics to the chest unit for bronchography. For a time, bronchograms were taken during full inspiration and full expiration, until a series of ten patients whose lungs proved to be normal had been investigated in this manner. In seven of these cases, in which the respiratory excursion had been marked, slight but definite bronchial dilatation was observed on full inspiration. The inspiratory increase in calibre was not so great in the vertically disposed bronchi of the lower lobe as in other parts of the bronchial tree, and in two cases no difference in calibre could be detected. It may be noted that Heinbecker[12], in 1927, reported that the vertically disposed bronchi of the lower lobes become narrower in some phases of inspiration. Inspiratory increase in bronchial calibre was most clearly apparent in the upper lobes. In the remaining three cases investigated, the respiratory excursion judged by the position of the diaphragm, was not of great extent, and no change in the bronchial calibre could be detected. It was concluded as a result of these experiments that changes in bronchial calibre during normal respiration must be very slight, and probably vary in different individuals. Three cases, illustrating the difference in bronchial calibre at full inspiration, and at full expiration, are shown opposite. It is unfortunate, but inevitable, that the change in calibre is almost imperceptible in the reproductions, but this emphasises the fact that it is slight even in the original plates.

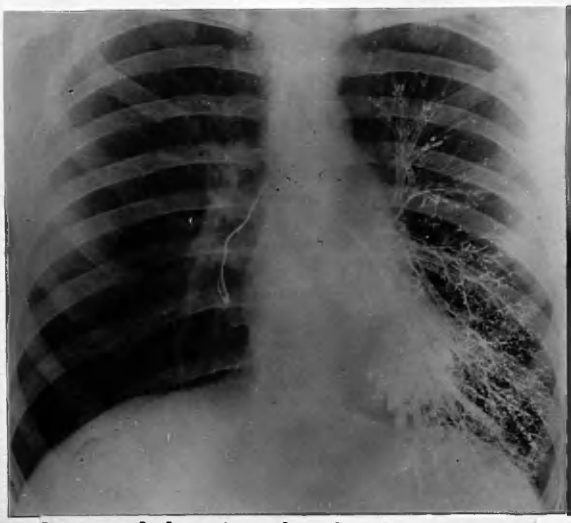
It is convenient to mention here that in cases of long standing bronchiectasis, Lander and Davidson[13] demonstrated widening of the dilated bronchi on inspiration. Their findings were challenged by Greenfield[14], but Lander[15]



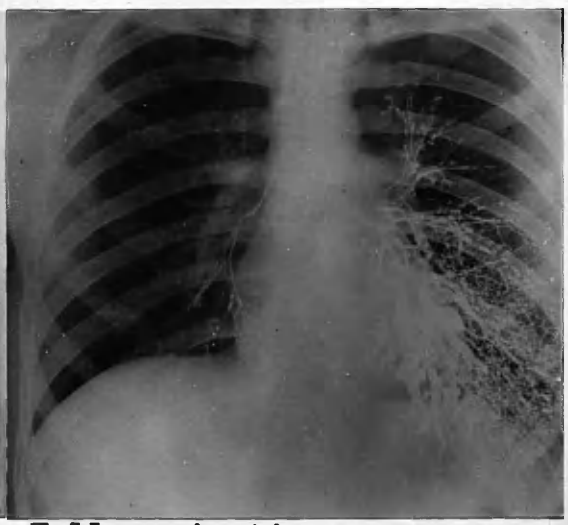
Bronchiectasis left lower lobes and lingula. Full inspiration.



Full expiration. Note good respiratory excursion. No diminution in calibre of the dilated bronchi.



Bronchiectasis left lower lobe. Full inspiration.



Full expiration. Note good respiratory excursion. No diminution in calibre of the dilated bronchi.

claimed that in Greenfield's bronchograms of the cases in which the latter author stated there were no bronchial calibre changes, the position of the diaphragm had little altered on inspiration indicating that the respiratory excursion had been small, and consequently little change in bronchial calibre could be expected.

Lander and Davidson, as a result of previous clinical, experimental, and pathological studies[16,17] were convinced at the time that bronchiectasis was almost entirely due to mechanical factors and not to infection, and they were attempting to prove that even in long established cases the elements of the bronchial wall remained intact and preserved their function. The matter will be fully considered later; it is enough to say here that examination of lobectomy specimens by Ogilvie[18] and others has shown beyond all doubt that in some cases the bronchial walls have been severely damaged by infective processes, and that extensive fibrous replacement of the destroyed tissue has taken place. In such instances it could scarcely be expected that any calibre changes would take place during respiration. None were seen in six cases of old standing bronchiectasis investigated by means of bronchography in the course of the present study, no doubt for this reason. Bronchograms illustrating two of these cases are shown opposite, and the difference in position of the diaphragm on inspiration and expiration demonstrates convincingly that the respiratory excursion in both instances was marked. Sometimes[17,18], however, pathological examinations of lobectomy material have revealed that the walls of the ectatic bronchi have suffered remarkably little damage even in cases of long duration, and it appears that only by postulating that the examples investigated by

Lander and Davidson[13] were of this nature can their findings be satisfactorily explained.

It must be admitted that bronchography has its limitations as a method of exploring bronchial movements during respiration. Ellis[19] pointed out that bronchoscopy is more satisfactory, as by this means it is possible to record volume changes.

The exact mechanism producing respiratory changes in bronchial calibre has not yet been fully established, but it is generally agreed that muscle and elastica act evenly and harmoniously together (Wilson[20] and Macklin[8]). Ellis[19] and Heinbecker[12] believe that calibre changes are the result of movements of the chest wall, and dilatation is a purely passive phenomenon. In inspiration, the lung increases in size in all diameters, and similar expansive movements must occur throughout the respiratory passages. Ellis[19] is of the opinion that the bronchial muscle is concerned entirely with maintenance of a moderate tone which prevents expiratory movements from occluding the lumen.

Bronchial Peristalsis.

Horvath[21] and Von Schrotter[22] described peristaltic movements of the bronchi. Bullowa and Gottlieb[23] further studied this problem, and observed waves of peristalsis 10 cms. in extent when radio-opaque material was introduced into the bronchi. Reinberg[24] confirmed these findings, and described the phenomenon as "tracheal vomiting." These movements could be differentiated from transmitted impulses and were independent of any other bronchial movement.

Jackson[25] was unable to confirm these observations with the bronchoscope, but this is not the best means for the exploration of this problem.

Macklin[8] summed up the evidence by saying that peristalsis is probably slight under normal conditions, but plays a major part in the expulsion of secretions and foreign bodies. The presence of abundant sensory nerve endings in the bronchial wall, and the existence of Remak's ganglion, show that an apparatus for peristaltic action exists.

Ogilvie[18] considers that in the expulsion of secretions and foreign substances from the lung, the ciliary action and bronchial peristalsis are together responsible for shepherding these substances to a point whence they may be expelled by coughing.

SECTION 2.

THE PATHOLOGY AND BACTERIOLOGY OF BRONCHIECTASIS.

PATHOLOGY.

In considering the pathology of bronchiectasis, it is well to remember that we are dealing with a disease which is exceedingly difficult to define. Until comparatively recent times, there was little doubt as to what the term "bronchiectasis" implied. It meant a disease characterised by permanent dilatation of the bronchi, the production of copious foul smelling sputum, and a more or less steady progression to a fatal termination. Fleischner[26] and others, however, have been able to demonstrate cases of so called "reversible bronchiectasis;" that is to say, a condition in which the bronchi are pathologically dilated, but eventually revert to their normal calibre. Several workers, including Lander and Davidson,[17] claim that all cases of bronchiectasis are at first "reversible", and maintain that the pathologist has been dealing only with the advanced stages of the disease. If this be true, it is plain that although much useful information may be gleaned from a pathological approach to the subject, it must be remembered that it could lead to totally fallacious conclusions as to the causation of the disease.

Turning now to a representative selection of pathological studies, the findings of Lander and Davidson,[17] whose brilliant work on the relationship between atelectasis and bronchiectasis has done so much to revolutionise conceptions of the latter disease in this country, will first be considered.

In a series of 140 bronchiectatic lobes removed by

surgical operation from patients at the Brompton Hospital, they declared that evidence of infection was negligible. "A histological study ... shows not only an intact mucous membrane, very often ciliated, ... but also the presence of all the normal layers of the bronchial wall." The only exceptions to these observations were found in sections through the saccules, and then only in saccules peripherally situated. Here, the walls were found to consist only of fibrous tissue with an epithelial lining. The authors suggested that these changes were probably due to extreme dilatation of minute bronchi and bronchioles, which normally contain no glands, no cartilage, and a minimal amount of muscle. Pleural adhesions were seldom seen. All the lobes showed atelectasis to a greater or lesser extent.

Robinson[27] examined 16 lobes removed by James from 10 patients with results very different from the foregoing. By far the most consistent finding was "a chronic inflammatory condition of the bronchial walls with various degrees of damage up to complete destruction of the musculo-elastic tissue." He discovered no evidence of significant pulmonary collapse. As in the previous series, however, pleural adhesions were rare, but it must not be imagined that unanimity even on this point is universal. Goodman[28], reviewing the results of a similar investigation which he had conducted, declared, "The lobes in all cases were markedly adherent to the surrounding structures in the chest."

Sauerbruch[29] described two types of bronchiectasis, a congenital type in which infective processes were little in evidence, and an inflammatory type in which they were present in greater or lesser degree. The "congenital" variety will be considered later. In the inflammatory

type, Sauerbruch[29] found that the different layers of the bronchial wall could be easily discerned, but the muscle and elastic fibres were split up, there was metaplasia of the epithelium, and resorption, even calcification of the cartilage. Granulation tissue capable of leading to clinical haemorrhage was seen, and the neighbouring pulmonary tissue was sometimes destroyed. Atelectasis was not a feature.

The results of these investigations are surprisingly diverse. Indeed, if the observers were politicians, and not passionless scientists, one would be tempted to infer that they found what they were looking for.

Ogilvie[18], who reviewed the findings of these observers among others, found that "such a conflict of experience is disconcerting and is difficult to explain as coincidental."

He therefore conducted a pathological examination of 35 bronchiectatic lobes excised from 28 patients, and his investigations are so obviously conducted with impartiality and painstaking care, that the results are worth setting down in a somewhat extensive summary.

Macroscopic Appearances.

The pleura was much thickened,and adhesions were present in 25 cases. In the other 10 specimens, changes were of a minor nature. He states that this does not lend support to the view that pleural changes are usually slight or absent.

The author then gives the naked eye findings in the specimens, pointing out that small sections of a lobe can give a completely erroneous picture of the pathology as a whole.

The table in which he summarises his observations is

reproduced below:-

COLLAPSE.	NO. OF SPECIMENS.	SIZE OF LOBE.	SCARRING OF PARENCHYMA.	EMPHYSEMA.
Extensive	22	All shrunken	10	6
Local or patchy	9	All normal	4	8
Negligible	4	All normal	1	4

He observes, "The high proportion of specimens showing extensive or apparently complete collapse is noteworthy, as is the fact that in all of these, the lobe was definitely shrunken. Apparent parenchymal scarring was noted in more than one third of the specimens."

Microscopic Appearance.

Ogilvie states:-

"Conditions varied in different specimens and in different parts of the same specimen. In the majority, however, more or less severe bronchial destruction was evident, and all showed well marked inflammatory changes. The condition of the parenchyma varied also, but the preponderance of extensive pulmonary collapse noted in the foregoing account of the macroscopic appearances was confirmed.

Other parenchymal changes were patchy or confluent broncho-pneumonic consolidation and emphysema. Emphysema was noted in all specimens, and varied from small patches to extensive areas, even in specimens in which collapse seemed complete on gross inspection. This last observation was interesting and rather unexpected, as little or no mention of emphysematous areas was made in the literature, and from accounts of atelectasis one gained the impression that collapse was complete.

Endarteritis was noted in all specimens, and in many it was gross and widespread. Lymphoid hyperplasia was conspicious throughout, and in most specimens there was a pronounced increase in the number of bronchial mucous glands. The latter changes are regarded as important, because they are so typically associated with inflammation of long standing."

Ogilvie, for the purposes of more detailed description then divided his specimens into two groups, an "atelectatic" group, where collapse was a prominent feature, and a "broncho-pneumonic" group, where it was not.

1. Specimens in which pulmonary collapse was extensive; the atelectatic group. This group comprised 21 specimens out of 31 examined.

"The bronchial lining was usually complete, though in places a break in continuity was observed. The lining was composed of columnar and squamous epithelium, and in most bronchi the transition from one to the other was seen. Cilia were rarely observed. Certain of the bronchi were lined completely by columnar or by squamous epithelium, as the case might be, but these were in the minority. A feature of the squamous epithelium was its tendency to be heaped up in layers.

The lumens of the bronchi were frequently stellate in cross section, giving an appearance which has been recorded in the literature, and has been variously interpreted. Opie[30] expressed the opinion that it was due to splitting of the bronchial wall in certain places, with subsequent sealing by scar tissue. Bronchial inflammation was easily apparent in all specimens, but wide variations in the condition of the wall were seen.

All grades of inflammation were observed, from the presence of a complete muscle layer and absence of scar tissue to more or less complete replacement of the bronchial structure by organising granulation tissue showing evidence of scarring.

In some specimens, the majority of the bronchi were relatively normal though inflamed, whereas in others most of them were extensively deranged. Some bronchi occupied a roughly intermediate position. In general, the cartilage did not appear to be grossly affected.

Eleven specimens (52%) showed evidence of severe destruction. Eight specimens (38%) showed evidence of more moderate destruction and two specimens (9.5%), evidence of slighter damage. It is clear that bronchial destruction was, on the whole, a prominent feature in those specimens.

Along with bronchial inflammation were noted changes in the vessels in the form of endarteritis, hyperplasia of the lymphoid tissue, and increase in the number of mucous glands."

He also noted that sometimes the normal bronchial structure was replaced by vascular granulation tissue.

Collapse was often far from complete, and emphysema was a prominent feature.

Parenchymal scarring was present to some extent in 10 specimens, but was mainly peribronchial, and patches of broncho-pneumonia were seen though inconspicuous.

2. Specimens in which collapse was patchy or negligible; the broncho-pneumonic group. This group comprised 10 specimens, 8 (80%) of which showed evidence of severe destruction,and 2 (20%) evidence of moderate destruction of the bronchial walls and lung parenchyma.

He called this the broncho-pneumonic group, because, though the bronchial appearances were similar to those of the previous group though more severe, broncho-pneumonic consolidation and parenchymal scarring were prominent. Collapse was "relatively" absent, though noted in some specimens.

For the most part, patchy or confluent consolidation, peribronchial in distribution, alternated with areas of emphysematous or airbearing pulmonary tissue, and evidence of destructive pulmonary changes was noted in a few areas.

Summing up his findings, Ogilvie stated:-

"It appears from this pathological study that bronchiectatic lobes removed from living patients commonly show pronounced changes.

Not only was considerable inflammation a prominent feature, but actual destruction of the bronchial wall was frequently found. Those other changes which are to be expected in association with chronic bronchial inflammation of any severity were observed. These were endarteritis, lymphoid hyperplasia, and hypertrophy of mucous glands.... Extensive collapse of the parenchyma was frequent in the excised lobes, though it was never really complete. These atelectatic lobes were by no means free from inflammation and bronchial destruction, however, quite the contrary. That these changes were the direct result of infective processes was indicated by the constant replacement of destroyed elements by granulation tissue, which showed definite evidence of organisation into scar tissue in the usual way."

He also commented on the large number of cases in which pleural adhesions were found in his study, and

stated in corroboration that a study of the records of 50 lobectomies on bronchiectatic subjects performed by Mason revealed that only 7 cases had a free pleura, while 21 showed dense adhesions. These findings, he points out, are the reverse of Lander's.[17]

His findings are also, of course, directly opposed to those of that author in point of prevalence of infection in the bronchi and the lung tissue, and he quotes Opie and his associates,[30] Erb,[31] McNeil et alia,[32] and MacCallum[33] in support of his views.

The most recent pathological investigation which will be considered, is that of Lisa and Rosenblatt,[34] the results of which were published in 1943. These authors are determined opponents of the atelectatic theory of the causation of bronchiectasis, and strenuous advocates of the view that infective processes are solely to blame. It is therefore interesting to compare their findings with those of Lander and Davidson,[17] the authors who have done so much to popularise the view that atelectasis is the prime causative factor. A short summary of their observations will therefore be given, and their article will be discussed in more detail when the causation of bronchiectasis is considered.

Lisa and Rosenblatt[34] found that in every case there was evidence of infection. The bronchi were dilated, and the walls usually thickened by fibrous tissue replacement. The epithelium was intact, though in some places modified, but relatively unimpaired. Plasma cell infiltration, and moderate lymphoid infiltration were features. Fibrosis of the parenchyma to a greater or lesser extent was found in almost every case. The blood vessels were generally healthy.

The authors maintained that the greater the destruc-

tion of the bronchial wall and parenchyma, the bigger the cavity. They also found that the only element of the bronchial wall which showed evidence of regeneration was the epithelium.

Atelectasis was not a prominent finding in this series.

Comment.

In the pathological investigations which have been studied, it is important to remember that in the lobes examined, the disease had been present for an indeterminate period. The findings are therefore not strictly comparable. Yet the diversity of results remains astonishing. Lander and Davidson,[17] who hold the view that atelectasis is the root cause of bronchiectasis, find little evidence of infection in the affected tissue. Lisa and Rosenblatt,[34] on the other hand, who believe that infection is solely responsible for the condition, discover it in every case.

The lungs examined by the latter authors, however, were all removed after death, and 68 of the 110 subjects studied were over 50 years of age, while only 14 were under 30. Since Lander and Davidson's[17] series was based on an investigation of lobes removed by surgical operation from presumably comparatively young patients, they were doubtless studying the disease at a much earlier stage. Nevertheless, their finding that evidence of infection was negligible must be regarded as quite exceptional even in lobectomy specimens. It is true that in all such series, cases are noted in which inflammatory changes are very slight, and this is a finding of first class importance. Ogilvie,[18] as has been seen, reported that "All grades of

inflammation were observed, from the presence of a complete muscle layer, and the absence of scar tissue, to more or less complete destruction of the bronchial wall," and even Lisa and Rosenblatt[34] admit that evidence of infection, though always present, is sometimes slight.

On the whole, however, it seems clear that some degree of infection is commonly present in the type of lobe which has so far been submitted to pathological investigation, but it must not be forgotten that it is the comparatively late stages of the disease which are being observed, even in a lobectomy series. It is safe to say that in the vast majority of cases, the surgeon is dealing with a patient who has been troubled with his chest for years before he comes to operation.

It will be shown later that in recent times more and more cases of practically symptomless bronchiectasis have been discovered by chance examination of the thorax, and in these instances it seems incredible that infection can be at all marked. Naturally, in this type of case there is little chance of a pathological investigation, as few surgeons would care to perform an operation on a patient who is apparently well.

It is clear, therefore, that as an approach to an understanding of the causation of bronchiectasis, pathology has its limitations. It tends to investigate end results rather than beginnings.

With regard to pleural adhesions, Lander and Davidson,[17] and Robinson[27] stated that they rarely encountered them, while Ogilvie[18] and Goodman[28] described them as usual concomitants of the disease.

In the course of the present investigation, artificial pneumothorax was induced in 15 cases of bronchiectasis.

Three were instances of "reversible bronchiectasis," and may therefore perhaps better be discounted. One of these showed a partially adherent pleura which was almost certainly due to an old tuberculous lesion, as the child had had this disease at one time, and as the adherent area was over the upper lobe and not over the bronchiectatic middle lobe. The other two cases had a free pleura.

The remaining cases were all examples of established bronchiectasis at a stage comparable to that seen in the lobectomy series which have been quoted. It is interesting to be able to record that in only one instance was an adherent pleura noted, and that to a very limited extent. A free pleura, therefore, can scarcely be regarded as a rarity in bronchiectasis. Radiograms and bronchograms of some of the cases are shown later in the work.

Ogilvie[18]'s finding that emphysema was present in all his cases is interesting, because as he points out, it is rarely stressed in the literature on the pathology of bronchiectasis, though it may be observed that Andrus[35] had already noted its constant occurrence, and emphasised its significance in 1937. This phenomenon, however, will come as no surprise to anyone who has examined many radiograms of the disease. Twining[36]'s observation that emphysema is a usual feature in x-ray plates of atelectasis has already been mentioned in the chapter dealing with the diagnosis of that condition, and it is equally common in bronchiectasis; in fact, the "ring markings" were formerly frequently mistaken for the dilated bronchi themselves.

With regard to the prominence of atelectasis as a pathological feature of bronchiectasis, perhaps Ogilvie[18]'s figures may be looked upon as giving a reasonable approximation to what one may expect to find. He holds no

strong views on the relationship of atelectasis to bronchiectasis, and yet, writing as late as 1941, is fully aware of the importance attached to it by many authors in recent years. His findings are therefore likely to be unbiassed and reliable, though, of course, the chance that the series of lobes he studied was unusual, cannot be excluded.

When that is said, the striking fact emerges from his investigations that as many as 21 bronchiectatic lobes out of the 31 submitted to microscopic examination showed evidence of extensive atelectasis, and some of the other lobes, though not affected to the same extent, also showed evidence of the condition.

Summing up the results of the pathological investigations that have been reviewed, it seems clear that although much of the evidence is conflicting, there is no doubt that atelectasis and infection are common concomitants of bronchiectasis, though the extent to which they are present varies widely in individual cases.

This agrees with clinical and radiological findings, as will later be seen.

BACTERIOLOGY.

The literature on the bacteriology of bronchiectasis is more voluminous than enlightening. Practically every organism which has been found in the lung has been put in the witness box and cross-examined, just as almost every respiratory disease has been condemned as a causative factor.

Smith[37], and Pilot and Davis[38] for example, accuse Vincent's spirochaete of causing the condition, while Pfeiffer's bacillus has been implicated by Leys[39], Blake and

Cecil,[40] and Opie and his associates.[41] Nor have even intranuclear inclusion bodies 'scaped calumny, for McCordoch[42] demonstrated them in the lung in whooping cough. He claimed that the interstitial broncho-pneumonia associated with this disease is due to a filterable virus with secondary bacterial infection, and that the destruction of the bronchial wall in such cases predisposes to the development of bronchiectasis. Needless to say, the pneumococcus and streptococcus have often been held responsible.

Like the pathology of bronchiectasis, the bacteriology is therefore somewhat perplexing.

Fortunately, in 1932, Greey[43] published the results of a most extensive investigation, in which he comes to the convincing and not wholly surprising conclusion that though there is a great variety of organisms associated with bronchiectasis, there is no specific or even predominating organism which can be regarded as the cause.

His findings are supported by Lisa and Rosenblatt[34] writing in 1943.

SECTION 3.

CLASSIFICATIONS OF BRONCHIECTASIS.

The classical description of bronchiectasis is one of a disease characterised by offensive breath, paroxysmal coughing, the production of large quantities of foul-smelling sputum, evidence of toxic absorption, clubbing of the fingers, and physical signs of cavitation in the lung. Cases of this nature, however, nowadays fortunately form a comparatively small proportion of the number dealt with by the chest physician. Advances in diagnosis, stimulated above all by the introduction of bronchography, have led to the recognition that this is merely the most advanced stage of the disease.

Leaving aside for the moment the controversial "reversible bronchiectasis", permanent cases are generally classified for convenience according to the severity of the symptoms, the distribution of the disease in the lungs, and the appearance of the bronchial dilatations.

Cases of bronchiectasis will therefore be considered under the following headings:-

A. SEVERITY OF SYMPTOMS.
B. ANATOMICAL DISTRIBUTION.
C. CHARACTER OF THE BRONCHIAL DILATATIONS.

A. SEVERITY OF SYMPTOMS.

Lisle Punch[44] recognised cases of bronchiectasis as falling into three groups based on the severity of symptoms.

1. Silent Bronchiectasis.
2. Bronchiectasis with Mild or Moderate Symptoms.
3. Bronchiectasis with Severe Symptoms.

This type of classification is fairly frequently employed, and the different categories suggested by Lisle Punch[44] will now be discussed.

1. Silent Bronchiectasis.

Under the first heading come examples of so called "dry" bronchiectasis which was recognised by Pinchin and Morlock[45] in 1930, and Moll[46] in 1932, though it is commonly associated with the names of Wall and Hoyle,[47] who published their well known article on the subject in 1933. These authors pointed out that many people displaying few of the classical symptoms of bronchiectasis, had undoubtedly permanent bronchial dilatation, but would never have been recognised as having the disease had it not been for the comparatively recent technical aid to diagnosis - bronchography. These patients had an unproductive cough, often not very troublesome, and with periods of remission. The symptom which led them to seek medical advice was occasional haemoptysis. Tuberculosis was, of course, suspected, but radiology failed to disclose evidence of that disease. Bronchography, however, revealed the bronchial dilatation.

These findings naturally raised the question as to whether previous conceptions of bronchiectasis had not been somewhat erroneous. If cases of the disease are encountered which would never have been recognised save for the occurrence of a haemoptysis, might there not be patients with bronchiectasis who remain undiagnosed because of the absence of that alarming symptom? Lisle Punch,[44] writing in 1939, stated that "If more cases of persistent dry cough were investigated in this manner (i.e. by bronchography), I am inclined to think that more cases of

bronchiectasis unassociated with haemoptysis would be detected."

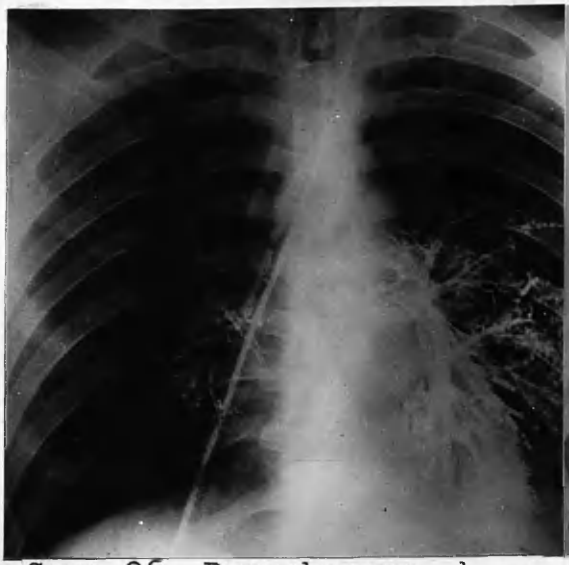
This contention is supported by the work of Martin and Berridge,[48] who in 1942, collected a series of 25 cases of bronchiectasis the features of which little corresponded to the classical conception of the disease. In many of them cough and sputum production were slight and only seasonal, and one was apparently symptomless. Seven had a history of slight haemoptysis. The patients were soldiers, many in the army category A.1., and the few who had sought medical advice in civilian life had escaped diagnosis. Most of them were well nourished, and showed no evidence of toxaemia; only three had slight clubbing of the fingers. The majority of the patients were admitted to hospital on account of inflammatory episodes, but some came under observation for non-respiratory ailments. Bronchiectasis was only recognised because of the interest of the authors in the disease, and the frequency with which bronchography was performed.

Further confirmation for Lisle Punch's[44] suggestion is also provided by Wearing[49] who in 1948 published the results of a bronchographic investigation of 214 Service men and women showing symptoms suggestive of chronic bronchitis. He discovered that bronchiectasis was present in 46 cases (21%). Wearing emphasised that the clinical findings did not include any of the features generally considered to be characteristic of classical bronchiectasis.

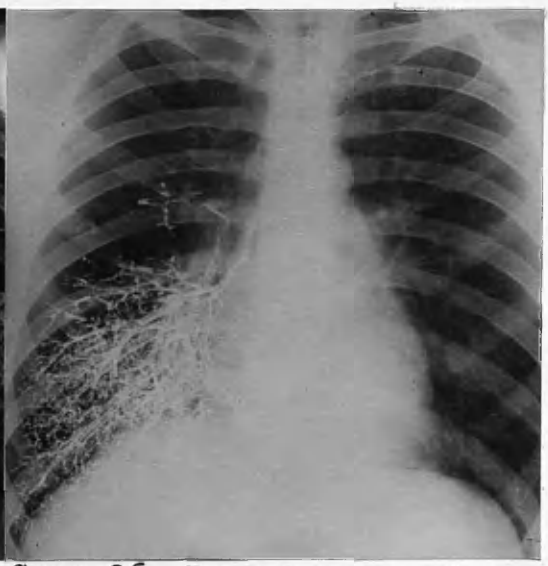
The two cases which follow are illustrative of "silent" or "dry" bronchiectasis.

Case 26.

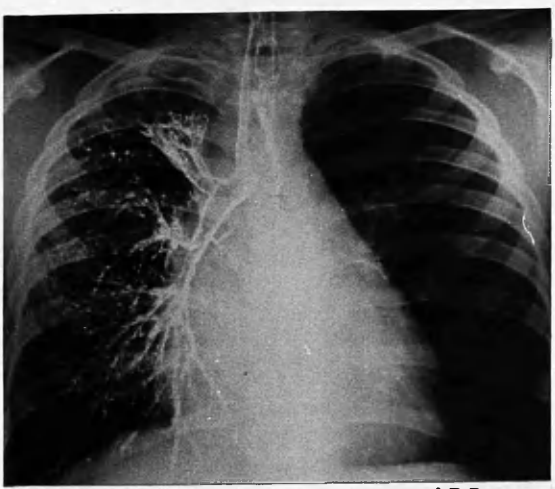
Male aged 22 years, notified as a pneumonia, and admitted to Ruchill hospital on 20/5/47. Examination revealed prolonged rather tubular expiration at the left



Case 26. Bronchogram showing cylindrical bronchiectasis in left lower lobe.



Case 26. Bronchogram of rt. lung shown to contrast the calibre of the rt. lower lobe bronchi with that of the left lower lobe bronchi.



Case 27. Bronchogram illustrating right upper lobe bronchiectasis.

base accompanied by medium moist crepitations. The temperature was elevated to 102. F., and there was a slight cough which produced a small amount of greenish-yellow sputum (less than 1 oz. per day). All the symptoms subsided in three days, but expiration at the left base remained prolonged and rather tubular, though not now accompanied by crepitations. There was no clubbing of the fingers. The illness had a duration in all of only 5 days.

The patient looked well, was six feet one inch in height, powerfully muscled, and had been demobilised from the Army a month before admission to hospital in category A.1., the grade in which he entered four years previously. He had experienced no illness of any kind during his service career in spite of the hard training and rigorous climatic conditions to which he had been exposed. Careful questioning failed to elicit any history suggestive of significant chest disease. The most he would admit was that he occasionally had a cold and cough in winter, but not, so far as he was aware, more than other people. He could not remember having a persistent cough after any boyhood illness such as whooping cough or measles, nor was there any history of haemoptysis. Because of the findings noted at the left base, and the interest of the writer in bronchiectasis, bronchography was, however, carried out. It revealed a uniform cylindrical dilatation of the bronchi of the left lower lobe. The bronchogram is shown opposite. The patient was dismissed from hospital on 12/5/47, and has been kept under observation. Over a year has elapsed since he was first seen, and so far he has remained symptomless.

Case 27.

This case is an example of bronchiectasis in which there were no symptoms of the disease at all, and the presence of the condition was suspected solely on the findings of a routine physical examination of the chest. The patient was a boy, nine years of age, who was being treated in Ruchill Sanatorium for a minimal tuberculous lesion of the left lung. Examination revealed bronchial breathing in the right upper lobe. There were no adventitiae, and the P.N. was resonant. The excellent general condition of the patient, the absence of cough and production of sputum, and a normal temperature, ruled out a tuberculous broncho-pneumonia. Bronchography revealed dilatation of the right upper lobe bronchi. The bronchogram is shown opposite. This patient exhibited none of

the symptoms usually associated with bronchiectasis, and in view of the excellent drainage in the upper lobe situation, it seems unlikely that he ever will. The mechanism of production of the condition will be later discussed.

Cases of "silent" bronchiectasis obviously present difficulties in prognosis and treatment as well as in diagnosis, and this aspect will be considered in a subsequent chapter.

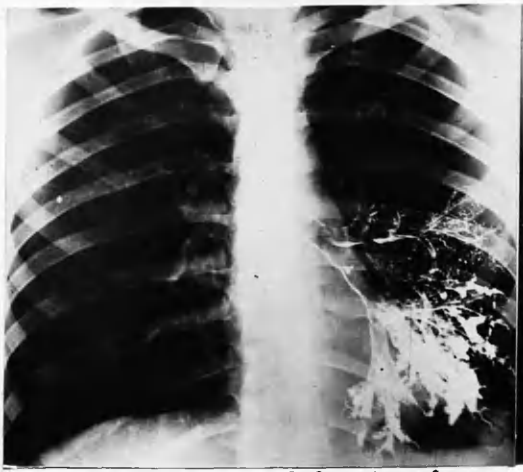
2. Bronchiectasis with Mild or Moderate Symptoms.

Cases of this degree of severity are the ones most commonly associated in the minds of modern chest physicians with bronchiectasis. There is a history of a persistent cough and moderate production of sputum (2 or 3 ounces per day), purulent, but not commonly offensive. There is no clubbing of the fingers or evidence of toxaemia, and little deterioration of the general condition. The physical signs are often slight - generally impairment of the percussion note at one base, usually the left, with weak broncho-vesicular breathing accompanied by medium, moist crepitations.

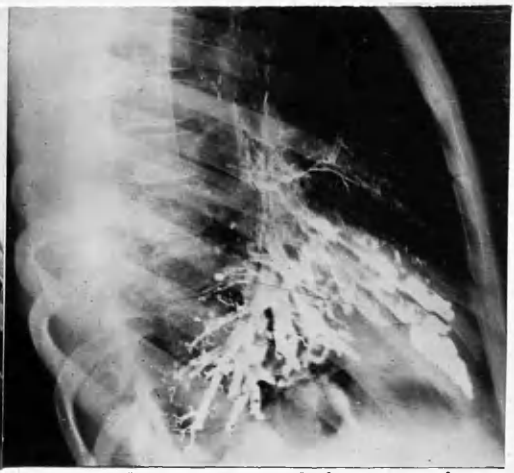
Lisle Punch[44] mentions that an occasional haemoptysis is rather common, but this was not a feature of any prominence in the cases seen in the course of the present investigations. Apart from chronic cough, the most suspicious history, especially in the case of children, was one of repeated attacks of "pneumonia."

Case 28.

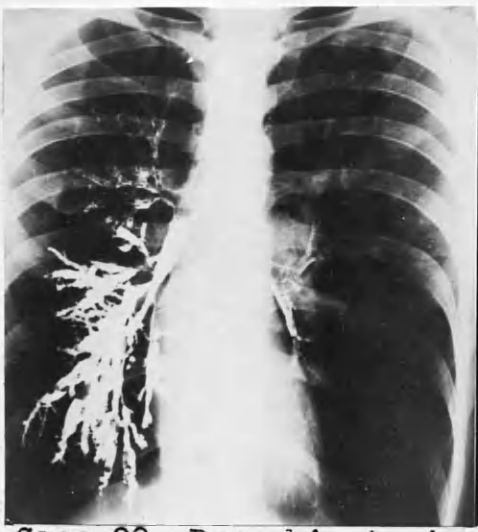
Male, aged 12 years, admitted to Ruchill Fever Hospital on 12/3/47 with "pneumonia." This was his fourth attack in two years. There was impairment of the P.N. at the left base, broncho-vesicular breathing, and a few moist crepitations. The temperature was 101. F. on admission, and settled in 3 days. There was a history of a persistent cough of some years duration. Cough while



Case 28. Bronchiectasis left lower lobe and lingula. P.A. bronchogram.



Case 28. Bronchiectasis left lower lobe and lingula. Lateral bronchogram.



Case 29. Bronchiectasis throughout rt. lung. P.A. bronchogram.

Case 29. Lat. bronchogram. Note how this view displays saccular bronchiectasis in the pectoral segment of the rt. upper lobe, not apparent in P.A. view.

in hospital was most marked in the mornings, and associated with sputum production of about 2 oz. per day. There was no clubbing of the fingers, and the general condition of the patient was good.

The bronchograms opposite show bronchiectasis of the left lower lobe and lingula.

3. Bronchiectasis with Severe Symptoms.

Cases of this nature are still, unfortunately, quite frequently seen. All the classical symptoms of bronchiectasis are present, that is to say, cough, production of evil smelling sputum, foul breath, toxaemia, and clubbing of the fingers.

Case 29.

Male, aged 37, admitted to hospital on 30/10/47 with "pneumonia." The temperature was 102. F. on admission, and settled in a few days. There was a history of chronic cough associated with the production of large quantities of foul sputum, especially in the mornings. Examination revealed medium crepitations in the lower lobes of both lungs, and rhonchi all over the chest. There was marked clubbing of the fingers, foul breath, and copious production of evil smelling sputum. Bronchography of the right lung was carried out, and revealed that there was bronchiectasis in every lobe, though in the right upper lobe only the pectoral branch of the right upper lobe bronchus showed marked sacculation. The left bronchial tree was not visualised as the patient's condition was poor, the diagnosis certain, and operation out of the question. He died five months later. Bronchograms are shown opposite.

B. ANATOMICAL DISTRIBUTION.

Bronchiectasis is frequently classified according to the lobe or lobes involved.

1. Upper Lobe Bronchiectasis.

Bronchiectasis of an upper lobe alone is uncommon except in association with tuberculosis, though bronchiectasis of the "silent" variety is sometimes seen. This

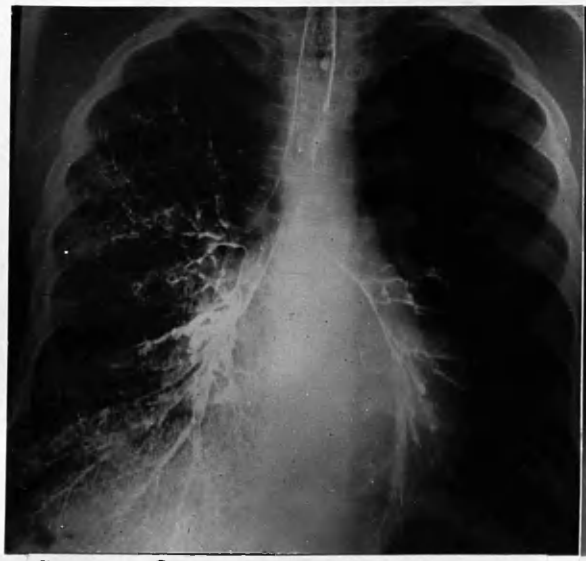
type, is, in all probability in the majority of cases due to a previous tuberculous collapse which has cleared up; reasons for this statement will be advanced when the causation of bronchiectasis is discussed.

It is obvious that cases of upper lobe bronchiectasis may present signs on physical examination consistent with tuberculous cavitation, but the differentiation of the two conditions does not present great difficulty. The good general condition, absence of toxaemia, and failure to find the tubercle bacillus on repeated examinations of the sputum, helps to exclude tuberculosis, while straight x-ray and bronchography clinch the diagnosis.

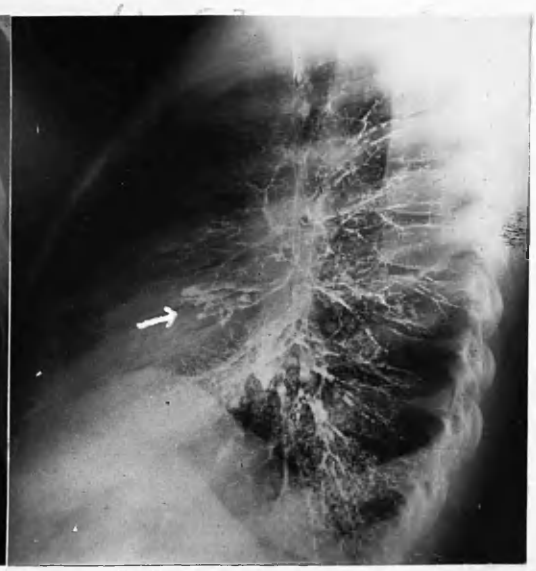
A case already shown (Case 27, p.68.) to illustrate silent bronchiectasis, also illustrates upper lobe bronchiectasis.

2. Right Middle Lobe Bronchiectasis.

Bronchiectasis confined to the right middle lobe is not at all uncommon. As with atelectasis in this region, the condition may very easily be missed, as no signs may present themselves on physical examination, particularly if there is an associated atelectasis, because, as has already been pointed out, the upper lobe may overlap its shrunken neighbour. It will be seen from the foregoing, that cases of bronchiectasis involving only the right middle lobe will be suspected by the physician more from the symptomatology, than from suspicious physical signs. In the experience of Lisle Punch,[44] slight haemoptysis was the feature which prompted an investigation. In the cases seen at Ruchill, a persistent cough with or without the production of sputum, was the leading symptom.



Case 30. P.A. bronchogram.



Case 30. Lat. bronchogram. Note how this view much more clearly displays the rt. middle lobe bronchiectasis.

A lateral bronchogram is always essential for accurate localisation of bronchiectasis no matter what portion of the lung is affected, but dilatation of the middle lobe bronchi may be overlooked altogether if this is not available.

Case 30.

Male, aged 23 years, referred from a tuberculosis clinic to Ruchill Sanatorium for investigation. There was a long history of persistent, though not severe cough, with some production of sputum. Physical examination revealed no signs of disease, and straight x-ray negatived tuberculosis. Bronchography demonstrated bronchiectasis of the right middle lobe. The bronchograms are shown opposite.

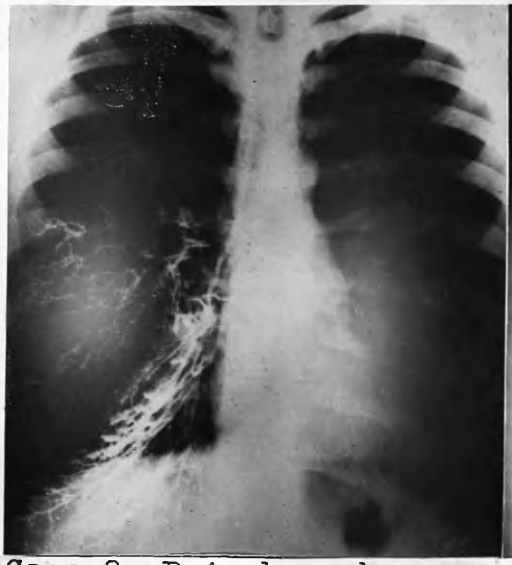
3. The Lingula. (Lingular Process of the Left Upper Lobe).

Bronchiectasis in the lingula alone is rather rarely seen, though it occasionally occurs. Physical signs, as in the case of the right middle lobe, are generally absent, and here again a lateral bronchogram is essential.

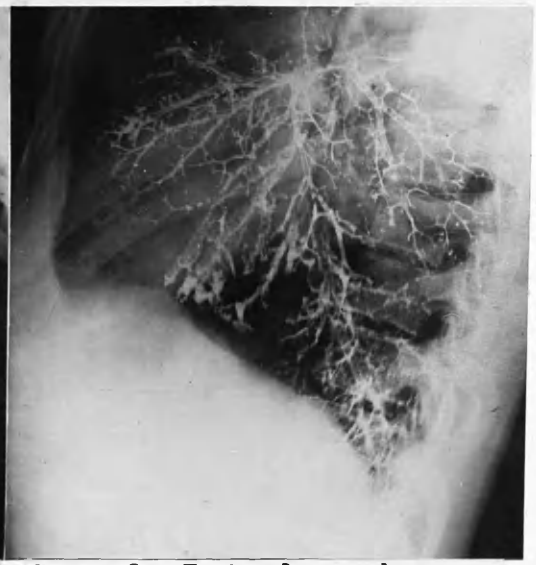
Bronchiectasis in the lingula is commonly associated with bronchiectasis in the left lower lobe, and as Tudor Edwards[50] points out, where surgery is contemplated, great care must be taken not to overlook it. Examples will be shown later.

4. Lower Lobe Bronchiectasis.

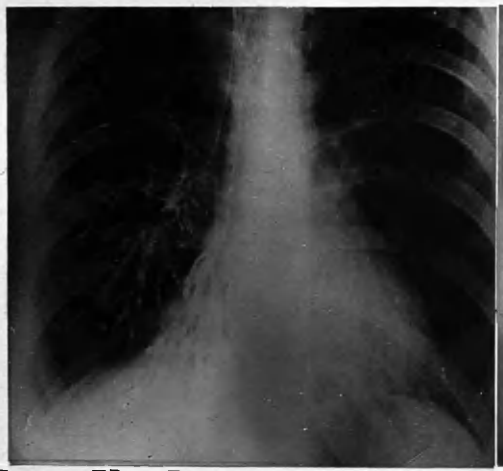
The lower lobes are by far the commonest sites of bronchiectasis, the left being predominantly affected. The physical signs - impaired P.N., weak bronchial or broncho-vesicular breathing, and medium or coarse crepitations have already been mentioned, and are frequently present. There is often an associated atelectasis, and there thus may be a triangular area of dullness close to



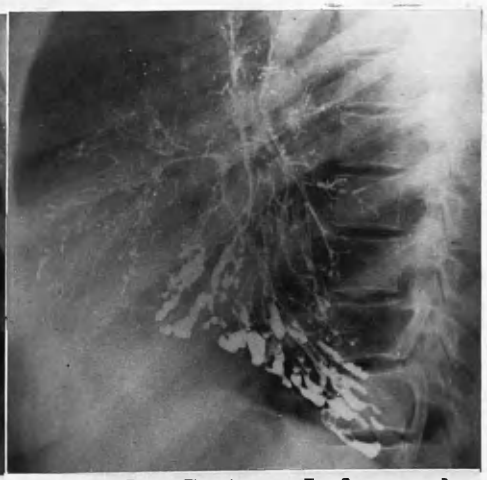
Case 8. P.A. bronchogram.



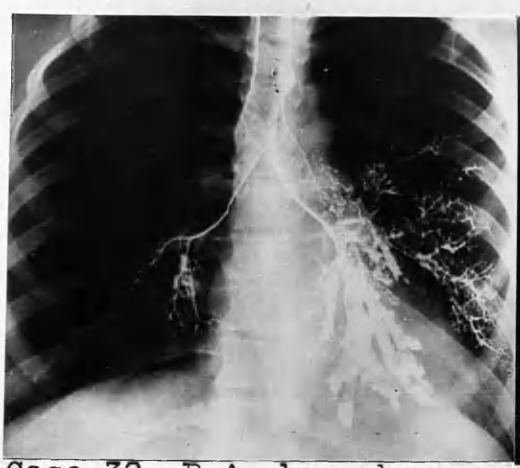
Case 8. Lat. bronchogram showing that the bronchiectasis is almost confined to the anterior basal segment of the rt. lower lobe.



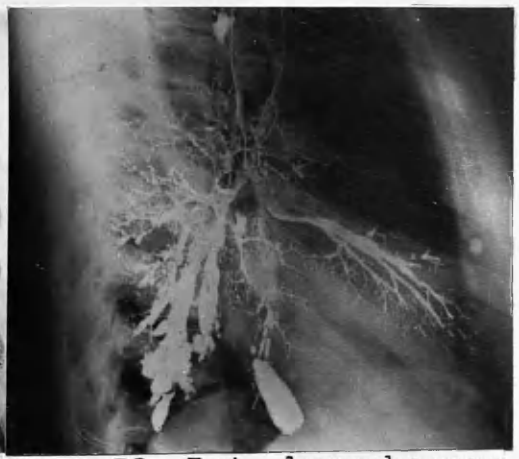
Case 31. P.A. bronchogram.



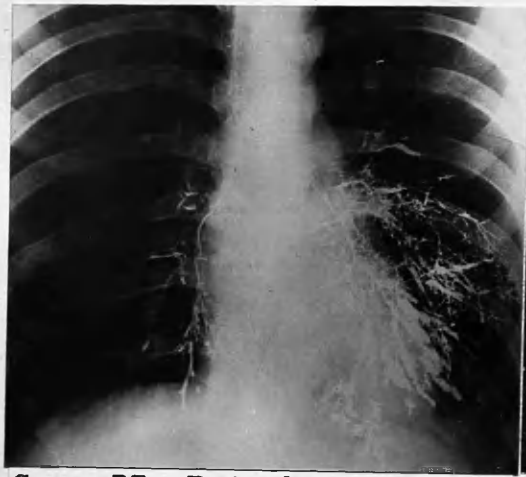
Case 31. Lateral broncho gram.



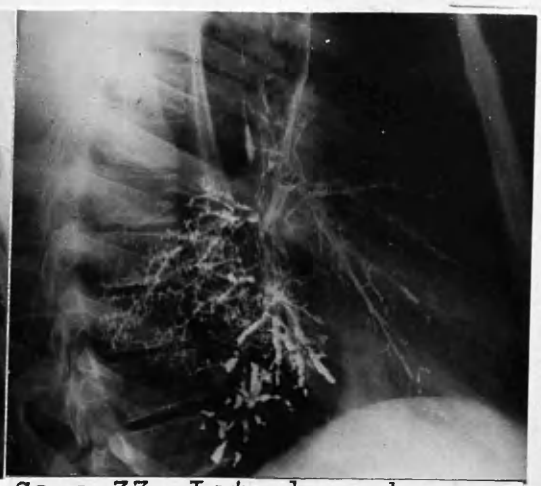
Case 32. P.A. bronchogram. Left lower lobe bronchiectasis.



Case 32. Lat. bronchogram showing that the bronchiectasis affects chiefly the posterior and axillary basal segment of the left lower lobe. There is radio-opaque medium in the oesophagus.



Case 33. P.A. bronchogram.



Case 33. Lat. bronchogram.

the spine. Lateral bronchograms demonstrate the particular segments of the lobe which are affected. A few typical cases follow.

Case 8.

Male aged 25 years, with right lower lobe atelectasis and cylindrical bronchiectasis. This case is fully discussed later. Bronchograms are shown opposite. A straight x-ray is shown opposite page 10.

Case 31.

Female aged 17 years, admitted for investigation on 12/11/46. The signs and symptoms were consistent with bronchiectasis of the right lower lobe, and this was confirmed by bronchography. The middle lobe bronchi were not involved though the dilated anterior basic bronchi of the right lower lobe are difficult to differentiate from them in the photograph shown opposite.

Case 32.

Female aged 25 years, admitted to hospital on 5/6/47 with the usual signs and symptoms of bronchiectasis in the left lower lobe. Bronchography confirmed the diagnosis. Bronchograms are shown opposite.

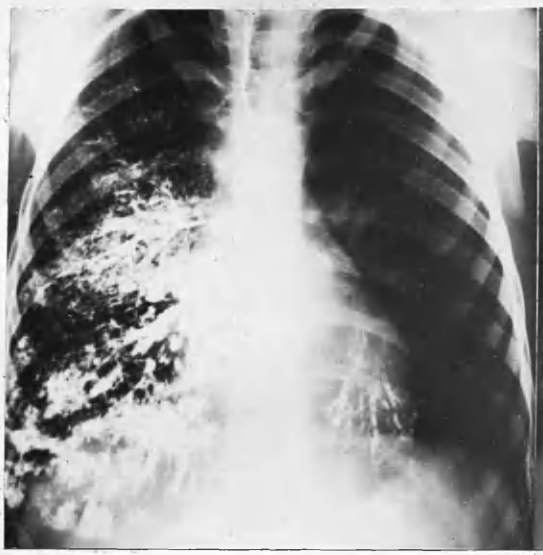
Case 33.

Male aged 22 years, admitted to hospital on 7/11/47 with bronchiectasis of the left lower lobe. Bronchograms are shown opposite.

5. Combinations of Lobes.

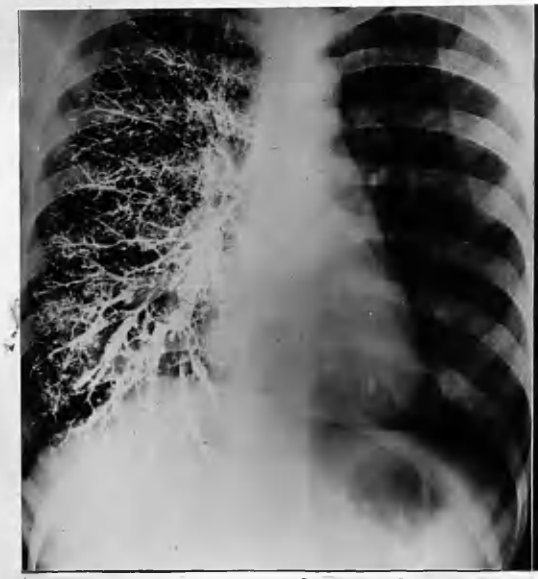
It is common to find that more than one lobe is affected by bronchiectasis at the same time, and as it is important from a surgical point of view that diagnosis should be accurate, a lateral view as well as a postero-anterior view should be taken as a routine.

Simultaneous involvement of the right middle and lower lobes is frequently seen, and the following two cases illustrate the condition.

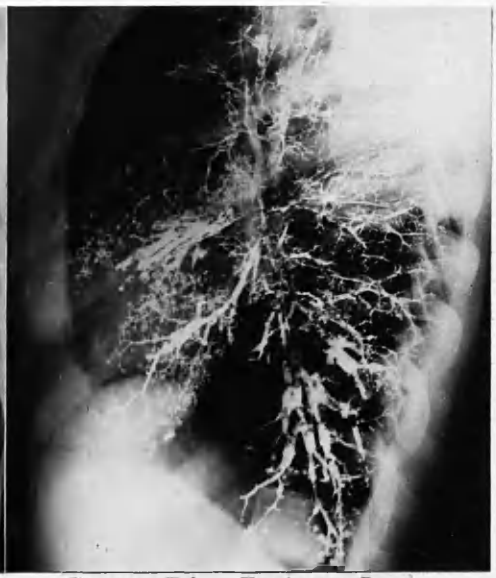


Case 10. P.A. bronchogram. Right middle and lower lobe bronchiectasis.

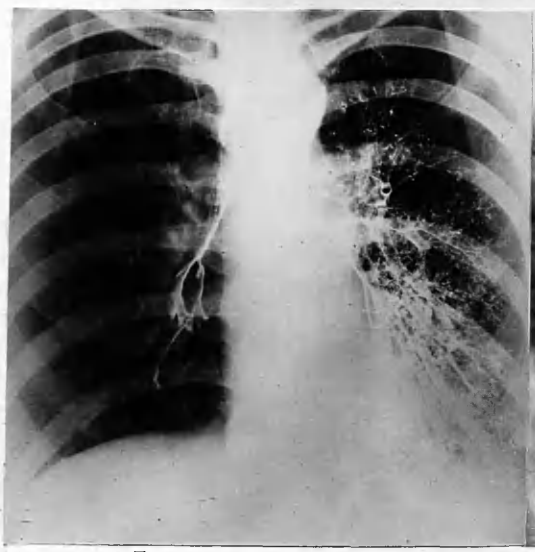
Case 10. Lat. Bronchogram. The blurred appearance of this and the preceding bronchogram is due to the presence of old neohydriol in the lung as a result of former bronchographic investigation.



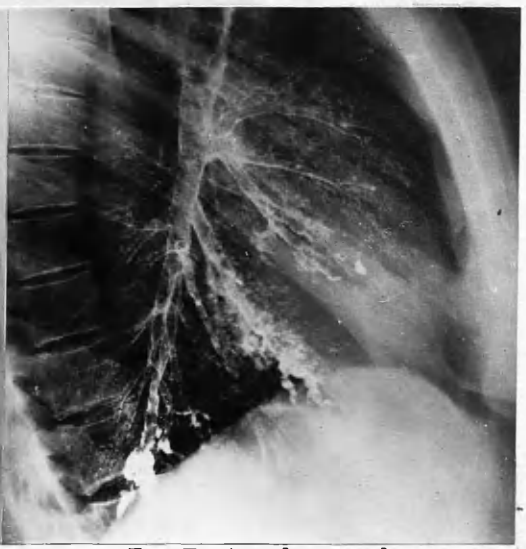
Case 34. P.A. bronchogram.



Case 34. Lateral bronchogram.



Case 35. P.A. bronchogram. Reproduction poor owing to heart shadow.



Case 35. Lat. bronchogram clearly showing cylindrical bronchiectasis affecting left lower lobe and lingula.

Case 10.

Female, aged 6 years, admitted to Ruchill Sanatorium on 12/5/47. Bronchography revealed bronchiectasis of the right middle and lower lobes. This case was extremely interesting in another connection, and is fully discussed later. Bronchograms are shown opposite. A straight x-ray appears opposite page 11.

Case 34.

Female, aged 25 years, admitted to Ruchill Sanatorium on 8/10/46 with a history typical of bronchiectasis, and medium crepitations at the right base. Bronchography revealed bronchiectasis of the right middle and lower lobes. Bronchograms are reproduced opposite.

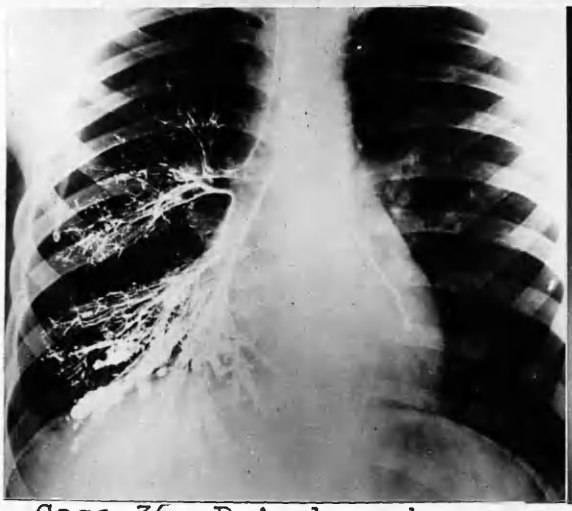
Churchill[51,52] and Tudor Edwards[50], among others, have pointed out that when the left lower lobe is bronchiectatic, the lingula (lingular process of the left upper lobe) is also frequently affected, and failure to note lingular involvement in such cases has often led to surgical removal of the left lower lobe alone with consequent persistence of symptoms.

Bronchograms of a case in which there was simultaneous involvement of the left lower lobe and lingula have already been shown (opposite page 70), and the condition is further illustrated by the following case.

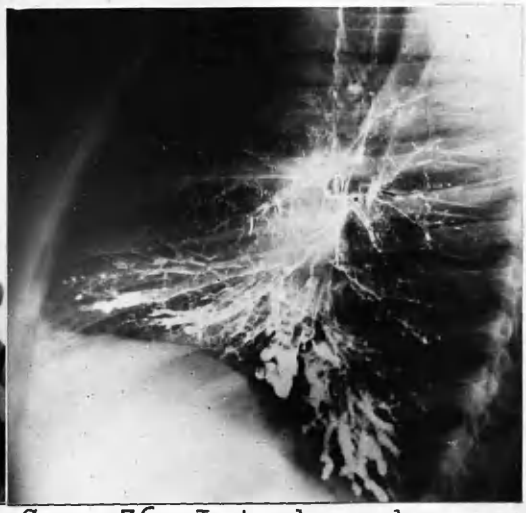
Case 35.

Female, aged 22 years, admitted to Ruchill Sanatorium on 24/10/46. The history was typical of bronchiectasis, and there were medium crepitations at the left base. Bronchography revealed bronchiectasis of the left lower lobe and lingula. Bronchograms are shown opposite.

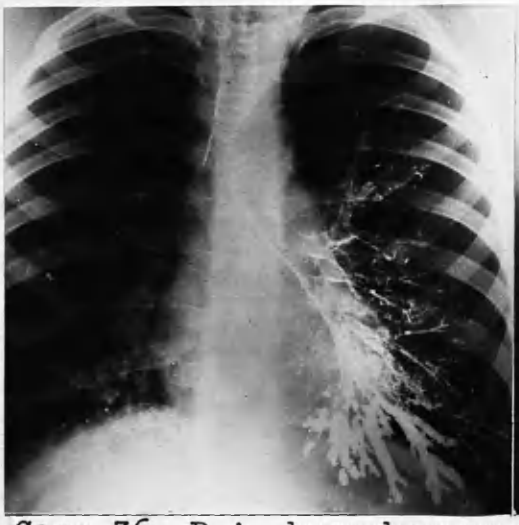
Bilateral distribution of bronchiectasis is very common, which is unfortunate from a surgical point of view. Simultaneous involvement of both lower lobes is particularly frequent. This is illustrated by the following case.



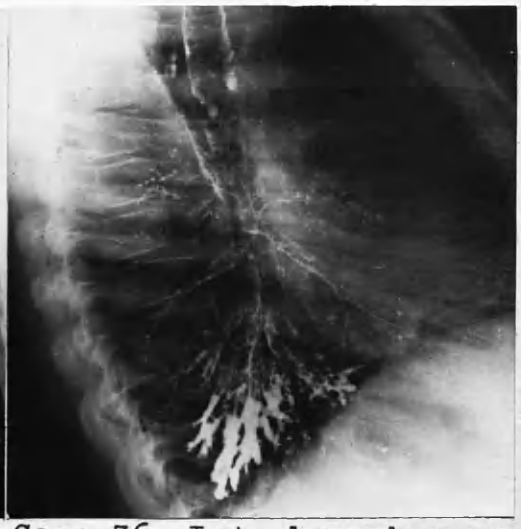
Case 36. P.A. bronchogram right lung.



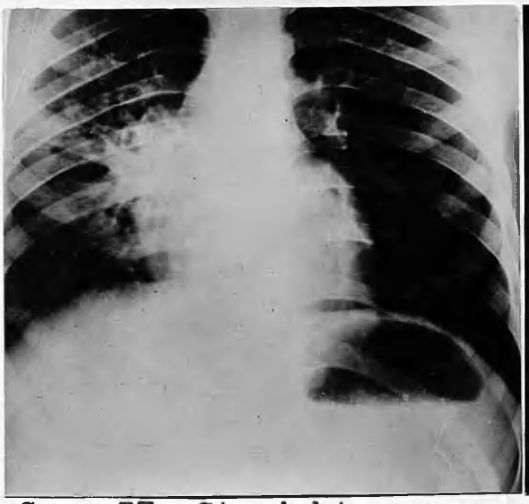
Case 36. Lat. bronchogram rt. lung showing more clearly the rt. middle lobe bronchiectasis.



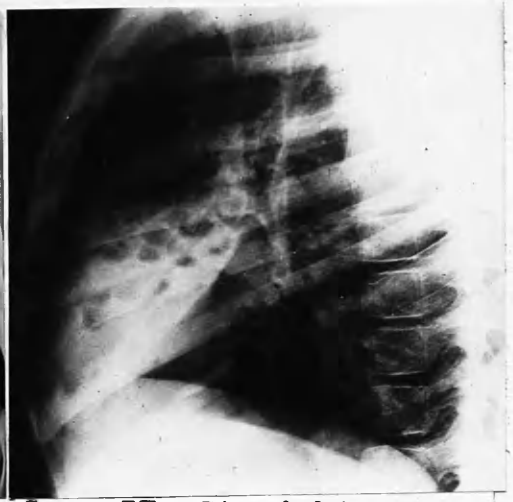
Case 36. P.A. bronchogram left lung.



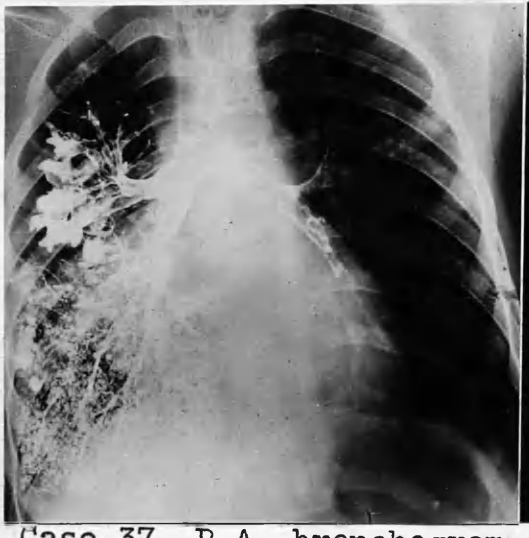
Case 36. Lat. bronchogram left lung.



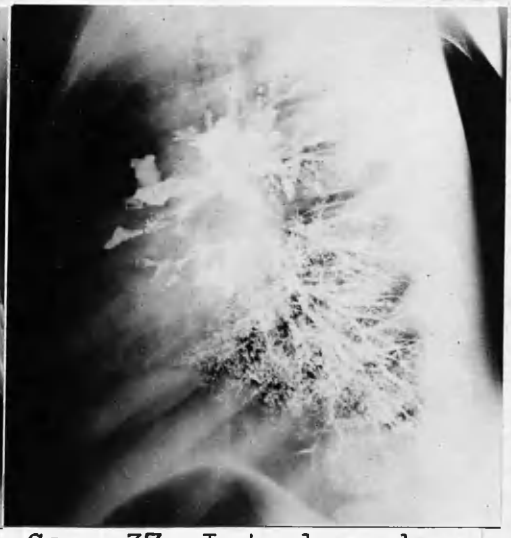
Case 37. Straight x-ray. P.A. view. Appearance suggestive of very marked bronchiectasis affecting the rt. upper and middle lobes.



Case 37. Straight x-ray. Lat. view. Honeycomb appearance in rt. middle lobe area.



Case 37. P.A. bronchogram rt. lung, showing cavitatory bronchiectasis in rt. upper and middle lobes.



Case 37. Lat. bronchogram showing that the radio-opaque medium has not well entered the bronchiectatic cavities in rt. middle lobe.

Case 36.

Male, aged nine years, admitted to Ruchill Sanatorium on 25/11/46 with a history of persistent cough and spit. There were crepitations at both bases. The bronchograms reproduced opposite reveal bronchiectasis affecting the left lower lobe, the right lower lobe and also the right middle lobe.

The following case illustrates a rather rare combination - bronchiectasis of the right upper and middle lobes.

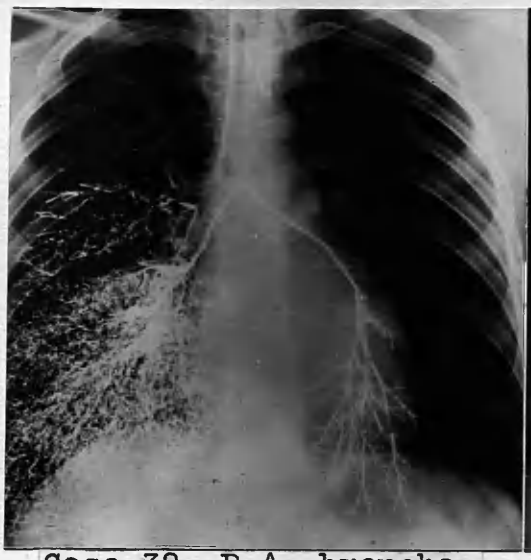
Case 37.

Male, aged 27 years, admitted to Ruchill Sanatorium on 12/3/44 with persistent cough and spit. Bronchography revealed bronchiectasis of the right upper and middle lobes. The bronchograms reproduced opposite were culled from the archives of Ruchill Sanatorium and were not taken by the author.

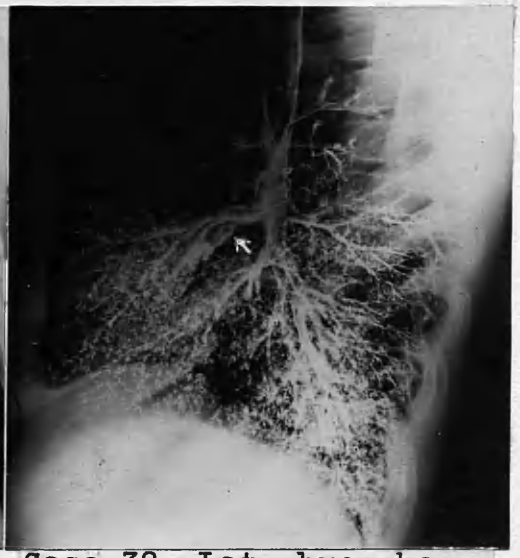
Bronchiectasis, of course, may affect all the lobes of the lung, and an example of this has already been shown (Case 29, p.70.). More striking instances are shown later.

The classification of bronchiectasis just given follows the usual method of grouping cases according to the lobe or lobes affected, but, as will have been noted from a study of the bronchograms accompanying the cases shown, not all the bronchi in a diseased area are equally dilated. Sometimes, indeed, only the bronchi in a particular segment of the lobe are ectatic and as has been mentioned in the first section of the chapter, this fact is of great importance from the point of view of modern surgical treatment.

The following case is an instance in which bronchiectasis appeared to be confined to a secondary bronchus of the right middle lobe.



Case 38. P.A. bronchogram. No evidence of bronchiectasis.



Case 38. Lat. bronchogram. Dilatation of the right middle lobe bronchus indicated by arrow.

Case 38.

Male, aged 19 years, referred from a tuberculosis clinic to Ruchill Hospital for bronchography. The patient complained of cough of about two years duration associated with occasional sputum production. Straight x-ray failed to reveal evidence of active tuberculosis, and the tubercle bacillus was not demonstrated in the sputum. Examination revealed a few crepitations in the right middle lobe area, and bronchography demonstrated ectasia of one of the secondary bronchi of the right middle lobe. Bronchograms are shown opposite.

C. CHARACTER OF THE BRONCHIAL DILATATIONS.

Bronchiectasis is often classified in accordance with the type of bronchial dilatation seen. Cylindrical, saccular, and cavitatory varieties are described.

1. Cylindrical.

In this type, as the name suggests, the dilatation of the bronchi is seen more in their proximal than in their peripheral parts, and is therefore cylindrical in form. Examples of this type have already been shown (Case 26, p.67; Case 8, p.73, etc.).

2. Saccular.

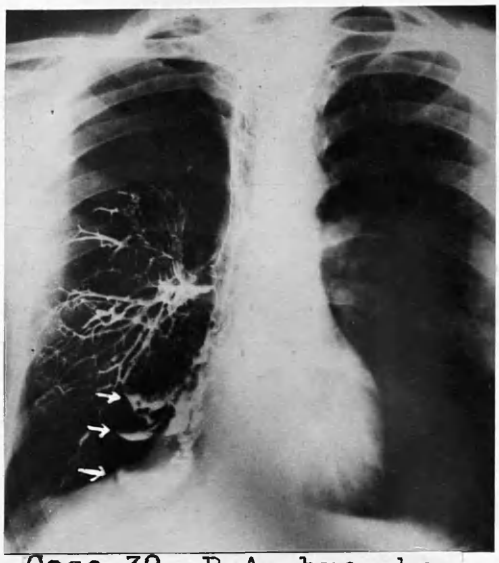
The dilatation here is seen most markedly in the peripheral parts of the bronchi, which assume a saccular form. Examples of this type have already been given (Case 31, p.73; Case 32, p.73; Case 36, p.75, etc.).

3. Cavitatory.

In the advanced stages of bronchiectasis, cavitation may be marked. An example follows.

Case 39.

Male, aged 27 years, admitted on 17/3/47 with long



Case 39. P.A. bronchogram rt. lung. Radioopaque medium lying at bottom of cavities indicated by arrows.

history of cough and spit. Physical examination revealed some impairment of the P.N. at the right base with prolongation of expiration and medium crepitations in the same area. Cough was only marked in the mornings, when copious, odourless sputum was expectorated. Straight x-ray revealed what looked like a large cyst in the right lower lobe, but bronchography demonstrated it was only one of several large bronchiectatic cavities. The photograph opposite shows the iodised oil lying at the bottom of these excavations. When lobectomy was performed, examination of the right lower lobe showed it to be honeycombed with bronchiectatic cavities.

SECTION 4.

THE CAUSATION OF BRONCHIECTASIS.

Bronchiectasis was recognised as a clinical entity by Andral[53] as long ago as 1824, and Laennec,[54] in his classical study published in 1826, gave an excellent description of the disease. He pointed out that it varied greatly in extent with regard both to dilatation and distribution, and although any part of the lungs could be affected the upper lobes generally escaped. He noted the cylindrical, saccular, and cavitatory forms of the condition, and described destructive changes in the elements of the bronchial walls often associated with fibrous thickening.

Laennec[54] attributed the bronchial dilatation to the progressive accumulation of infected secretions, and although many other explanations have since been put forward it will be seen that this postulate still has its advocates.

Factors which have been held responsible for the causation of bronchiectasis will be considered under the following headings:-

1. TRACTION FIBROSIS.
2. CHRONIC COUGH.
3. ALLERGY.
4. VASCULAR DISEASE.
5. DEVELOPMENTAL ABNORMALITIES.
6. INFECTION.
7. BRONCHIAL STENOSIS.
8. ATELECTASIS.

1. TRACTION FIBROSIS.

Pulmonary fibrosis, as has been seen in section 2, is frequently noted on pathological examination of lungs in which the disease is present to a marked extent. Corrigan,[55] in 1838, put forward the theory that the bronchi were dilated by the contracting fibrous strands, and this explanation was still supported in 1927 by Findlay and Graham.[56]

There are many shortcomings in this argument. Bronchograms illustrating cases of cylindrical bronchiectasis have already been shown, for example, those of Case 26 opposite page 67, and those of Case 8 opposite page 73. No fibrosis is obvious in the bronchograms nor was it in the straight x-rays, but let it be supposed for a moment that it was present, but had escaped demonstration.

If the theory of traction fibrosis is correct, what arrangement of the fibrous strands must be postulated, in order that the smooth cylindrical dilatation of the bronchi might be effected by their contraction?

It must surely be imagined that the fibrous strands radiate from each bronchus like the spokes of a wheel, and have their peripheral ends attached to various fixed points. It must further be surmised that the fibrous strands exert a long pull, and a strong pull, and a pull altogether to produce the uniform dilatation seen in the bronchograms. This appears a distinctly unlikely state of affairs, and as has been seen, pathological studies of bronchiectatic lobes fail to reveal any such ingenious arrangement; in fact, they often demonstrate very little fibrosis at all.

In cases where fibrous tissue is much in evidence, it seems a more reasonable expectation that fibrous strands

running in various directions in the pulmonary tissue, will, on contraction, compress and distort the bronchi, rather than dilate them, and this, in point of fact, is exactly what Lisa and Rosenblatt[34] discovered in their pathological researches.

Again, if the theory were correct, it might certainly be assumed that in diseases in which fibrosis is a feature, bronchiectasis would be a common finding. Silicosis is the disease par excellence associated with the production of pulmonary fibrosis. A high incidence of bronchiectasis in this malady would therefore confidently be expected. But what are the facts? - the very opposite. At the Johannesburg Conference in 1930, Irvine, Simson, and Strachan[57] declared with regard to silicosis that "bronchiectasis is definitely a rare complication."

In cases of tuberculosis, Erwin[58] ran iodised oil into the bronchi which were situated in the midst of extensive pulmonary fibrosis. Distortion, rather than dilatation, was observed. At the present time, therefore, traction fibrosis can scarcely be taken seriously as a vital factor in the causation of bronchiectasis.

2. CHRONIC COUGH.

Andral,[59] over a century ago, placed great stress on the importance of chronic cough as a causative factor in bronchiectasis; but it is of interest, particularly in this present work, to note that he also observed that the condition could occur in a very short time, as in the case of children who died 2 or 3 months after the onset of whooping cough.

Some writers have attributed bronchial dilatation to the forced expiration in the act of coughing, but, as

Andrus[35] points out, the force and strain of coughing are due to spasmodic contraction of the abdominal and thoracic muscles, and the effect produced is an expiratory elevation of the pressure in the air chambers, and compression of the bronchi. There is, therefore, no reason why the expiratory phase of coughing should produce bronchial dilatation.

Other observers have suggested that the long, full inspiration accompanied by partial laryngeal obstruction, as evidenced by the whoop in whooping cough, may cause bronchial dilatation. Here again, there is little evidence that this is the case. During inspiration, the thoracic cage enlarges, thus causing a fall in pressure in the alveoli and bronchi. This naturally causes a tendency towards dilatation in the alveoli and bronchi, and in normal respiration, the alveoli do dilate, and air enters the lungs. As has been mentioned, there is also slight inspiratory dilatation of the bronchi, in the upper and middle lobes at least, but this dilatation, even in the fullest inspiration, is very slight, and the fact that it has occurred at all is only apparent when the calibre of the bronchi on full inspiration is compared with the calibre of the bronchi on full expiration. A partial laryngeal obstruction, while causing an excessive lowering of intrathoracic pressure, will not cause either the bronchi or the alveoli to dilate to a greater extent than they would on full inspiration; their total capacity is determined by the capacity of the thoracic cage. There is thus no evidence that the inspiratory phase of coughing is likely to produce bronchial dilatation.

Andrus[35] states that observation over a period of 15 years of some 2,500 soldiers pensioned for chronic bronchitis, revealed no cases of bronchiectasis, except

following an intercurrent pneumonia. In chronic bronchitis, therefore, there is the factor of infection of the bronchial wall as well as chronic cough, but even then coughing does not produce bronchial dilatation.

In the one hundred and fifty cases of whooping cough which were investigated during the course of this work, bronchography failed to reveal an instance of bronchial dilatation unassociated with atelectasis. As thirty of the cases had broncho-pneumonia, these findings suggest that neither the long inspiration causing the whoop, nor the bouts of paroxysmal coughing, cause bronchial dilatation whether the bronchi are inflamed or not.

3. ALLERGY.

Watson and Kibler[60] believe that bronchiectasis is caused by allergic bronchitis. According to them, oedema of the mucosa, and outpouring of tenacious secretion are responsible for atelectasis, which is associated with bronchiectasis. With regard to allergy as a factor, it can be said quite definitely that although it may be an occasional cause of atelectasis, it is certainly not a usual one. The view that bronchiectasis is directly caused by atelectasis, is, of course, implicit in this theory, and will be discussed at length in due course.

4. VASCULAR DISEASE.

Ameuille and Lemoine[61] have claimed that vascular disease of the bronchial arteries, by interfering with bronchial nutrition, is a contributory factor in the pathogenesis of bronchiectasis, and Daly,[62] in support of this view, points out that the bronchial musculature and mucosa, and the intraplumonary nerves and ganglia, are all dependent on the bronchial arteries. These authors state

that obliterative changes in these vessels may therefore influence respiratory movements of the bronchi, and also produce degenerative changes in the bronchial walls. They claim that if the walls are sufficiently weakened, the dilating forces of respiration will be enough to cause permanent ectasia. It will later be suggested that there is little ground to suppose that this latter statement is true. In any case, as has been seen in the section on pathology, obliterative changes in the blood vessels were little in evidence. Where there was chronic infection in the parenchyma of the lung, vascular disease was sometimes noted, but this, of course, is a concomitant of any infective process.

It may be said that there is no real evidence that vascular disease is responsible for bronchiectasis.

5. DEVELOPMENTAL ABNORMALITIES.

Several authors have put forward the view that developmental abnormalities are responsible for the production of bronchiectasis.

Congenital cystic disease of the lung is a well recognised clinical entity. Sauerbruch[63] claimed that bronchial dilatation seen in childhood was really often congenital cystic disease of the lung without the cysts. He described a series of fifty cases in which evidence of infective processes was lacking, and where the disease was confined to one lobe, usually the lower lobe of the left lung. This preponderance of left sided unilobar disease he explained on embryological grounds. The ducts of Cuvier, which run in the septum transversum, elongate, and enter the sinus venosus at a much more acute angle in the fifth week of foetal life, because at that time the

thoracic cavity enlarges. Sauerbruch suggested that during this period, when the long buds are growing rapidly, in certain cases the left duct in particular presses on the embryonic lung, and interferes with the development of that portion which forms the lower lobe.

It may be noted, however, that the left lower lobe is also predominantly affected in cases where a congenital origin can be excluded, and therefore, ingenious though this theory may be, it is not completely convincing.

Miller[64] believes that developmental anomalies predispose to bronchiectasis, but that other factors are necessary in the production of the disease.

Reisner and Tchertkoff[65] suggest that the factors which indicate congenital origin are lack of anthracosis, regularity of the saccules, patency of the affected bronchi, and the absence of marked inflammatory changes. It has already been seen, however, in the section on pathology, that many specimens in which a congenital origin could be ruled out, satisfied these conditions with the exception of the lack of anthracosis, which was not specifically noted. Quite possibly some of them fulfilled this qualification also.

Although a congenital origin for bronchiectasis cannot be excluded in all cases, it seems clear that the vast majority are due to other factors. Specimens will be shown later where bronchiectasis has actually been seen from its earliest stages, and in which the lung had previously shown no abnormality.

6. INFECTION.

The theories so far discussed can scarcely be regarded as supplying a conception of the causation of

bronchiectasis capable of accounting for the different manifestations of the disease. The theory of an infectious origin does, however, purport to do this. Naturally, in a disease in which for long the expectoration of copious, foul smelling sputum was regarded as a sine qua non, the possibility that the malady was due to infective processes could not be overlooked.

Gairdner[66] regarded bronchiectasis as in most instances an ulcerative excavation of the lung in which the cavities communicated with the bronchi. He considered that in the saccular variety especially, the dilatations were not due to a true ectasia of the bronchial wall, and Virchow,[67] writing about the same time, commented on the similarity between bronchiectatic cavities, and infected pulmonary cavities.

If Gairdner's view is correct, it would be better of course, to call the disease broncho-pulmonary necrosis, rather than bronchiectasis which would be rather misleading. Gairdner's concept of the disease is not without its supporters even in recent times. Boyd,[68] for example, as late as 1934, holds similar views, which are also put forward by McNeill[32] and Opie,[30] and this explanation has at least the merit of being perfectly comprehensible. It can, nevertheless, be quite definitely demonstrated that in many instances this theory as to the causation of bronchiectasis is not true. A statement of fact is made, viz., that the apparent dilatations of the bronchi are really cavities formed by a necrosis of the bronchial wall and the surrounding lung parenchyma. All that has to be done, therefore, to disprove this assertion, is to discover cases of bronchiectasis in which pathological examination reveals a relatively intact bronchial wall, and as has

been seen in the section on pathology, this is a common finding. In many instances, not only were the layers of the bronchial wall found to be in this comparatively unimpaired state, but evidence of infective processes was not in any way considerable - certainly there was no suggestion of bronchial necrosis and excavation of the lung.

To rule out the hypothesis of broncho-pulmonary necrosis as one generally applicable to the genesis of bronchiectasis, it is unnecessary to go further, but there are other insuperable objections which may be put forward. How, for example, can this theory account for the cases of so called "dry" bronchiectasis, to which attention has already been called? The older writers such as Gairdner, it is well to remember, did not even know that they existed. If the bronchogram of a case already cited (Case 26, p.67) is considered, it will be seen that there is a smooth dilatation of the lower lobe bronchi. If the theory of broncho-pulmonary necrosis is correct, these apparent bronchial dilatations are in reality elongated infected pulmonary cavities. Yet the patient, during his stay in hospital, had only a slight elevation of temperature which returned to normal in three days, and a negligible production of sputum, which in less than a week, ceased altogether. Up to the present time, over a year later, he has remained perfectly well. In the face of such a history, the presence of cavitation to the extent shown in the bronchogram appears to be incredible. If the onset of the disease is taken to have occurred in the few days preceding his admission to hospital, surely cavitation of such considerable proportions could not have taken place without symptoms of much greater gravity.

If, on the other hand, the onset is postulated as having taken place at some former date, it must have been in early childhood, because the patient was unable to recall having had any serious respiratory trouble. The most he would admit to was having an occasional cold and cough in the winter time, like most other people. On this assumption, therefore, the patient, if the theory we are considering is correct, must have had extensive pulmonary cavitation for many years without suffering any serious disability. Although the advocates of broncho-pulmonary necrosis maintain that the disease is a progressive one, let it be considered for the sake of argument, that in the present case the patient had a severe pulmonary infection in early childhood, which he and his parents had forgotten, and that this infection gave rise to the "cavitation" which accounts for the bronchographic appearance. It must then be assumed that the infection was overcome, healing took place, and the cavities were re-lined with epithelium. But reparative processes are associated with fibrosis, of which there was no radiological evidence, and they are rarely uniform, while the bronchogram shows that the "cavities" were remarkably regular in appearance.

Cases such as the one cited have forced advocates of the infective theory to change their ground somewhat in recent years. Best and Taylor,[49] for example, state that the primary change in bronchiectasis is a weakening of the bronchial wall as a result of infective processes. The elastica gradually deteriorates, its resilience becomes progressively less, and after inspiration, the bronchial lumen does not regain its normal calibre, but remains dilated, and gradually expands. It must be observed that such statements are mere speculation, and in many instances

at least can be proved to be wrong, for it has already been noted that pathological examination of lobectomy specimens frequently shows an almost normal bronchial wall, and even in the advanced cases studied by Lisa and Rosenblatt[34] post mortem, the elastica was ordinarily found to be intact. The anatomical basis postulated by Best and Taylor[69] to account for the weakening of the bronchial wall has therefore often in practice been found lacking. Muir[70] admitted he was unable to explain the generalised weakening of the bronchial musculature which he thought must precede the cylindrical bronchiectasis sometimes seen following whooping cough and measles. Stokes,[71] as long ago as 1839, advanced the theory which has been revived from time to time since, that inflammation of the bronchial walls may produce paralysis of the muscular tissue, and the bronchi dilate in a manner comparable to that seen in paralytic ileus. While no real evidence has ever been adduced that such an event actually takes place, it is an interesting problem in physics whether the bronchi would dilate, if neuro-muscular paralysis, or other "weakening" of the wall short of necrosis, occurred.

Advocates of such views on the genesis of bronchiectasis obviously believe that should the myo-elastic tissue of the bronchial wall lose its tone, the elastic tension of the lung would cause the affected bronchi to dilate; pulmonary cavities, for example, tend to increase in size, and become spherical for this reason. It would not be safe, however, to assume that the elastic tension of the lung would cause progressive dilatation of the bronchi in a similar manner.

Ellis,[19] as has already been mentioned in a previous section dealing with the physiology of the bronchial tree, found in his careful study of the calibre changes of the

bronchi in respiration, that during the respiratory excursion they act as passive conducting tubes for the air, and that the tone of the myo-elastic tissue, far from having the function of preventing undue bronchial dilatation during inspiration, prevents the bronchial walls from collapsing in expiration.

If his conclusions are correct, therefore, mere loss of tone in the myo-elastic tissue of the bronchial walls would not lead to progressive dilatation of the bronchi, since the dilating effect of inspiration would be counterbalanced by the collapsing effects of expiration during each respiratory excursion.

It is possible to argue, however, that if changes took place in the elements of the bronchial wall as a result of inflammation, for example, a fibrous replacement of the myo-elastic tissue, the slight dilating inspiratory force would be sufficient to increase the bronchial calibre, while the less resilient character of the walls would prevent the usual diminution in expiration. A progressive ectasia might, therefore, ensue.

It seems safe to infer, on the other hand, that the dilating effect of inspiratory forces on the bronchi is very slight; it has been mentioned earlier in the chapter that disputes have arisen even among experts as to whether bronchograms demonstrate inspiratory increase in bronchial calibre or not, and although there is general agreement that the phenomenon can be observed in the upper and middle lobes, Heinbecker[12] and others claim that there is rather narrowing of the lumen in the vertically disposed bronchi of the lower lobes which are the ones most commonly involved in bronchiectasis.

Even if Heinbecker's findings were correct, of course, it would not necessarily refute the argument which is

being considered, for though inspiration might lengthen and narrow the normal elastic tube, it might tend to lengthen and dilate a non-elastic tube. Yet, when the dilating force of inspiration produces such small changes in the calibre of the normal highly elastic bronchi, it appears improbable that it would have much effect on a more rigid fibrous structure. When it is remembered, also, that the bronchi are surrounded by millions of thin-walled, easily distensible alveoli, it is difficult to imagine an inspiratory force being transmitted to the bronchial walls great enough to cause the marked and often rapid dilatation of the lumen seen in cases of bronchiectasis.

Even if the suggestion is accepted, therefore, that bronchial inflammation may cause a "weakening" of the wall, the balance of evidence appears to be definitely against the conclusion that the normal pulmonary tensions would give rise to ectasia.

The possibility cannot be wholly excluded, however, and it is interesting to inquire if the theory is supported by practical experience.

If, for example, inflammation of the bronchial wall causes neuromuscular paralysis or other weakening which in turn results in bronchial dilatation from the effects of the normal pulmonary tensions, it would certainly be expected that bronchiectasis would be an inevitable complication of chronic bronchitis. It has already been pointed out, however, that Andrus[35], after observing over a period of 15 years 2,500 patients suffering from this disease, stated that bronchiectasis was a rare complication, and was only seen following an intercurrent pneumonia. These findings are, therefore, strongly against the theory that inflammatory "weakening" of the bronchial wall leads to

bronchiectasis. The probable nature of the "pneumonia" will appear in later discussion.

Summing up the views of those who hold that infective processes are primarily responsible for bronchiectasis, it is seen that there are two main theories: the one is that so called bronchiectasis is really broncho-pulmonary necrosis, and the dilatations are, in fact, infected pulmonary cavities communicating with the bronchial tree; the other is that the condition is a true ectasia of the bronchi due to "weakening" of the walls by infection, and subsequent dilatation by the normal pulmonary tensions.

The most vigorous exponents in recent times of the theory that infective processes are sufficient by themselves to cause bronchiectasis, are Lisa and Rosenblatt,[34] writing in 1943. Unfortunately, although they make it quite plain that they believe infection alone is responsible for the condition, and that other factors, particularly atelectasis, have little bearing on the problem, it is difficult to gather from their publication how exactly they consider the bronchial dilatation arises. They quote a large number of authors who hold the same views as themselves, including those whose opinions have been considered above. The substance of the theories advanced by these writers has already been given, and objections put forward which are not answered in their publications.

Lisa and Rosenblatt[34] themselves do not appear to believe that mere weakening of the bronchial wall will by itself give rise to bronchial dilatation, for they state on page 115 of their essay - "If a bronchus is so weakened as to dilate in inspiration, it will contract in expiration." It seems, indeed, quite clear that they support the theory of broncho-pulmonary necrosis, for on page 103, they declare - "Bronchial dilatation is not

due to widening of the lumen with the elements of the wall intact, but is due to necrosis of the wall itself. The greater the destruction of the bronchial wall and parenchyma, the bigger the cavity." Yet, in the summary given of their actual findings, they stated that the pleura was generally adherent, the bronchi dilated, and the walls usually thickened by fibrous tissue replacement. The epithelium was intact but occasionally modified. The basement membrane was preserved, while the muscle layer showed hypertrophy and moderate fibrous tissue replacement. The elastic fibres were thickened, but relatively unimpaired, and there was plasma cell infiltration and moderate lymphoid infiltration. The blood vessels were generally normal, and there was fibrosis of the parenchyma.

Surely the theory which they advocate is quite incompatible with their own observations on the pathology of the disease? As they said themselves, the postulate which they put forward implies that there should be complete destruction of the bronchial wall and the surrounding parenchyma, so that a cavity is formed. According to their own statement, the dilatation can be explained in no other way, since they ruled out mere weakening of the bronchial wall as being sufficient, and on page 112 of their work, discarded fibrosis as a possible factor, for they stated - "The actual effect of fibrosis is to contract the bronchus, and not to widen it." How can they possibly explain, therefore, their own findings that the basement membrane of the bronchus is usually preserved, that the muscle layer shows hypertrophy with moderate fibrous tissue replacement, while the elastic fibres are generally unimpaired?

Discussing the pathological findings of Lander and Davidson,[17] which were, as has been seen, that evidence of

infection was negligible in the series of one hundred and forty bronchiectatic lobes which they examined, Lisa and Rosenblatt[34] stated - "The only way we can reconcile the pathological findings of Lander and Davidson with those of other observers, and with our own, is to assume that the lesions they studied were those of mild degree, so that there was little destruction of the bronchial wall and adjacent parenchyma."

But surely the findings cannot be reconciled at all. If there is little destruction of the bronchial wall, according to Lisa and Rosenblatt[34] there should be no dilatation, since mere weakening of the bronchial wall is not sufficient; it must be destroyed so that a cavity is formed. Yet it is difficult to imagine that one hundred and forty lobes were excised at the Brompton Hospital on the erroneous impression that bronchiectasis was present. And assuming that it was present, one can scarcely imagine that a pathologist, though he might have missed minor degrees of infection, could possibly have overlooked extensive bronchial necrosis and pulmonary cavitation.

One is forced to the conclusion that Lisa and Rosenblatt are rather illogical in the deductions they draw from the evidence.

A consideration of the theory of a purely infective origin of bronchiectasis would appear to lead to the following conclusions:-

1. The suggestion that the normal forces of respiration lead to the dilatation of a bronchus, the walls of which have been "weakened" by infection, has never been supported by any satisfactory evidence. Investigations bearing on this point all lead to the opposite conclusion.

2. It is quite possible, and even probable, that a severe infection of the bronchi may lead to necrosis of the bronchial wall and the surrounding lung parenchyma, thus giving rise to cavitation. In the vast majority of cases, however, it has been seen that the elements of the bronchial wall are present, though they may be damaged to a greater or lesser extent; it is only the most advanced stages of the disease which sometimes correspond to the picture of broncho-necrosis. Even Lisa and Rosenblatt,[34] the most strenuous advocates of the theory in modern times, examining lungs which had mostly been affected for many years, apparently failed on their own showing to demonstrate pathological conditions consistently consonant with their views on the causation of the disease.

7. BRONCHIAL STENOSIS.

Retention of secretions, due to occlusion or partial occlusion of a main bronchus, whether from intrinsic or extrinsic factors, has been regarded as an important cause of bronchiectasis from the time of Laennec[54] to the present day. Escudero and Adams,[71] in 1938, for example, after extensive experiments in which they produced bronchial stenosis in dogs, stated that in their opinion Laennec had been substantially correct when he attributed the disease to the damming back in the bronchi of infected secretions.

Bronchial stenosis as a factor in the production of bronchiectasis is therefore worthy of the closest consideration, but as it is also intimately concerned with atelectasis, it is more convenient to discuss it under the next heading along with that condition.

8. ATELECTASIS.

The introduction in 1922 by Forrestier and Sicard[73] of radio-opaque iodised oil as a means of outlining the bronchial tree gave a great impetus to research on bronchiectasis, and it soon became apparent that atelectasis was closely associated with the disease. Miller,[74] Singer and Graham,[75] Pinchin and Morlock,[45] Sells,[76] Richards,[77] Warner and Graham,[78] Anspach,[79] Warner,[80,81] Lander and Davidson,[13,15,16,17] Erwin[58] and many others have shown that where there is lobar atelectasis, there is almost invariably a gross dilatation of the contained bronchi. There is thus no doubt whatever that there is a close relationship between atelectasis and bronchiectasis. Whether atelectasis gives rise to bronchiectasis, however, is a point which is still much in dispute. It is proposed to discuss this problem in detail, but before doing so, it is necessary to study the causation of atelectasis itself, and a brief account of the leading work on the subject will there fore be given.

Causation of Atelectasis.

Atelectasis, as an entity arising in post natal life, was first described by Joerg,[82] in 1832, and the work of Gairdner[83,84] and Lichtheim[85] later confirmed his findings.

Gairdner[83,84] suggested that the condition might be caused by blockage of a main bronchus by a plug of sputum, and experiment tended to support his contention, for in 1879, Lichtheim[85] was able to produce atelectasis by blockage of a main bronchus in animal studies. Various workers have confirmed Lichtheim's observations, the most recent well known investigation being probably that of Tannenberg and Pinner[86] published in 1942. These authors, like Lichtheim, produced obstruction of a bronchus supplying a lung or lobe. They found that within an hour there was dimin-

ution in volume of the lung, or portion of lung, supplied by the bronchus which had been occluded. This was evidenced by the occurrence of compensatory phenomena - shift of the mediastinum to the affected side, rise of the diaphragm on that side, emphysema in the unaffected portion of the isolateral lung where the bronchus supplying one lobe only had been ligated, and emphysema in the contralateral lung. Post mortem examination revealed the typical pathology of atelectatic lung tissue.

Lichtheim[85] also studied the mechanism of the absorption of the alveolar air. He discovered that plugging of a bronchus did not result in pulmonary collapse, if, at the same time, ligation of the blood vessels to the obstructed area was carried out. He therefore concluded that the entrapped alveolar air was absorbed by the circulating blood, but he was unable to give a satisfactory explanation for this phenomenon. The invention of a micronometer by Krogh,[87] however, provided the means of solving this problem. Henderson and Henderson,[88] Haldane and Priestley,[89] and others, have shown that the tension of the gases in arterial blood is in approximate equilibrium with the gaseous tension in the alveoli, but that this is not the case with the tension of the gases in venous blood. During the flow of blood through the systemic circulation, there is a fall in the oxygen tension of 60 mm. of mercury, while there is an increase in carbon di-oxide tension of 6 mm. of mercury; consequently venous blood has a total gas tension of about 54 mm. of mercury lower than that of arterial blood or alveolar air. This explains the absorption of alveolar air by the blood, when the free communication between the atmosphere and the alveoli is interrupted.

After Lichtheim's original work,[85] it was plain that any factor causing obstruction of a main bronchus, whether by external pressure, as in the case of enlarged glands, aneurysm, etc., or by internal blockage of the lumen as by the presence of a tumour or foreign body, could give rise to atelectasis. Many instances in which these obvious factors operated were, of course, recorded, but in the majority of cases they were found to be absent. It was necessary therefore to postulate some much more ubiquitous agent, and Gairdner's[83,84] plug of sputum seemed to answer these requirements. There was, however, one grave objection; even in the days of routine bronchoscopy, the mucous plug proved most elusive, though occasionally it was possible to demonstrate it.

In 1936, Lander[16] provided a simple, satisfactory, and, once it had been pointed out, obvious solution. As the air in the alveoli and bronchi distal to the block is absorbed, the pressure in this system will fall, and thus the atmospheric pressure will tend to push the mucous plug down the bronchus. Lander visualised it splitting at the bronchial bifurcations till eventually the fragments lodged in the small peripheral bronchi. Hence the reason for bronchoscopy, in the majority of cases, failing to reveal the plug in the main bronchus.

Lander[17] in 1938, was able to produce experimental corroboration of his contention, and this work will be discussed later. His explanation satisfactorily accounts for the fact that in the majority of cases of atelectasis, investigation during life and after death reveals that the bronchi lying in the collapsed area are patent up to their terminal parts.

There are so many reasons for believing that this conception of the causation of atelectasis is correct,

that little doubt can now be entertained on the subject. For example, in the light of these findings, it would be expected that atelectasis would be common after pulmonary haemorrhage, where material comparable to sputum finds its way into the bronchial tree, and, in point of fact, Stivelman[90] in 1934 found that it was a usual, if fugitive phenomenon after haemoptysis in tuberculous patients.

It would also be expected that atelectasis would be common in diseases associated with the production of sputum, above all, in whooping cough. As Erwin[58] says, "the tenacious nature of the sputum bespeaks collapse." As has been seen in the preceding chapter on the results of the present investigation of whooping cough, atelectasis occurred in forty three per cent of the cases. Had they been x-rayed at daily instead of weekly intervals, the percentage might even have been higher. As it was, the duration was often brief, sometimes less than a week. It would be difficult to explain the short-lived nature of the collapse on any other hypothesis than a blockage of the bronchi by sputum.

In tuberculosis, too, it would be anticipated that absorption collapse would be a frequent complication. Erwin,[58] who dealt with this aspect of the subject, showed that such was the case, and collected one hundred examples.

An association between the pneumonias and atelectasis would likewise be expected, and the work of Coryllos[91,92] and others suggests that this may be so. It is unlikely that atelectasis takes place in a typical case of lobar pneumonia, because the alveoli become filled with exudate, and hence diminution of volume of the lung is not to be expected. In cases of broncho-pneumonia, however, in which the main stress of the infection fell on the bronchioles or finer bronchi, these might become occluded

at an early stage of the disease with consequent absorption of air from the alveoli. Again, at any stage of the disease in which sputum is being produced, atelectasis might be caused by aspiration in the manner described by Lander.[16,17]

The fact that occlusion of bronchioles or finer bronchi by localised infection seems likely on occasion to give rise to atelectasis, prompts the question if atelectasis is generally brought about by this mechanism, and not by aspiration of sputum. It seems clear that this is not the case. For example, atelectasis frequently occurs in thoracic operations in the course of which secretion is expressed from diseased parts of the lung. Holst, Semb and Frihmann-Dahl[93] found that atelectasis occurred in sound lung tissue unaffected by operative measures in fifty per cent of their thoracoplasties. This high figure can be accounted for by the fact that their cases were x-rayed early and frequently, and thus instances in which the collapse cleared up without trouble were noted.

Again, cases in which complete collapse of a lung suddenly takes place can scarcely be accounted for on the theory that the lumina of the peripheral bronchioles have been occluded by a local production of sputum, or by swelling of the mucosa; it would be strange if these individual blockages all took place more or less simultaneously. An example of simultaneous collapse of the right lung and left upper lobe in an eleven months old baby has already been given. (Case 15, p.16). There was no evidence of the occurrence of an acute infective complication which might have lent colour to the supposition that the bronchioles had all been locally occluded at the one time - indeed, the child could scarcely have survived such an

event. There was also at first absence of breath sounds which was followed the next day by weak bronchial breathing, though the collapse persisted. This was, of course, very strongly suggestive of an aspirated plug of mucus blocking first the main bronchus, and then being drawn down into its peripheral branches. Bronchography was not carried out in this case in view of the age of the patient and the gravity of his condition, but the absence of enlarged root glands in the straight x-ray, the speedy appearance of weak bronchial breath sounds, and the fact that the condition cleared up in a month, appear to preclude the alternative postulate that the collapse was due to extrinsic pressure on the bronchus.

Atelectasis in whooping cough at least, does not appear to be locally produced for if it were some evidence of a preceding infection in the affected area would be expected. In the majority of cases, however, there was no elevation of temperature before the collapse took place, or even at the time of its occurrence, nor were there any signs on physical examination of preceding local activity. Moreover, as has been observed in the last chapter, it occurred less frequently in cases where broncho-pneumonia was a complication than in cases where it was absent, and in the cases in which it did occur in the former group, the atelectasis did not necessarily take place in the lobe affected by the broncho-pneumonia. It is therefore probable that the majority of cases of atelectasis occurring in whooping cough are best accounted for on the basis of aspiration of sputum. It has already been noted that atelectasis occurs frequently after pulmonary haemorrhage, and here aspiration seems certainly the mechanism.

In general, it appears clear that this is the usual

mechanism in the production of atelectasis, though in infections such as pneumonia, it may be brought about by the obstruction of the peripheral bronchi as a result of the local production of exudate. When the effect of atelectasis on the bronchial calibre is discussed, other evidence supporting the aspiration theory will be adduced.

Theories that atelectasis may be produced by mechanisms other than that of bronchial or bronchiolar occlusion have been put forward from time to time, but none have withstood close investigation.

Pasteur[94,95] suggested that diphtheritic massive collapse was due to paralysis of the diaphragm. Elevation of the diaphragm is one of the compensatory phenomena observed when atelectasis takes place, as has been noted above, and Pasteur mistook this for paralysis. His theory was disproved in 1927 by the work of Jackson[25] and his colleagues who showed that here again the atelectasis was due to bronchial obstruction, caused in this instance by diphtheritic membrane, the removal of which led to re-expansion of the lung. Erwin[58] pointed out that phrenic crush with paralysis of the diaphragm does not per se, cause atelectasis, though by inhibiting the elimination of sputum from the bronchi, it may in some instances be a contributory factor. Diaphragmatic paralysis is, of course, of little importance in a general way, as the vast majority of cases of collapse occur in patients in whom it can be excluded.

Bradford[96] in 1918-19, called attention to the frequency of atelectasis following thoracic trauma even when inflicted on the contralateral side of the chest. By itself, trauma can scarcely be regarded as a usual cause of atelectasis, as it can be excluded in so many cases in which the condition is present, as for example in the

whooping cough series investigated in this work. But in thoracic operations, or in cases of fractured rib it no doubt plays an important secondary role, for pain, by inhibiting coughing, would tend to cause an accumulation of secretion.

Because of suggestions that upset of the vascular supply to the lung may lead to atelectasis, Tannenberg and Pinner,[86] in 1942, studied the effects of ligation of the pulmonary artery in rabbits.

They found that the lung, the blood supply of which had been interrupted, became shrunken, and thus bore a superficial resemblance to a lung the bronchus of which had been ligated. But the two conditions were essentially entirely different. The lung, the blood supply of which had been interrupted in this manner, showed evidence on pathological examination of haemorrhages into its substance. There was no true atelectasis; the lung eventually became shrunken owing to supervening fibrosis.

All the evidence therefore points to the fact that atelectasis can only be produced by bronchial or bronchiolar obstruction.

Having discussed the factors responsible for the causation of atelectasis, the relationship between atelectasis and bronchiectasis may now be considered.

Experimental Evidence of the Relationship Between Atelectasis and Bronchiectasis.

Much of the confusion on the relationship between atelectasis and bronchiectasis arises from conflicting interpretations of experimental evidence, and it has therefore been considered best to study the leading experimental work on the subject at the outset.

The publications in recent times which have caused most interest and controversy are those of Adams and Escudero,[72] Tannenberg and Pinner,[84] and Lander and Davidson.[17] From a study of the literature on bronchiectasis, it appears that many writers have inaccurately quoted the findings in these experiments, or recorded the conclusions of the observers without mentioning the work upon which their deductions were based, thus quite unnecessarily complicating the issue. The experiments performed, and the conclusions drawn from them, will therefore be set out in some detail and discussed.

Experiments of Adams and Escudero.[72]

Dogs were used in this series of experiments, and the animals were divided into three groups.

In the first group, eight dogs were used. Massive collapse of the left lung was produced by completely obstructing the main bronchus. Incomplete obstruction of the right lower lobe bronchus was then produced, and a saline suspension of organisms obtained from a human lung abscess introduced beyond the partially occluded part of the bronchial lumen. The bronchial obstruction whether partial or complete, was effected by painting the lumen with 35% silver nitrate, though in some cases thermal cautery was used.

The left lung was collapsed so that the effect of a lowered intra-thoracic pressure on the partially obstructed and infected bronchi of the right lower lobe could be observed.

As the right lower lobe bronchus was painted at its first branching, there was sometimes partial obstruction of one secondary bronchus, while the other was completely patent. The animals were sacrificed 11 days to 3 weeks after inoculation with the saline suspension of organisms.

Post mortem examination revealed that the left lung of the dogs in this group was completely collapsed, and there was no bronchiectasis.

Apart from the right lower lobe, the right lung was normal. The right lower lobe was usually diminished in size, and bronchiectasis was seen to a greater or lesser extent in every case. If a secondary bronchus had escaped stenosis, there was no bronchiectasis in the area supplied, and no evidence of damage to the lung tissue or bronchial wall. Wherever there was bronchiectasis, there was stenosis of the bronchus supplying the area.

The bronchiectatic dilatations had either no epithelial lining at all, or were lined by flattened cells, and there was round cell infiltration and some fibrosis. The bronchiectasis was saccular in type except in one case where it was cylindrical. Anaerobic cultures grew C. welchii.

In the second group, the same procedure was carried out with seven dogs, except that atelectasis was not produced in the left lung. The results were the same as in the first group, except that the bronchiectasis was less marked. Again the right lower lobe, while diminished in size, was not completely atelectatic.

In the third group, eleven dogs were used.

In five, complete collapse of the left lower lobe was induced. The right lower lobe was partially obstructed as before, but this time the organisms introduced beyond the partial obstruction were suspended in iodised oil, the idea being that the more viscid medium would cause longer retention of the organisms in the lung. Post mortem examination revealed massive atelectasis of the right lower lobe, but no bronchiectasis or evidence of infection.

In the remaining six dogs, the same procedure was carried out except that this time the left lower lobe was not collapsed. In this series, the right lower lobe in one case presented the same signs as before, in another it was normal, and in two there was pneumonia without bronchiectasis. In the fifth case, the dog died of a left pyothorax after operative interference, the right lobe remaining normal, and in the last case there was bronchiectatic dilatations containing pus distal to incomplete stenosis of a secondary bronchus.

Adams and Escudero[72] explained that when the experiment was undertaken, they had reason to believe from the work of Hedblom,[97] that a lowered intrapleural pressure would play an important part in the development of bronchiectasis. Therefore, in the first group, the left lung was rendered atelectatic. This caused the intrapleural pressure to become doubly negative on the left side, and 50 per cent more negative on the right side - the side on which the right lower lobe bronchi were partially stenosed and infected. Thus, whereas in most normal dogs, the intrapleural pressure ranged between -6 and -8 cms. of water, when atelectasis of the entire left lung was produced, the pressure in the left chest fell to -14,-18 cms., and in

the right to -10,-12 cms. of water.

In the second group the left lung was not collapsed, and the intrapleural pressure therefore not lowered, so that this series acted as a control for the first group.

As bronchiectasis was produced in the second group where partial bronchial stenosis and infection were alone acting, the authors concluded that these factors were the essential ones in the genesis of bronchiectasis. In the first group, however, where the additional element of lowered intrapleural pressure was present, the bronchiectasis was somewhat more severe. This finding led Adams and Escudero to believe that although it was not a fundamental factor in the production of the disease, it was likely to aggravate it once it had been established.

The bronchus leading to the left lung was, as has been mentioned, completely occluded in the first group of dogs. Bronchiectasis was never detected in the lung at post mortem examination. Hence it was deduced that complete bronchial obstruction cannot lead to bronchiectasis.

In the third group, where the organisms were suspended in iodised oil, the authors realised that they had made an unfortunate choice of medium, as it apparently exerted an antiseptic effect. In this way they accounted for the absence of bronchiectasis. They did not comment on the fact that in six cases in this group, there was massive atelectasis of the lobe into which the viscous medium was injected, although at post mortem no bronchiectasis was detected.

This is, however, of great interest, because there is a vital difference from the atelectatic lung in the first group, in which also no bronchiectasis was seen. There the main bronchus was completely occluded, and the distal

portion shut off from the atmosphere. Here the obstruction was only partial, and the bronchial tubes in the atelectatic system were thus in communication with the outer air. The atelectasis was therefore caused by obstruction in the peripheral, finer bronchi.

Adams and Escudero summed up their findings by declaring that in their opinion bronchiectasis is caused by infection and incomplete bronchial obstruction, the reason probably being the one originally put forward by Laennec,[54] that dammed up stagnant secretions give rise to the dilatation. They added that its development does not appear to depend on the presence of any particular organism, as they had been able to isolate from the affected areas in different cases aerobic and anaerobic pyogenic organisms, the tubercle bacillus, and distemper organisms.

Experiments of Tannenberg and Pinner.[86]

In 1942, Tannenberg and Pinner[86] published the results of the following series of eight experiments performed on rabbits:-

1. Complete intrinsic obstruction of the bronchus of a lung or lobe.

This was done by introducing rubber tubes, filled with barium paste and surrounded by rubber sheets in umbrella or parachute formation, into the proximal part of the bronchus of supply. In two to four hours, complete atelectasis of the lung or portion of lung supplied by the obstructed bronchus had taken place. The mediastinum moved to the atelectatic side, the diaphragm was elevated on the same side, the isolateral intrapleural pressure was greatly lowered, and the contralateral pressure lowered to a lesser extent.

If only one lobe was rendered atelectatic, there was over inflation and eventually emphysema of the remaining isolateral lobes, and similar though less marked changes in the contralateral lung. If a complete lung had been rendered atelectatic, then these changes were seen in the other lung.

At post mortem there was no bronchiectasis in the atelectatic lung or lobe. On the contrary, the smaller bronchi were completely collapsed, and the walls of the larger bronchi only kept apart because of the resistant cartilage.

In some cases, the obstruction was removed after 10 days, and resulted in the complete return of the lung to normal.

2. The same procedure as in group 1 was carried out, but here an artificial pneumothorax was induced on the same side as the atelectasis. This eliminated the diaphragmatic elevation, mediastinal shift, and emphysema, but the post mortem findings were the same.

3. The main bronchus of a lung was ligated just below the tracheal bifurcation. The same results were observed as in group 1.

4. The same procedure as in group 3 was carried out, but an artificial pneumothorax was induced on the same side. The same findings as in group 2 were noted.

5. The pulmonary artery of one lung was ligated. The post mortem findings were a fibrotic organisation of the lung with haemorrhagic necrosis. There was no real resemblance to atelectasis, although the roentgenological appearances were the same.

6. The pulmonary artery was again ligated, but infection of the lung was superadded. Here, in addition to the

appearances noted in group 5, there were also pulmonary abscesses.

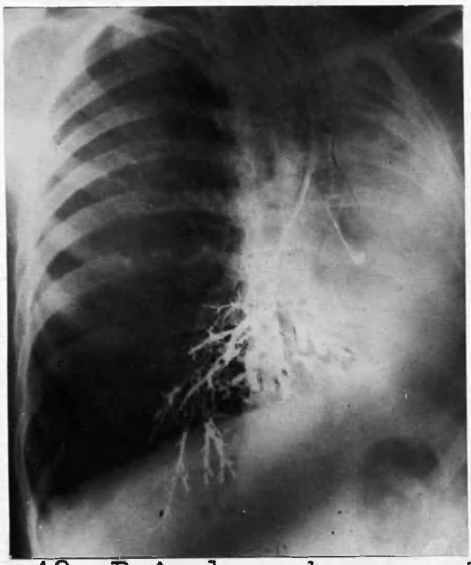
7. Infected material was deposited in the main bronchus of a lung. The bronchus was then ligated just below the tracheal bifurcation. Bronchiectasis, saccular or cylindrical, developed, whether or not an artificial pneumothorax was induced.

8. The main bronchus of a lung was partially obstructed. Patchy atelectasis combined with over inflation and emphysema of the lung was noted, but bronchiectasis was not produced.

As a result of their experiments, Tannenberg and Pinner[86] concluded that bronchiectasis arises when infection occurs in a bronchus distal to a complete or almost complete occlusion. In the absence of infection, complete obstruction results in absorption of air from the alveoli and bronchi, and the loss of space is compensated for by shift of the mediastinum, elevation of the diaphragm, emphysema of the unaffected parts of the lung, if any, and emphysema of the contralateral lung. The intrapleural pressure of the atelectatic side is lowered, but this does not cause the bronchi to dilate. On the contrary, they collapse as far as the nature of their walls will permit. When infection is present, bronchiectasis is produced, and lowering of the intrapleural pressure has no effect on its development, as was shown by the fact that it arose to just as marked an extent when an artificial pneumothorax was present on the same side.

Like Adams and Escudero's[72], their conception of the origin of bronchiectasis is therefore essentially the same as Laennec's[54] - it is caused by the damming back of infected bronchial secretions.

An interesting feature was the extent of the forces



Case 40. P.A. bronchogram. The radio-opaque medium has not entered the atelectatic left lung although the patient was tilted to the left side; this indicated obstruction of the left main bronchus. The right lower lobe bronchi have been visualised and show that the rt. lower lobe has been partly drawn into the left hemithorax. Note the extreme tracheal deviation to the left.

set in motion when the atelectasis took place. The authors demonstrated that often the lower lobe on the contralateral side was actually partly drawn into the affected hemithorax, and a groove caused by the pressure of the spine upon it, could be observed post mortem. Incidentally, this latter finding showed that atelectasis could also be produced by direct pressure. They considered that the inter-alveolar openings (Kohn's pores), which made possible Van Allen's collateral respiration, are responsible for the disappearance of air from the pulmonary tissue which is exposed to such localised pressure.

The following case illustrates, in the human subject, the drawing of the contralateral lobe into the affected hemithorax.

Case 40.

Female, aged 22 years, admitted to Ruchill Sanatorium on 2/11/47 for investigation. Clinical findings pointed to complete atelectasis of the left lung, and this was confirmed radiologically. A bronchogram of the right lung taken on 10/11/47 demonstrated that the right lower lobe had been partially drawn into the left hemithorax, and the right lower lobe bronchi were dilated. A photograph of the bronchogram is shown opposite.

Experiments of Lander and Davidson.[17]

In 1938, Lander and Davidson[17] performed brilliant and illuminating experiments which appear to go a long way towards solving the riddle of bronchiectasis.

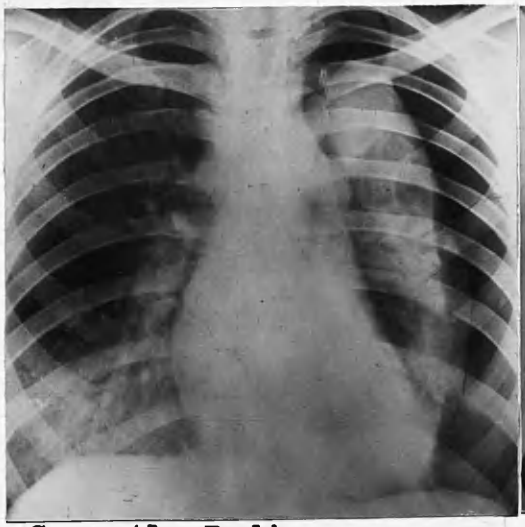
As has been mentioned, Lander had suggested two years previously[16] that atelectasis is caused by aspiration of a quantity of mucus, which, lodging at first in the proximal part of a main bronchus, is drawn towards the periphery, and splits at the bronchial bifurcations, with the final result that the peripheral bronchi become plugged with fragments of the material. The bronchi running in the atelec-

tatic area are therefore patent up to their terminal parts, and in free communication with the atmospheric air.

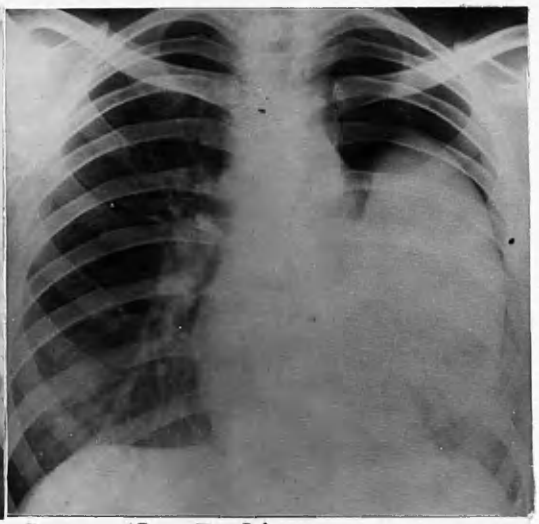
In the experiment under consideration, Lander and Davidson[17] prepared artificial tenacious sputum in the shape of a viscid solution of gum acacia. Using cats as their subjects, they introduced a quantity of this material into the main bronchus of a lobe, so that it produced occlusion of the lumen. It was "sucked" to the periphery as Lander[16] had suggested. Radiology showed that the lobe became atelectatic with all the usual evidence of compensatory changes. They then carried out bronchography. The bronchi of the unaffected lobes of the lung were seen to be normal, but marked dilatation of the bronchi in the atelectatic lobe was demonstrated.

Pursuing this line of investigation, bronchography was carried out in a human subject in whom atelectasis of a lower lobe had recently occurred. Patent dilated bronchi were demonstrated in the collapsed area. The intrapleural pressure was markedly lowered. The readings given are actually -30,-24 mm. of water, but this seems an obvious misprint for -30,-24 cms. of water. A pneumothorax was then induced, 325 c.c. of air being given, after which the readings were -10,0. Subsequent readings of -16,-4 cms. of water were followed by the introduction of 500 c.c. of air, when readings of -4,+4 cms. of water were recorded. Bronchography was then carried out, and revealed that the bronchial dilatation had almost disappeared. The subsequent absorption of air from the pleural cavity caused a reversion to the original condition of marked bronchial dilatation.

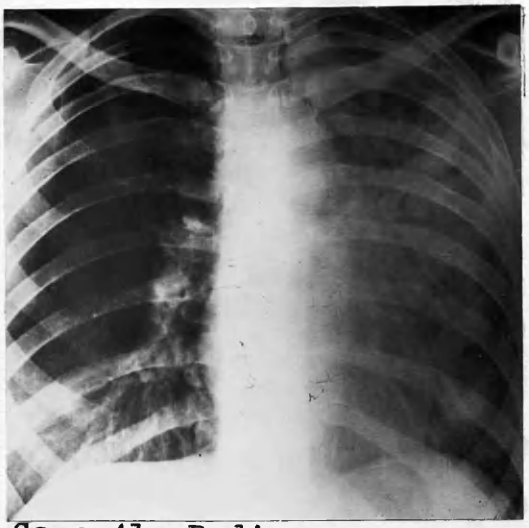
The same phenomena were demonstrated in a case of atelectasis following haemoptysis.



Case 41. Radiogram, 6/6/47. Tuberculous disease in left lung which is partially relaxed by a left A.P.



Case 41. Radiogram, 7/8/47. Although the left A.P. is no greater in extent than before, the partially relaxed lung is much denser in appearance, indicating that atelectasis has occurred.



Case 41. Radiogram, 25/9/47. The L.A.P. has absorbed. It is now clearly seen that the left lung is atelectatic. Note the shift of the heart to the left after absorption of the A.P.

In a case of atelectasis complicating pulmonary tuberculosis, bronchography showed bronchial dilatation in the affected area. Thoracoplasty was performed in this case, and after the operation it was noted that the bronchial dilatation had almost disappeared, although atelectasis remained.

During artificial pneumothorax treatment for pulmonary tuberculosis, the phenomenon of "black lobe" is sometimes seen - that is to say, in the shadow of the relaxed lung a denser shadow can be observed. Lander[17] showed in his article that this is an area of atelectasis, for as well as having the suggestive radiological features, he demonstrated in a case that came under his care that the intrapleural pressure was -22,-8 cms. of water, much lower than normal instead of higher than normal, as would be expected in the presence of an artificial pneumothorax. Bronchography revealed dilated bronchi in the "black lobe" area. The introduction of 200 c.c.s of air into the pleural cavity increased the intrapleural pressure to -6, -2 cms. of water and restored the calibre of the bronchi to normal.

Case 41.

The following case is an example of "black lobe" occurring in pulmonary tuberculosis.

Female, aged 21 years, admitted to Ruchill Sanatorium on 16/5/47 suffering from tuberculosis of the upper part of the left lung.

Left artificial pneumothorax was induced on 29/5/47, and a radiogram on 6/6/47 indicated that collapse was satisfactory. The photograph opposite shows that the lung was airbearing at that date. The reproduction opposite of a radiogram taken on 7/8/47 demonstrates that intrinsic or absorption collapse has occurred, producing the phenomenon known as "black lobe" or rather "black lung" in this instance. What the term means is that the opacity produced on the radiogram by the lung relaxed by the A.P. has become denser owing to the absorption of air from its alveoli.

The patient was too ill for bronchography, and the air in the pleural cavity was allowed to absorb. This process was complete when the last radiogram, reproduced opposite page 112, was taken(25/9/47); it is clearly seen that the left lung is atelectatic.

Pleural pressures in this case were taken with a machine which gave a reading roughly double that of the water manometer. At induction, and before refills, the readings averaged -15, -10, and after 400 c.c. of air had been given, -7,-4, cms. of water. On 4/8/47, the day when a refill was due, it was noted on screening that the lung had not "come up," and no more air was required. X-ray on 7/8/47 demonstrated the "black lung." On 13/8/47, the pleural readings were -28,-15 cms. of water, and after 600 c.c. of air had been given, -10,-5. Thereafter the A.P. was abandoned. The marked reduction in the intrapleural pressure after the occurrence of the "black lung" is therefore in keeping with Lander's findings.

Lander and Davidson[17] pointed out that when atelectasis takes place, there is a marked lowering of the isolateral intrapleural pressure, and quoted Habliston[98] as recording in cases of atelectasis of a lung, intrapleural pressures as low as -337,-432 mms. of water. Lander and Davidson claimed that the low intrapleural pressure produced shift of the mediastinum to the atelectatic side, isolateral elevation of the diaphragm, emphysema of the remaining non-atelectatic lobes in the isolateral lung, and, to a lesser extent, emphysema of the contralateral lung.

Since the obstruction is caused by the plugs of mucus in the peripheral bronchioles, the bronchi running through the atelectatic tissue are patent up to their extremities; like the alveoli, their walls are elastic, and as they are subjected to the same forces, they will therefore also expand. As they had demonstrated, when the lowered intrapleural pressure was restored to normal by inducing an artificial pneumothorax, all the compensatory changes, including the bronchial dilatation, disappeared, only to return when the artificial pneumothorax was allowed to

absorb.

Lander and Davidson[17] also gave an explanation as to why bronchiectasis was sometimes saccular, and sometimes cylindrical. If the plugs of sputum were drawn down as far as the terminal bronchioles, the resultant dilatation would be saccular, as the comparatively feeble walls of the bronchioles proximal to the obstruction would be exposed to, and easily distended by, the forces acting. If, on the other hand, the plugs lodged in the terminations of the finer bronchi, the bronchioles, being distal to the obstruction, would collapse, and cylindrical dilatation of the stronger bronchi proximal to the obstruction would be seen. They concluded, "It would appear to us that bronchiectasis ... is seen to be a reversible process, so to speak, and this even after years of collapse."

Discussion of the experimental evidence.

Lisa and Rosenblatt[34], commenting on the experimental production of atelectasis, suggested that as the experiments led to totally different conclusions on the part played by atelectasis in the causation of bronchiectasis, it could not be of great significance. Careful analysis of the experimental studies, however, shows at once that it is not the evidence which is conflicting; it is merely the inferences drawn from it which are at variance.

The relationship of atelectasis to bronchial dilatation may well be discussed first. The term "bronchial dilatation," rather than "bronchiectasis", is used advisedly, since at the present time bronchiectasis means different things to different writers.

It may be noted that the authors of the experiments are all agreed on one point - when atelectasis of a lobe occurs certain phenomena are constantly observed. The

diaphragm moves up on that side, the mediastinum shifts to the affected side, emphysema is seen in the unobstructed isolateral lobes, and, to a lesser extent, in the contralateral lung. There is also a marked fall in intrapleural pressure on the affected side, and as Adams and Escudero[72] pointed out, also on the contralateral side, though in a lesser degree.

The reason for these changes is in general obvious enough, though there is room for dispute on the exact mechanism. When atelectasis occurs, part of the contents of the thorax vanishes, as it were, from the area affected. Equilibrium could be restored in two ways; the walls of the thorax could fall in, or the contents of the thorax which remain could expand. Both these mechanisms, as has been seen, come into play as far as the physical nature of the materials will permit. The chest wall is too rigid to collapse, but the more mobile diaphragm moves up. The contents of the affected hemi-thorax expand, as is shown by the marked emphysema of the remaining unaffected isolateral lobes. If the mediastinum is mobile, as it was in the experiments reviewed, it moves to the affected side, and the contralateral lung also expands. The increased tension existing in the new state of equilibrium, is reflected in the lowering of the intrapleural pressure.

Now, if the obstruction causing atelectasis is in the periphery of the bronchial system, that is, in the bronchioles or terminal parts of the finer bronchi, the bronchi proximal to the obstruction are patent, and in communication with the atmospheric air. Their walls are elastic, and hence they are capable of expansion. There is therefore every reason to suppose that just as the other elements in the thoracic cavity capable of ready expansion

do expand, so also will the patent elastic bronchi in the atelectatic lobe; in fact, it will be suggested later that the forces at work bear more directly on the bronchi in question than on the alveoli of the remaining normal lobes.

Lander and Davidson's experiments on cats[17] show that these theoretical expectations are, in fact, realised in practice.

The emphysema of the remaining normal lung tissue, and the dilatation of the bronchi in the atelectatic lobe, are thus seen to be part of one process, that of compensation for the "lost space."

Lander and Davidson[17] suggest that the changes referred to are due to a lowering of the intrapleural pressure. It is believed that this conception is extremely misleading, but the matter will be more conveniently discussed in the next chapter. It is sufficient to state here that the lowering of the intrapleural pressure is considered to be merely an indication of new tensions arising in the thorax as a result of the atelectasis.

There are two ways in which these compensatory mechanisms of alveolar and bronchial expansion could be made unnecessary; either the outer wall of the affected hemithorax could be collapsed, or the lost space could be restored, for example by introducing air into the pleural cavity. In both cases, the original proportion of the size of the thoracic cavity to its contents could be attained.

The first method was illustrated by Lander and Davidson's thoracoplasty case[17] in which the operation caused the disappearance of the compensatory phenomena including the bronchial dilatation. The induction of an

artificial pneumothorax, the second method, produced the same results. Absorption of the air from the pneumothorax, by once more upsetting the relationship between the size of the thoracic cavity to its contents, caused the re-appearance of the compensatory phenomena, including the bronchial dilatation.

It is now easily seen that when Tannenberg and Pinner[86] declare that atelectasis does not cause bronchial dilatation, they claim much more than the evidence of their experiment will support. It showed merely that where there is obstruction of a main bronchus, atelectasis will not produce bronchial dilatation, but bronchial collapse. This, of course, is just what would be expected, and does not in the least invalidate Lander's[17] observations. The bronchi in the atelectatic lobe are not this time in communication with the atmosphere; hence the air they contain, will, like the alveolar air, be absorbed, and their walls will collapse as far as their structure allows.

The findings in the third group of the experiment of Adams and Escudero,[72] must next be reconciled with the other evidence. It will be remembered that here a viscid solution was introduced into the partially occluded bronchus of a lobe. In six cases, massive collapse of the lobe resulted. Lander's explanation[15] that the viscid material completed the obstruction, and was then sucked into the peripheral bronchioles, seems by far the most probable deduction as to what took place. But if this is true, then, since the bronchial occlusion was not complete, we are dealing with an atelectatic lobe in which there are bronchi patent up to their extremities, and in communication with the atmosphere. Theoretically, therefore, the bronchi should have been dilated, but Adams and Escudero[72] found that this was not the case. The explanation is

fairly obvious. Had the authors performed a bronchography while the animals were yet alive, they would doubtless have demonstrated the bronchial dilatation. They conducted the examination, however, post mortem, when it was found that there was little evidence of infection, and that the bronchial walls were intact. Naturally, therefore, once the thorax was opened, air entered the pleural cavity, and the bronchi resumed their normal calibre just as they did when Lander and Davidson[17] induced an artificial pneumothorax.

The experiments thus provide excellent evidence that there will be dilatation of the bronchi in an atelectatic lobe, provided that the obstruction is in the periphery of the bronchial tree. None of the findings on close examination really contradict this view. A reversal of the physical conditions created by atelectasis has also clearly been shown to cause the disappearance of the bronchial dilatation.

Lander and Davidson[17] concluded that bronchiectasis, even after several years duration, is a reversible process, but although the experimental evidence which they produced is extremely suggestive, it is nevertheless surely insufficient grounds on which to base such a hypothesis. A consideration of this aspect of the problem will be deferred until later.

Turning now to the experiments in which Adams and Escudero,[72] and Tannenberg and Pinner,[86] introduced infected material into a main bronchus which was then completely, or almost completely proximally obstructed, there seems no reason to doubt the excellent evidence they have produced that bronchiectasis can be caused in this manner. Their conclusions that the distension is due to a damming back of infected bronchial secretions seems also unexceptional.

Tannenberg and Pinner[86], however, claimed that the dilating forces of atelectasis had no effect in producing bronchiectasis in the case of complete obstruction of a main bronchus with infection, on the grounds that the induction of an artificial pneumothorax on the same side as the atelectasis did not prevent the distension of the bronchi. There is no reason, of course, why it should. Ligation of a main bronchus without infection produces collapse of the bronchi distal to the ligation. When infection has been introduced, the degree of distension will depend solely on the quantity of secretion in the bronchi, and therefore the bronchial calibre may be less, the same, or greater than normal, depending on the amount of pus. If the bronchial walls are destroyed, then broncho-pulmonary necrosis rather than bronchiectasis would result, though the term bronchiectasis has been applied to both conditions.

Infection in the presence of a partial obstruction could obviously lead to complete obstruction, thus giving rise to the same results. If the obstruction remains incomplete, then the dilating effects set up by an atelectasis caused by bronchiolar occlusion would once more act on the bronchi, and the distension due to accumulated secretions would be aggravated. This was noted by Adams and Escudero.[71]

If the infection were not severe, it is likely that a patchy atelectasis due to areas of peripheral bronchial obstruction would result. This was observed by Tannenberg and Pinner[86] in some cases of incomplete proximal obstruction, and they noted that emphysema was an ancillary radiological feature. Bronchial dilatation was not observed, but here again the investigation was made post mortem.

It is probable that bronchial dilatation had, in fact, been present during life, but since the bronchi were relatively unscathed, the opening of the thorax would permit them to assume their normal calibre.

Clinical Evidence of the Relationship Between Atelectasis and Bronchiectasis.

The matter of first class importance which emerges from a consideration of the experimental evidence reviewed, is that in practice two types of bronchiectasis are likely to be encountered; bronchiectasis caused by proximal obstruction of a main bronchus, and bronchiectasis caused by obstruction of bronchioles or finer bronchi.

If clinical material could be produced satisfying the expectations aroused by theoretical speculation and experimental evidence, it is obvious that a considerable step would be taken towards solving the problem of the causation of bronchiectasis.

The two types of bronchial obstruction will now be considered, and cases shown which illustrate the effects produced.

1. Proximal obstruction of a main bronchus.

Clinically, this can arise from intrinsic or extrinsic causes.

Intrinsic factors are generally neoplasm, foreign body, or a mucous plug, though in the last case the obstructing agent is generally either coughed up or drawn to the periphery of the bronchial tree. Diphtheritic membrane is another occasional cause, and, as has been mentioned, Pasteur[94,95] mistook the diaphragmatic elevation consequent on atelectasis for diaphragmatic paralysis due to diphtheritic toxin.

Extrinsic factors include pressure from enlarged glands, tumour, and aneurysm.

Complete obstruction of a main bronchus without infection of the associated area of collapse, as Tannenberg and Pinner[86] admit, must be somewhat rare in actual practice, and is only likely to exist when the obstruction is of comparatively brief duration. Inflammation occurring at the site of the obstruction is obviously likely to extend down the bronchus to the atelectatic system. It has also been shown that micro-organisms are normally present in the bronchial tree, but Livingston and Adams[99] state that it is by no means proven that mere bronchial occlusion will transform them into pathogens.

At all events, it is well known that the uncomplicated condition can arise from aspiration of a foreign body, for prompt removal, after location by the bronchoscope, leads to speedy re-expansion of the lung, and complete return to normal.

No doubt in these cases, as Tannenberg and Pinner[86] discovered, there is collapse of the bronchi beyond the obstruction as well as collapse of the alveoli, but it is, of course, impossible to demonstrate it clinically.

Membrane or thick secretion, like foreign bodies, can produce proximal bronchial occlusion without infection of the associated atelectatic system, and Jackson[45] and many others have shown that removal or expulsion of the peccant material results in complete recovery.

The following interesting case illustrates this point, and also supports Lander's[16] theory that viscid secretion may be drawn to the periphery of the bronchial tree.

Case 42.

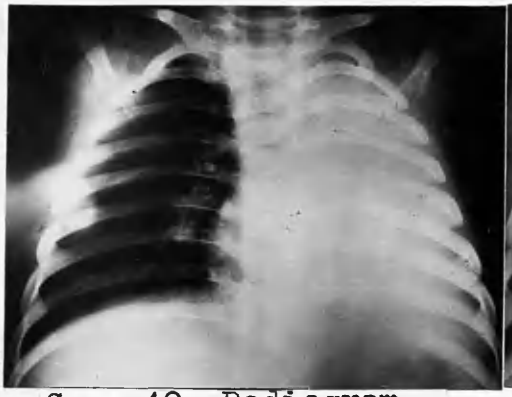
Male, aged 1 year, notified as a "laryngeal croup," and admitted to Ruchill Hospital on 3/12/47. Examination

revealed that there was laryngeal stridor and inspiratory indrawing of the lower intercostal spaces and the suprasternal area. The throat was slightly inflamed but clean, and there was no evidence of pulmonary infection. The patient was given 8,000 units of anti-diphtheritic serum, put in a steam tent, and penicillin and sulphadiazine administered. The temperature which was 100. F. on admission, returned to normal on 5/12/47; the croup, however, remained.

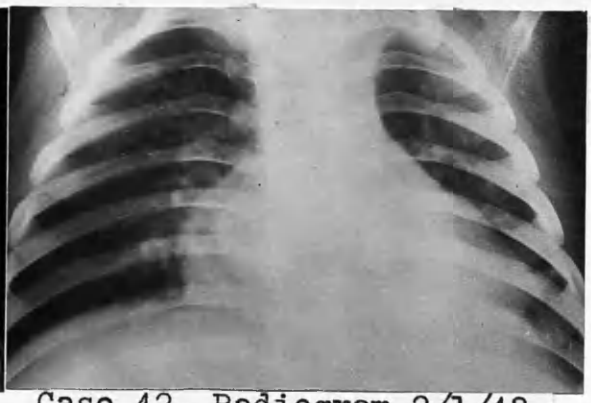
On the evening of 7/12/47, the temperature was sharply elevated to 102. F.. Next morning it was discovered that the P.N. over the left upper lobe was impaired, the R.M. was bronchial and powerful, and the trachea was slightly deviated to the left. There was still marked evidence of laryngeal obstruction. It was concluded that there was either consolidation of the left upper lobe, or occlusion of the left upper lobe bronchus with resultant atelectasis. The fact that there was a bronchial R.M. over the affected area did not exclude the latter alternative, as it might have been transmitted from the trachea by the collapsed lung tissue. On 9/12/47, the P.N. was impaired over the entire left lung, the R.M. was absent except in the upper lobe where it was bronchial, and the mediastinum had shifted markedly to the left. Complete collapse of the left lung was diagnosed, and this was confirmed by a radiogram taken on the same day. The absent R.M. pointed to the fact that the cause was obstruction of the main bronchus of the left lung. The intrapleural pressure on the affected side was lower than -16 cms. of water; this was the lowest reading on the instrument, and the water would have been aspirated into the pleural cavity from the manometer had the needle not been promptly withdrawn. A reading was not taken on the contralateral side, as the patient was gravely ill. Laryngoscopy did not reveal diphtheritic membrane, but the child succeeded in coughing up from time to time tough, inspissated material from which C. diphtheriae could not be grown. Laryngeal swabs were also negative. Unfortunately, bronchoscopy could not be employed, as a small enough bronchoscope was not available. The temperature settled on 10/12/47.

On 15/12/47, it was noted that the P.N. and R.M. over the left upper lobe were now normal; there was a triangular area of dullness at the left base over which weak tubular R.M. could be heard.

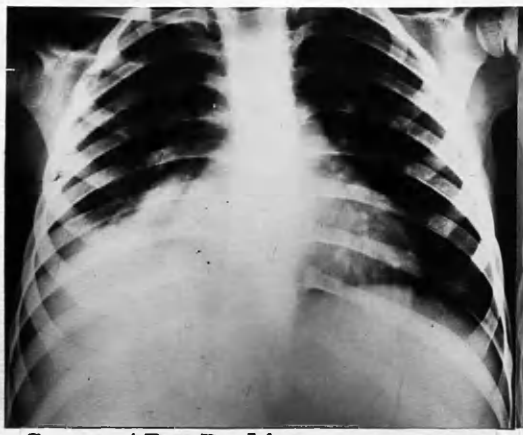
It was concluded that the left upper lobe bronchus was now clear, and, as the collapse of the left lower lobe remained, while weak tubular R.M. could be heard, it was believed that some of the obstructing mucus had been drawn



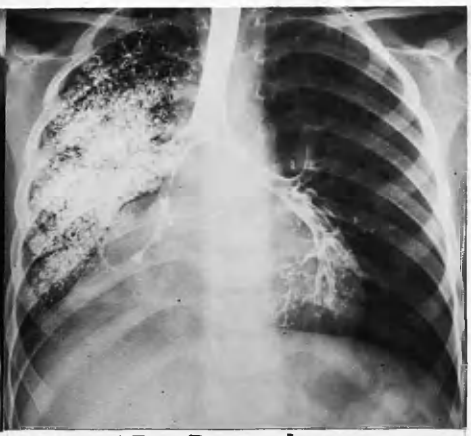
Case 42. Radiogram, 9/12/47. Atelectasis left lung.



Case 42. Radiogram 2/1/48. Re-expansion of left lung.



Case 43. Radiogram, 13/1/47. Atelectasis rt. lower lobe.



Case 43. Bronchogram, 13/5/47. The radio-opaque medium has failed to enter the rt. lower lobe.

down the left lower lobe bronchus, and finally occluded the finer bronchi or bronchioles in the manner described by Lander.[16,17] This theory was made more probable by the fact that the material coughed up was now not so tough in consistency. The laryngeal obstruction had by this time gone, and the general condition was good. By 17/12/47, it appeared that the atelectasis of the left lower lobe had largely cleared, and this was confirmed by a radiogram taken on the same day. There was still radiological evidence of slight atelectasis at the left base on 2/1/48, but as the patient was now clinically in excellent shape, he was dismissed with a request to the parents to bring him back for observation as an out-patient. He remained perfectly well, and a radiogram taken 3 months later, showed a normal lung.

An interesting feature of the case was the sharp rise of temperature when the atelectasis occurred. This phenomenon,as has been noted, is frequently observed when collapse occurs after abdominal operations, but proved a totally unreliable guide in the cases investigated in the whooping cough series.

Another noteworthy point was the presumptive change in the nature of the atelectasis of the left lower lobe from the proximal bronchial obstruction type to the peripheral bronchial obstruction type in the manner described by Lander.[16,17]

It is perhaps not strictly correct to regard this case as one of proximal bronchial obstruction without infection, but infection of the lung must clearly have been minimal.

Bronchography was not carried out after re-expansion of the lung to make certain that bronchiectasis had not resulted, because a general anaesthetic would have been required, and there was no reason to suppose that the complication had arisen.

The atelectasis of the left upper lobe seemed to be due solely to proximal obstruction of the main bronchus, and once it had cleared,complete recovery could be expected. In the left lower lobe, where the mucus was apparently drawn to the periphery, it is considered that a mechanical dilatation of the bronchi probably took place, but it must have been of brief duration, as the atelectasis speedily cleared up. During the period of observation since dismissal from hospital, the child has had no respiratory symptoms or abnormal signs on physical examination. Radiograms are shown opposite.

Case 43.

Male, aged 2 years, admitted to Ruchill Hospital on 1/10/46 with "pneumonia." The temperature was elevated to 102. F., but apart from a few scattered crepitations in the lung fields there was little to be made out on examination. The Mantoux test was positive. The temperature settled down in a day or two, but radiography revealed enlargement of the hilar glands, expecially the right. The tubercle bacillus was not demonstrated in the sputum, or in stomach washings. On 13/1/47, atelectasis of the right lower lobe was noted in a radiogram; clinically there was impairment of the P.N., and absent R.M. at the right base. On 13/5/47 the collapse was still present, and bronchography on that date showed that the iodised oil had failed to enter the affected area. The indications were, therefore, that this was an example of collapse due to pressure of enlarged hilar glands occluding the lumen of the main bronchus supplying the area.

Bronchoscopy would have clinched the diagnosis, but was not employed. The author, after he had performed the bronchographic investigation, did not see the patient again. A bronchogram is shown opposite page 123.

The reason for including this case in the group comprising bronchial occlusion without infection, is that the general condition of the patient improved with sanatorium treatment in comparison with what it had been even before the collapse took place, and there were no signs or symptoms to suggest that septic processes were at work in the affected area. It is almost certain, of course, that in time infection would supervene if the condition did not resolve; no doubt, however, cases in which occlusion of the bronchus is due to extrinsic cases escape infection for a longer period than those in which the cause is intrinsic.

Incomplete bronchial obstruction with infection almost invariably leads in practice to complete occlusion, and thus the two conditions may be dealt with simultaneously. The experimental evidence suggested that bronchiectasis would be produced due to distension of the bronchi by dammed up secretions. The following cases illustrate that this is what actually does take place.

Case 2.

Male, aged 37 years, admitted to Ruchill Fever Hospital with "pneumonia" on 6/9/47. On examination the

P.N. was impaired at the left base; the R.M. was also diminished, and accompanied by a few crepitations. The temperature was elevated,but settled in a few days. X-ray on 12/9/47 showed some infiltration in the left lower lobe. The temperature became elevated again on 25/9/47, though the patient felt fairly well. There was moderate production of sputum, examination of which failed to reveal the presence of the tubercle bacillus. On 14/10/47, the patient complained of severe pain in the left chest and neck. Examination revealed that the heart was displaced to the left side, the P.N. was impaired over the left lung, and there was absent R.M. on that side.

Atelectasis due to obstruction of the main bronchus of the left lung was diagnosed; had the obstruction been in the peripheral bronchioles, weak tubular breathing would have been expected over the affected area.

When the intrapleural pressure on the left side was taken, it was so low that the water in the manometer would have been aspirated into the pleural cavity had the needle not been promptly withdrawn; that is to say, it was much lower than -16 cms. of water, the lowest reading the apparatus recorded.

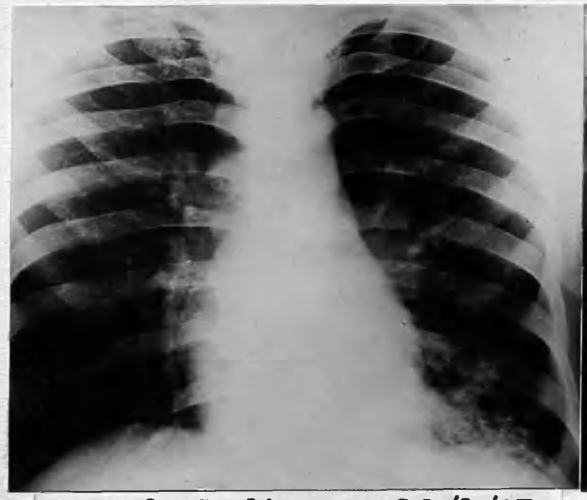
The intrapleural pressure in the right side was -12, -10 cms. of water; it was therefore also reduced if the normal is taken to be approximately -8,-4 cms. of water.

Straight x-ray confirmed the diagnosis of complete atelectasis of the left lung, and bronchography on 7/11/47 indicated that it was due to obstruction of the main bronchus of the left lung; although the patient was tilted well to the left, the iodised oil failed to enter the left lung, but ran into the right instead.

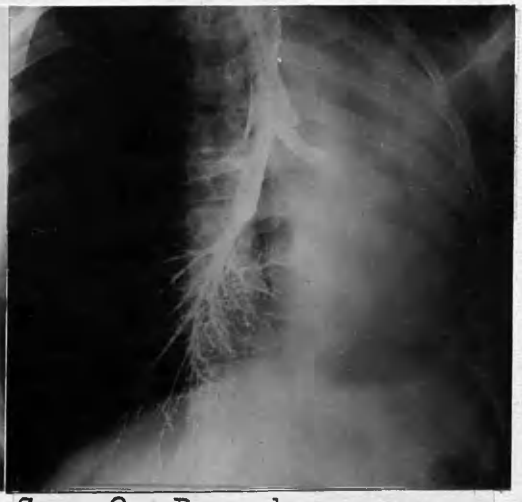
Bronchoscopy, performed by Mr. R.S.Barclay on 20/11/47, revealed what appeared to be caseous material blocking the left main bronchus.

The presence of M. tuberculosis in this substance was demonstrated when a biopsy specimen was examined. Following bronchoscopy, the bronchial lumen appeared to have been partly canalised, for bronchial R.M. could be heard in the upper part of the left lung; the R.M. was again absent in a few days.

On 28/11/47, the temperature,which had been unsettled, became elevated to 103. F.. Examination revealed that percussion over the left lung gave the "feel" associated with underlying fluid, and it was noted that the heart now appeared to have moved to more or less its normal position. Aspiration produced stinking pus. The temperature remained swinging violently until the death of



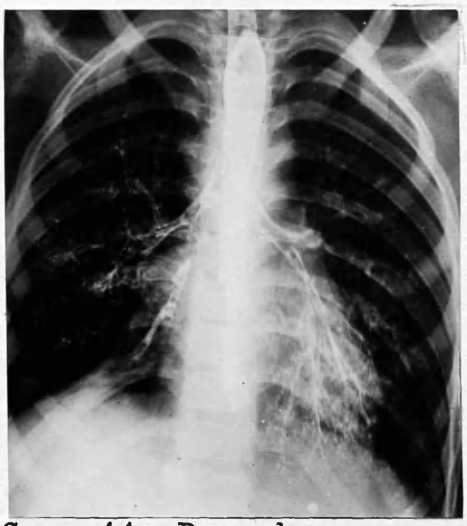
Case 2. Radiogram 12/9/47. Infiltration in left lower lobe.



Case 2. Bronchogram 7/12/47. Complete collapse of left lung. The radio-opaque medium has failed to enter left lung. The rt. lower lobe is partially drawn into the left hemithorax.

Case 2. Section of left lung. Note the destruction of bronchial walls and excavation of surrounding lung parenchyma. Broncho-pulmonary necrosis rather than bronchiectasis.

Photograph of section through lobectomy specimen from case in which the dilated bronchi were in communication with the atmosphere. Shown for contrast. Note the thick, fibrous bronchial walls.



Case 44. Bronchogram, 21/11/44. The radio-opaque medium has failed to enter the rt. lower lobe.

the patient on 6/12/47.

Post mortem examination was carried out. The left lung was completely atelectatic. The main bronchus was involved in a mass of caseating, tuberculous glands which had ulcerated into and occluded the lumen.

The bronchi were filled with pus, and grossly distended, especially those of the lower lobe. In some areas, the bronchial wall was more or less intact, but the striking feature was the marked broncho-necrosis. There were numerous areas in the left lower lobe in which the bronchial wall had been completely destroyed, and the surrounding parenchyma excavated. One of these large broncho-pulmonary cavities communicated with the pleural space which contained over two and a half pints of foul smelling pus.

This case, except for the development of a broncho-pleural fistula, is therefore an exact clinical counterpart of the experimental group of Tannenberg and Pinner, in which the main bronchus of a dog was infected and ligated.

Photographs of a radiogram, a bronchogram and of a section of the affected lung are shown opposite.

The following case is another example of the effects produced by obstruction of a main bronchus with infection distal to the block.

Case 44.

The author did not examine or perform bronchography on this case. The facts were culled from the records of Ruchill Sanatorium.

Male, aged 5 years, examined at a tuberculosis clinic on 12/6/44 because of persistent cough and night sweats. Admitted to Ruchill Sanatorium on 7/9/44. Examination revealed impaired P.N. and absent R.M. over the right lower lobe, and straight x-ray showed that the lobe was atelectatic. A bronchogram, reproduced opposite, taken on 21/11/44, demonstrated that the iodised oil failed to enter the right lower lobe. Atelectasis due to pressure of enlarged hilar glands on the main bronchus of the lobe was diagnosed. Lobectomy was performed by Mr. R. S. Barclay on 11/11/44, and the enlarged glands were noted at the operation to be pressing on and occluding the bronchus. Examination of the excised right lower lobe demonstrated that its bronchi were ectatic and filled with pus.

It has been suggested that a fixed, partial, bronchial obstruction even without infection might lead to bronchiec-

tasis, but Andrus[35] has shown convincingly that this is most unlikely to be the case.

He points out that in inspiration the obstructed and non-obstructed sections of lung are subjected to equal initial expansile movements. But, if, for example, the main bronchus of the left lower lobe is obstructed, because of the retarded inflow of gas the pressure will rise less rapidly in this lobe than in the remainder of the lung. The surrounding lung will therefore bulge into, and encroach upon, the gas-starved area. Because the obstructed section of the lung operates at subnormal gas pressure and subnormal elastic tension during the inspiratory phase of respiration, it would appear that it is specifically protected against mechanical injury at this period. The lower gas pressure, however, means a greater gas pressure difference for the propulsion of gas past the obstruction. This will partially, but, of course, not completely, counter-balance the obstructing effect. Also, since the rate of gas flow has slowed down distal to the obstruction, the gas pressure difference between the interior of a bronchus and its surrounding alveoli will be less under these conditions than for the bronchi elsewhere. The bronchi of an obstructed section of lung would therefore appear to be still further protected against mechanical injury on this account.

During expiration, gas will escape more rapidly from the normal than from the obstructed section of the lung, and therefore the normal part of the lung will recede from its position of encroachment on the obstructed part, until a point is reached when the gas pressure in both sections of the lung will be the same. As expiration proceeds, since gas will still escape more rapidly from the non-

obstructed section than from the partially obstructed, the position will be reversed, and as the obstructed section will now have the higher gas pressure, it may become slightly over-distended and encroach upon the area of the normal lung. But, of course, although the obstructed section contains relatively more gas at this time than the remainder of the lung, it still contains much less gas than it would in normal circumstances at the end of full inspiration. During the pause which normally follows expiration, the excess of gas in the obstructed section relative to the non-obstructed will escape, until equilibrium is obtained. But suppose it does not, and the obstructed section still contains an excess as opposed to the normal part, in the ensuing inspiration the inflow of gas to the obstructed section will be delayed until the pressure in the normal part increases and equilibrium is obtained.

According to Andrus,[35] the relative over-distension of the obstructed section at the end of expiration could not become an absolute over-distension as proposed by MacCallum.[100] He states, however, that if there were a pause at the end of inspiration, it would be conceivable that the obstructed portion of lung might receive its full quota of air, and therefore an actual or absolute over-distension would be expected to ensue during expiration. But this pause at the end of inspiration could only be produced voluntarily, and therefore this state of affairs is most unlikely to arise in practice.

A valvular bronchial obstruction has been proposed by MacCallum,[100] Warner,[81,101] Hedblom,[97] Brunn and Faulkner,[102] Ballon, Singer and Graham,[103] and a number of other authors, and Andrus[35] admitted that on some occasions, extrinsic pressure as from aneurysm, neoplasm, or enlarged lymph glands has

been observed to cause over-distension of the lung, but pointed out that even if this caused bronchiectasis its frequency must be negligible.

Bodies which are movable within the lumen of the bronchus such as foreign bodies, pedunculated tumours, or secretion, might move to, and be arrested at, positions of bronchial narrowing, and be moved away by the expiratory outflow of gas. They are therefore more likely to empty than over-distend the related section of lung. If, of course, there was already a fixed partial obstruction of a bronchus, and such a movable body were situated distal to it, it might block the obstructed portion on expiration, and allow an inflow of gas on inspiration, but it is scarcely conceivable that such a state of affairs could exist for long without causing inflammation, the production of secretion, and complete stenosis of the bronchus. Its practical importance is therefore slight.

It has also been suggested that the buckling of a layer of secretion occurring as a result of alterations in length of the bronchus during the respiratory excursion might give rise to a valvular action causing accumulation of air distal to the intermittent blockage; the ball-valve proposed by Warner[101] is of this type. Andrus[35] drew attention to the fact, however, that the mean size of the aperture would be the same in inspiration as in expiration, and the valve would thus act equally in both phases of respiration, and the effects would neutralise each other.

It therefore seems unlikely that partial bronchial obstruction without infection plays any great part in the production of bronchiectasis.

Comments on the effects of proximal bronchial obstruction.

A consideration of the clinical features of proximal bronchial occlusion confirms the experimental findings of Tannenberg and Pinner.[86]

If the occlusion is of such brief duration that infection does not supervene in the bronchial tree distal to the obstruction, atelectasis without bronchiectasis is the result. Clinically, this can only be inferred from the fact that the lung and bronchial tree return to normal when the obstruction is removed.

If the occlusion is of long duration, infection of the bronchi distal to the block almost invariably ensues, and as a result, they become distended with retained secretions.

If the infection is severe enough, the walls of the bronchi may speedily become necrotic, and excavation of the surrounding pulmonary tissue follow, thus giving the picture of broncho-pulmonary necrosis. This was well illustrated in Case 2 (p. 124).

It has already been pointed out in the discussion on the experimental evidence, that the mechanical stresses set up by the occurrence of atelectasis have probably nothing to do with the production of the bronchiectasis or broncho-pulmonary necrosis seen in these cases - the essential factor is obviously the damming back of the infected secretions.

Yet although bronchiectasis can be produced by proximal occlusion of a main bronchus, it seems strange that observers such as Adams and Escudero,[71] and Tannenberg and Pinner,[86] can seriously put it forward as the main mechanism in the production of the disease; examinations of post mortem and lobectomy specimens prove beyond all

question that in the vast majority of cases there is no obstruction of the main bronchus. Besides, where there is proximal bronchial occlusion, iodised oil will not enter the affected area, and in modern times bronchiectasis can scarcely be called to mind without evoking the mental picture of a bronchogram demonstrating the dilatations. Further, although factors such as neoplasm, enlarged glands, or foreign body may obstruct a main bronchus and incidentally give rise to bronchiectasis, it cannot be said that interest in these cases centres on the bronchial dilatation; it is focussed rather on the cause of the obstruction. The clinical features of the illness, also, do not correspond in the least with the generally accepted impressions of bronchiectasis.

Proximal obstruction of a main bronchus quite clearly cannot be looked on as a usual cause of the disease.

2. Obstruction of bronchioles or finer bronchi.

Various authors, as has been noted earlier in the chapter, have commented on the close association between atelectasis and bronchiectasis, and as the bronchograms which illustrate their work invariably show dilated bronchi in the atelectatic area, it is obvious that the obstruction causing the atelectasis is in the peripheral part of the bronchi. When this is the case, Lander and Davidson's experiments[17] supply an excellent theoretical basis for explaining why the bronchi contained in the atelectatic area should be dilated.

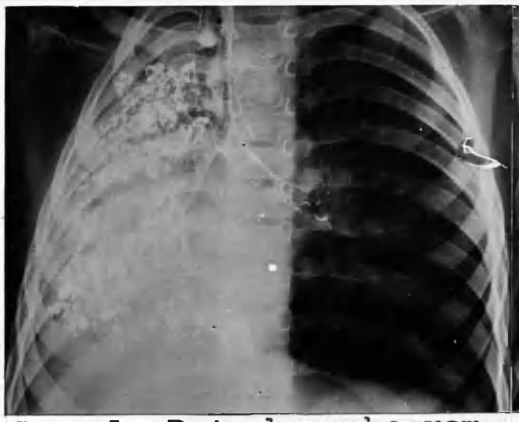
If atelectasis causes a mechanical dilatation of the bronchi in the manner they suggest, however, then in every case of collapse of a lung or lobe, it should be possible to demonstrate dilatation of the bronchi in the obstructed

area, provided, of course, that the bronchi are patent up to their terminal parts. If this could not be done, then the presumption would be that there has been some flaw in the calculations of those advocating the theory.

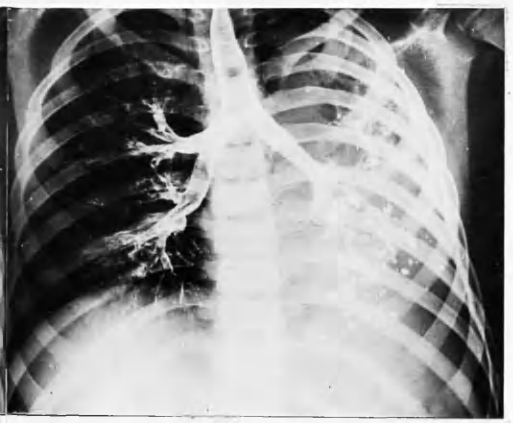
It should be noted that in testing these postulates, it is well at the outset to consider only atelectasis of the extent mentioned. Speculation on the effects of collapse of lesser extent somewhat complicates the issue. It is not to be expected, for example, that collapse involving only a few lobules would lead to bronchial dilatation, as expansion of the surrounding lung tissue could quite obviously completely compensate for the "lost space." Assuming, therefore, for the moment, that atelectasis may cause bronchial dilatation, the exact extent required may be regarded as a fine point which has not yet been investigated.

If, however, collapse of the greater part of a lobe did not produce bronchial dilatation then grave doubts would arise as to whether the theory had any substance at all, and in order to obtain first hand evidence on the matter, bronchography was carried out in fourteen consecutive cases of atelectasis of the "peripheral obstruction" type encountered in Ruchill Fever Hospital or Ruchill Sanatorium. The collapse involved at least the greater part of a lobe. In every case bronchial dilatation in the atelectatic area was demonstrated.

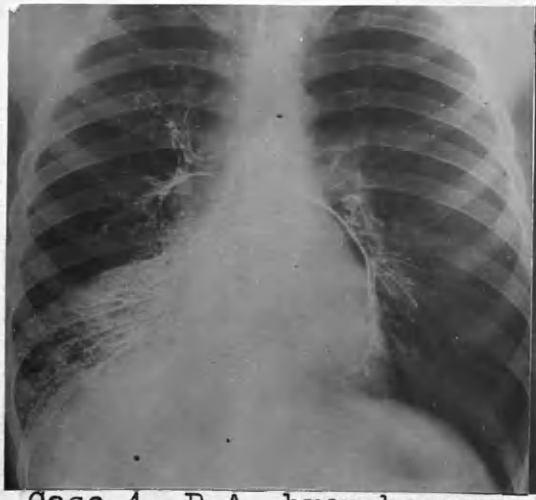
The findings in this series of cases certainly strongly support the atelectatic theory of the causation of bronchiectasis, and they are in accordance with those of many other authors. Warner[80] speaks of bronchial dilatation "invariably" occurring in lobar atelectasis, and Richards[77] declares that a triangular basal shadow in an



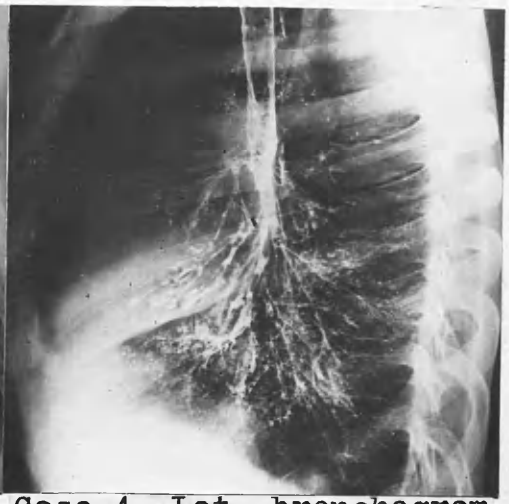
Case 1. P.A. bronchogram showing complete collapse of rt. lung with associated bronchial dilatation.



Case 45. P.A. bronchogram showing complete collapse of left lung with associated bronchial dilatation.



Case 4. P.A. bronchogram in which little evidence of bronchiectasis can be detected.



Case 4. Lat. bronchogram clearly showing bronchiectasis in collapsed rt. middle lobe.

x-ray is "pathognomonic" of bronchiectasis. These statements are, of course, not strictly correct, because as has been seen, the relationship need not arise if the atelectasis has been caused by proximal bronchial obstruction, but the writers are obviously referring to the type of collapse now under discussion, and a perusal of the leading articles on bronchiectasis does not reveal a case which conflicts with their views. If instances have occurred which do, it is extremely surprising that the opponents of the atelectatic theory have not brought them forward, since it would be difficult to imagine any more devastating evidence. The presumption must be that no such cases have been available.

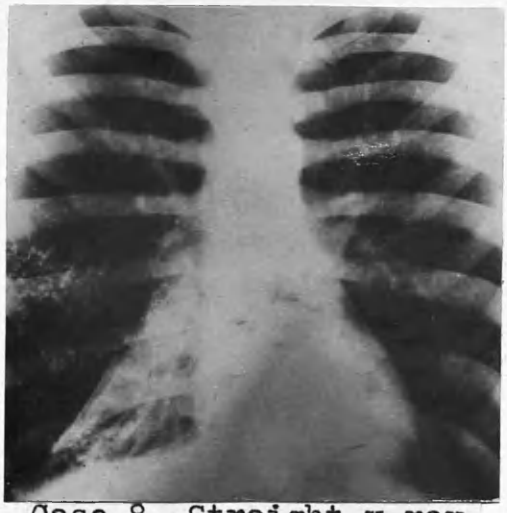
Notes on ten of the fourteen examples of atelectasis of a lobe or lung in which bronchial dilatation was demonstrated are given below, and the appropriate bronchograms accompany the cases. As the only point relevant at the moment is the bronchial dilatation in the atelectatic area, details of the cases are not given, though some are fully considered in other connections elsewhere. The remaining four cases are dealt with in the discussion on reversible bronchiectasis which follows immediately afterwards. (Case 17,p.136; Case 18,p.137; Case 13,p.137; Case 16,p.137.)

Case 1.

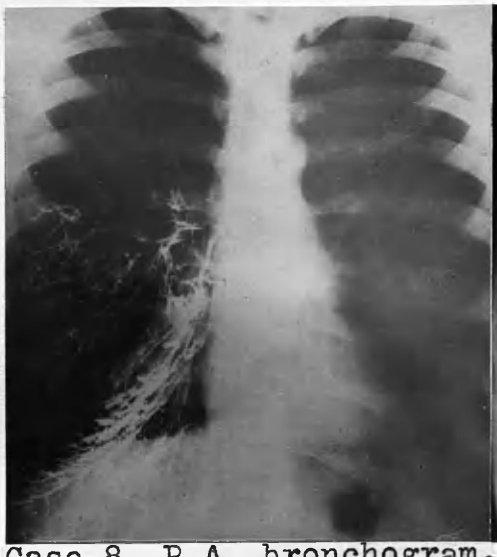
Female, aged 5 years, seen at a tuberculosis clinic on 4/2/46, and later admitted to Ruchill Sanatorium for investigation. Complete atelectasis of the right lung was diagnosed clinically and radiologically. A bronchogram is shown opposite demonstrating the bronchial dilatations in the right lung. The straight x-ray has already been shown. (Opposite page 6.)

Case 45.

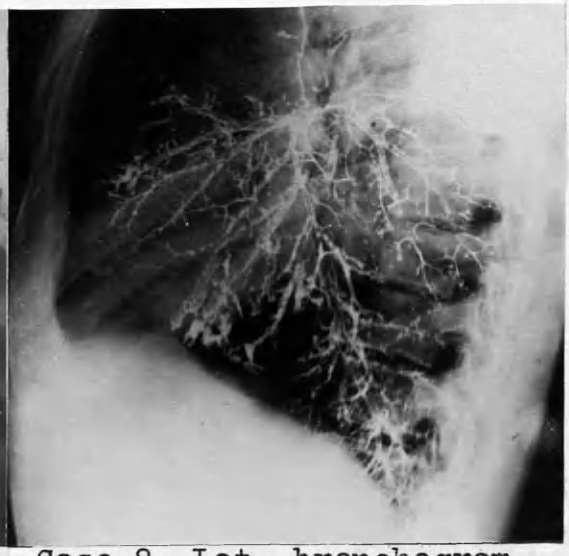
Male, aged 7 years, admitted to a Glasgow hospital on 20/12/45 with pain in the left chest. "Effusion with consolidation of the left lung," was diagnosed. Examination of the sputum failed to reveal M. tuberculosis.



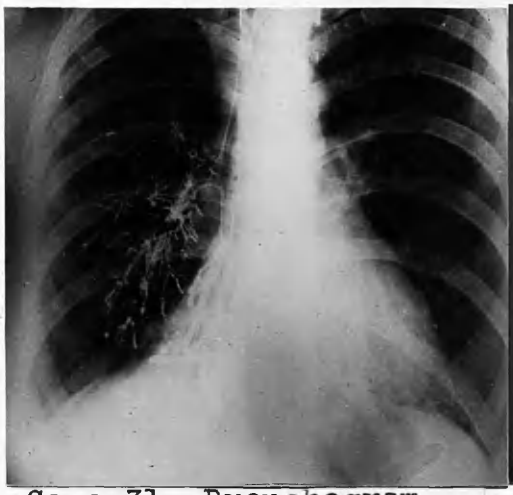
Case 8. Straight x-ray showing collapse of rt. lower lobe. Some old iodised oil is present.



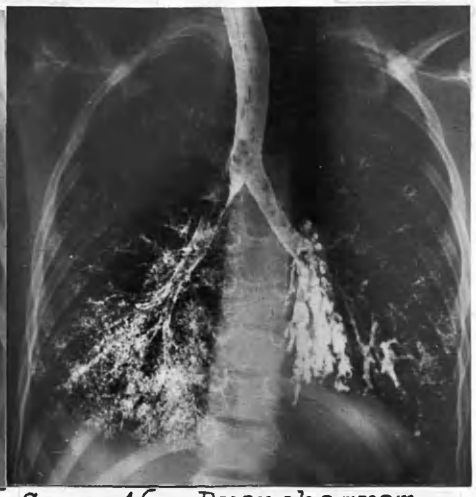
Case 8. P.A. bronchogram. Cylindrical bronchiectasis rt. lower lobe.



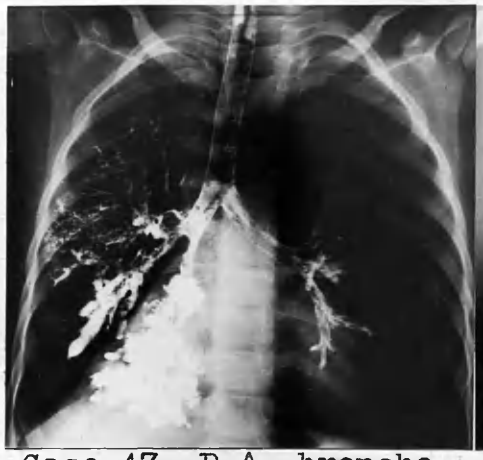
Case 8. Lat. bronchogram. Cylindrical bronchiectasis is seen to affect chiefly the ant. basal segment of the rt. lower lobe.



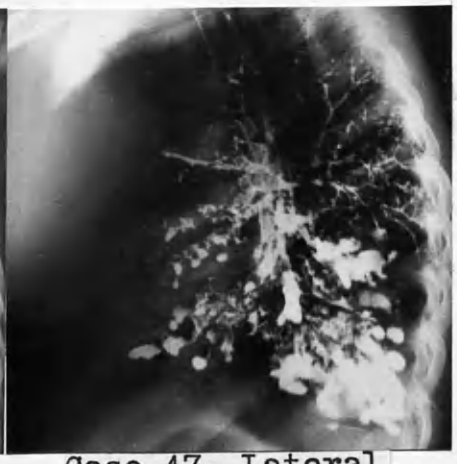
Case 31. Bronchogram showing collapse of rt. lower lobe with associated dilatation of the contained bronchi.



Case 46. Bronchogram showing atelectasis and bronchiectasis of left lower lobe. There is also bronchiectasis affecting the lingula.



Case 47. P.A. bronchogram showing atelectasis and bronchiectasis of rt. lower lobe, and also bronchiectasis of the rt. middle lobe.



Case 47. Lateral bronchogram.

The case was seen by Mr. R. S. Barclay on 20/2/46. On examination, the heart was displaced to the left, the P.N. was impaired over the left lung, and weak tubular breathing could be heard on the left side of the chest. From the clinical findings, and also the radiological picture, complete atelectasis of the left lung with bronchiectasis was diagnosed, and the case was transferred to Ruchill Sanatorium. A bronchogram, reproduced opposite page 133, taken on 29/7/46, revealed patent dilated bronchi in the left lung. The condition showed no improvement, and left pneumonectomy was performed on 7/8/46. The patient was dismissed on 9/11/46.

The author did not see this patient until after the pneumonectomy was performed. He is indebted to M. R. S. Barclay for information on the clinical findings, and permission to photograph the bronchogram.

Case 4.

Male, aged 12 years, admitted to Ruchill Fever Hospital with "pneumonia" on 20/8/47. Radiograms showing collapse of the right middle lobe have already been shown (Opposite p.9.) and further details are given in the next chapter. Bronchograms are shown opposite page 133.

Case 8.

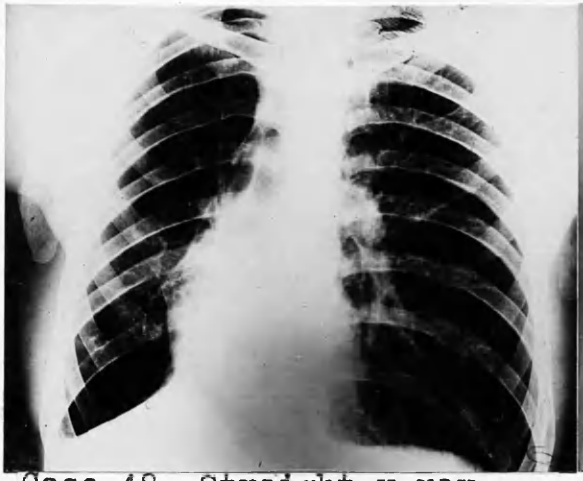
Male, aged 25 years. Admitted to Ruchill Sanatorium with collapse of right lower lobe on 22/4/47. The case is again considered in the next chapter. Bronchograms are shown opposite, and also a straight x-ray.

Case 31.

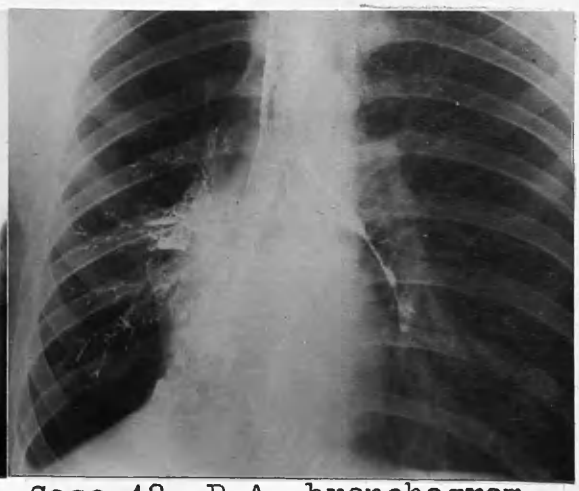
Female, aged 17 years, admitted on 12/11/46 on account of persistent productive cough. Examination revealed a triangular area of dullness close to the spine at the right base. Weak tubular breathing and crepitations could be heard over this area. A straight x-ray on 17/12/46 revealed atelectasis of the right lower lobe, and bronchography demonstrated dilatation of the contained bronchi. A bronchogram is shown opposite.

Case 46.

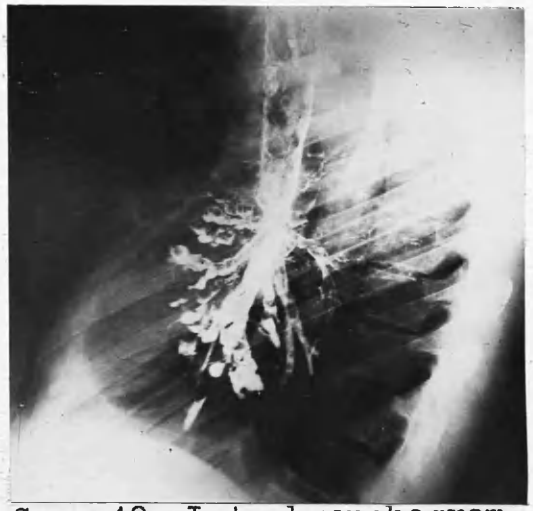
Male, aged 12 years. Investigated as an out-patient on 9/5/47. Clinical and radiological evidence of atelectasis of the left lower lobe. The bronchogram opposite reveals the marked bronchial dilatation in the affected area. There is also bronchiectasis of the lingular process.



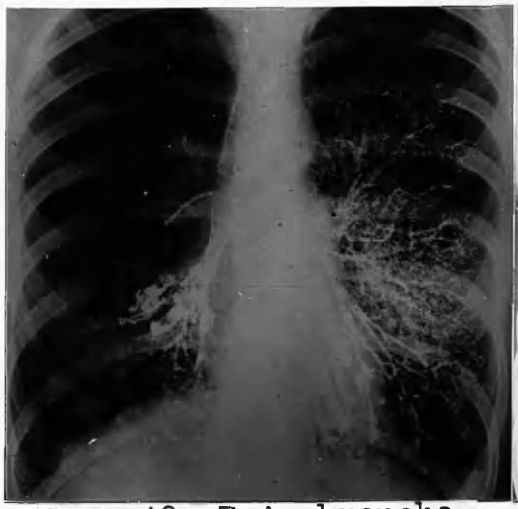
Case 48. Straight x-ray. Collapse of right lower lobe. Appearance in rt. middle lobe area suggestive of bronchiectasis.



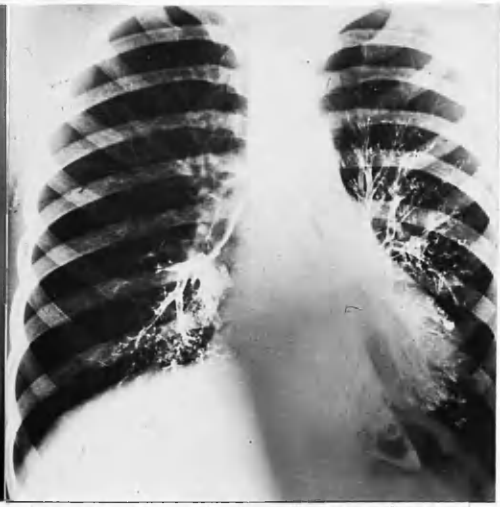
Case 48. P.A. bronchogram rt. lung. Bronchiectasis in collapsed rt. lower lobe. Also bronchiectasis in rt. upper and middle lobes.



Case 48. Lat. bronchogram, right lung.



Case 49. P.A. bronchogram. Atelectasis and bronchiectasis of left lower lobe. Also rt. middle lobe bronchiectasis.



Case 7. P.A. bronchogram. Atelectasis and bronchiectasis of left lower lobe.

Case 47.

Female, aged 21 years, with a long history of productive cough and a number of attacks of "pneumonia." Referred from a tuberculosis clinic to Ruchill Hospital for bronchography on 15/1/47. Physical examination revealed a triangular area of dullness at the right base close to the spine, with broncho-vesicular breathing and medium crepitations in the area. Radiology confirmed the diagnosis of collapse of the right lower lobe, and the bronchogram opposite page 134 shows the crowded, dilated bronchi in the atelectatic area. The middle lobe bronchi are also ectatic.

Case 48.

Male, aged 26 years, admitted to Ruchill Sanatorium on 20/3/47 with a long history of productive cough and attacks of "pneumonia." Clinically, crepitations all over the right lung and broncho-vesicular breathing over an area of dullness at the right base. Straight x-ray revealed ring-like markings in the right middle lobe area suggesting ectatic bronchi, and collapse of the right lower lobe. Bronchography demonstrated bronchiectasis of the right upper and middle lobes, and also of the collapsed right lower lobe. A radiogram and bronchograms are shown opposite.

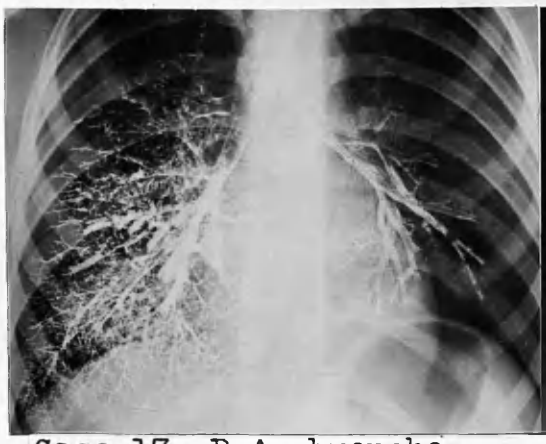
Case 49.

Female, aged 28 years, admitted to Ruchill Sanatorium on 12/6/47 with a long history of productive cough. Physical examination and straight x-ray revealed collapse of the left lower lobe. Bronchography demonstrated the bronchial dilatation in the left lower lobe, and also bronchiectasis of the right middle lobe. A bronchogram is shown opposite.

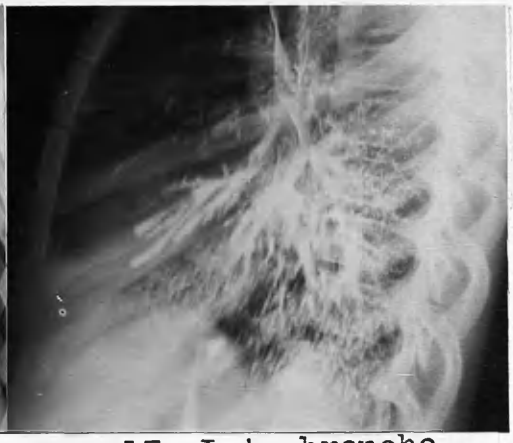
Case 7.

Female, aged 5 years. Admitted to Ruchill Sanatorium on 10/1/47. Clinically and radiologically, atelectasis and bronchiectasis of the left lower lobe. The straight x-ray has already been shown (Opposite p.10), and the case is more fully discussed in the next chapter in another connection. A bronchogram is shown opposite.

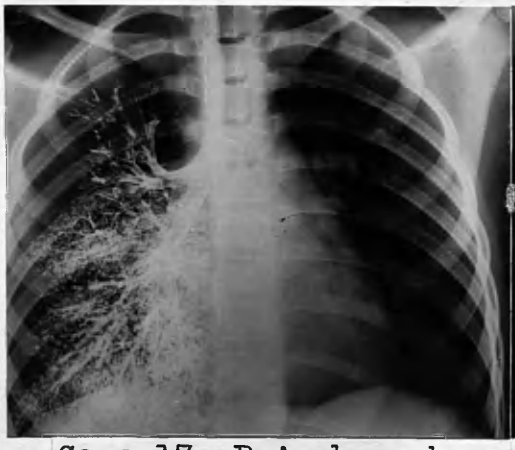
It will be remembered that, as a result of their experiments, Lander and Davidson[17] came to the conclusion that bronchiectasis was a reversible phenomenon. What they actually showed was that bronchial dilatation occurr-



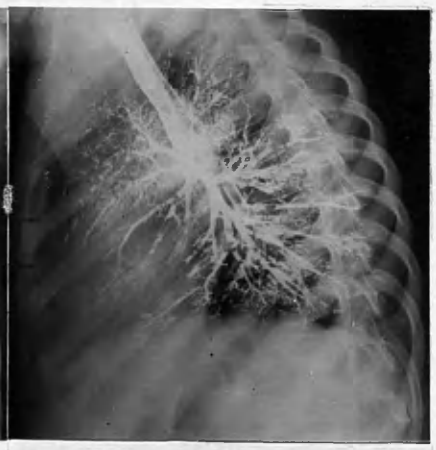
Case 17. P.A. bronchogram, 25/12/46. Bronchial dilatation in rt. middle and rt. lower lobe areas.



Case 17. Lat. bronchogram, 25/12/46, which shows that the bronchial dilatation is in the rt. middle lobe and the ant. basal segment of the rt. lower lobe.



Case 17. P.A. bronchogram, 3/3/47. Normal bronchial tree.



Case 17. Lateral bronchogram, 3/3/47. Normal bronchial tree.

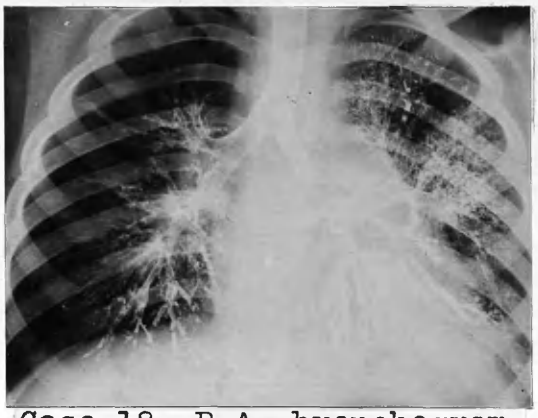
ing in an atelectatic lobe could be abolished by inducing a reversal of the forces which produced it. Their experiments therefore merely suggested, and did not prove their deduction, but evidence has accumulated which tends to show that, with reservations, their conclusions were correct. Cases have been demonstrated by Ochsner,[104] Findlay,[105] Jennings,[106] Lander and Davidson,[17] Franklin,[107] Fleischner,[24] Ogilvie,[18] Blades and Dugan,[108] Lander,[15] Wearing[49] and other writers in which atelectasis and "bronchiectasis" have been present, and with the clearing of the atelectasis, the bronchial dilatation has disappeared.

In the course of the present investigation on whooping cough, eight cases were brought to light. In four of them, the extent of the atelectasis was comparatively small, and consequently, if the inference be permitted at this stage, the bronchial dilatation was also slight. These cases will therefore be considered later in another connection.

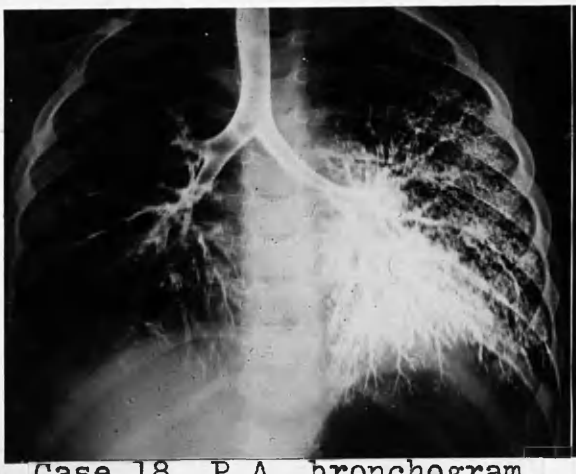
In the others, which will now be discussed, the atelectasis was marked, and the bronchial dilatation also obvious.

Case 17.

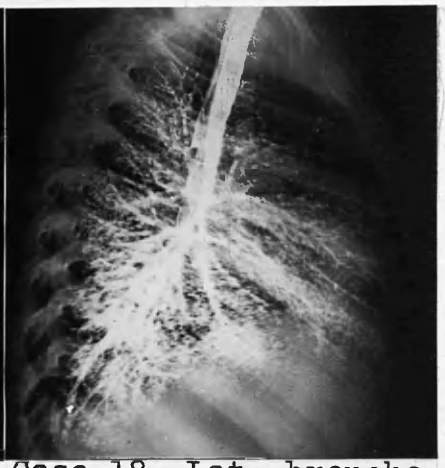
Female, aged 6 years. Illness noted on 12/11/46, and admitted to hospital with whooping cough on 16/11/46. Straight x-ray on 4/12/46 showed evidence of atelectasis affecting the right middle lobe, and later it was seen that the anterior basal segment of the right lower lobe had become involved. By 25/12/46 the collapse in the area mentioned had become marked. Bronchography on that date revealed marked dilatation of the bronchi contained in the collapsed portion of lung. A right A.P. was induced, details of which will be given later. A bronchogram taken on 3/3/47, after the A.P. had absorbed, showed that the atelectasis had cleared up, and the bronchial dilatation had disappeared. Radiograms and bronchograms, taken in September 1947 revealed a normal lung and bronchial tree. Bronchograms are shown opposite.



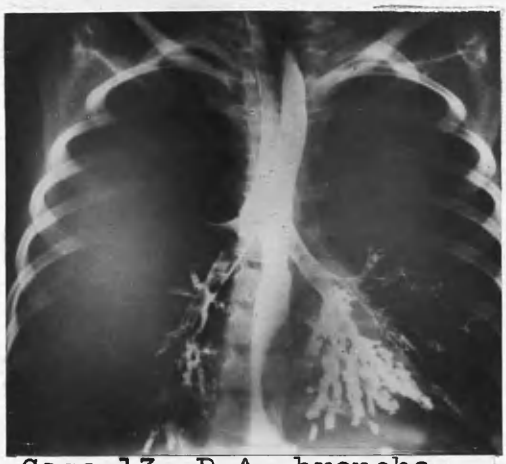
Case 18. P.A. bronchogram, 25/3/47. Collapse of left lower lobe with bronchial dilatation. Difficult to see in the reproduction owing to the heart shadow.



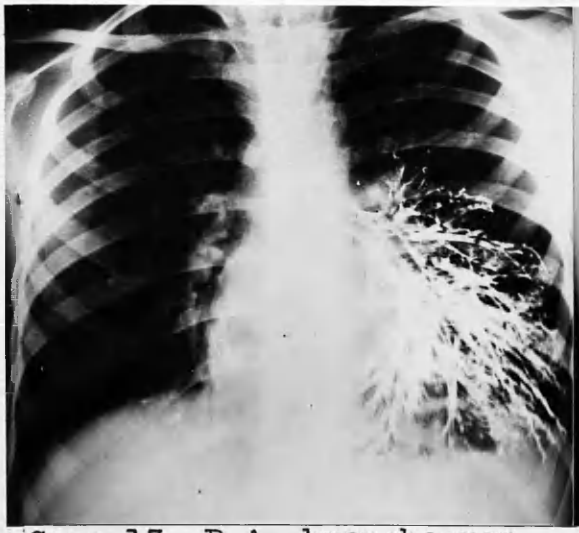
Case 18. P.A. bronchogram, 22/5/47. Normal left bronchial tree.



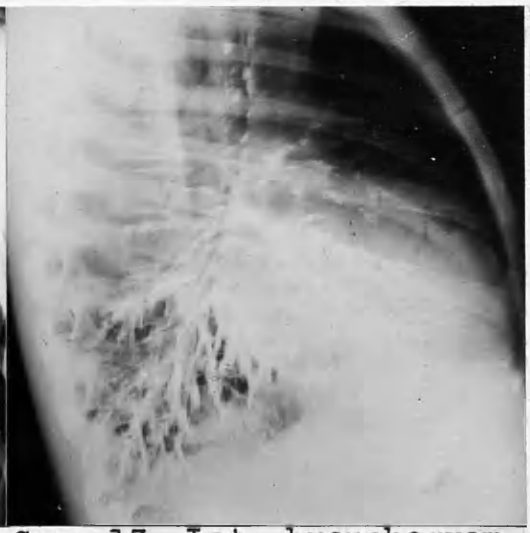
Case 18. Lat. bronchogram, 22/5/47. Normal left bronchial tree.



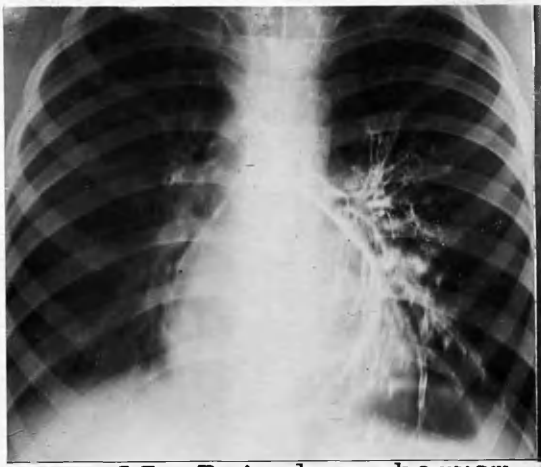
Case 13. P.A. bronchogram, 13/5/47. Atelectasis of left lower lobe with marked dilatation and crowding of the contained bronchi.



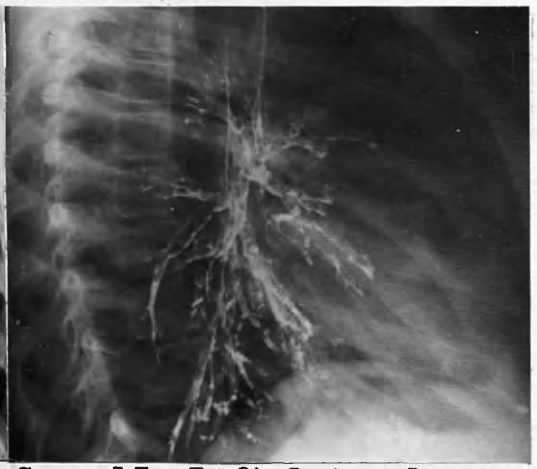
Case 13. P.A. bronchogram, 17/9/47. The left lower lobe has re-expanded, and the left bronchial tree is normal. The heart shadow in the reproduction has been lightly shaded with pencil to make the outlines of the bronchi in this area more obvious.



Case 13. Lat. bronchogram, 17/9/47. Normal left bronchial tree.



Case 13. P.A. bronchogram, 25/2/48. Normal left bronchial tree.



Case 13. Left lateral bronchogram, 25/2/48. Normal left bronchial tree.

Case 18.

Male, aged 3 years. Illness noted on 12/12/46, and admitted to hospital on 16/12/46 with whooping cough. A straight x-ray on 4/2/47 revealed that atelectasis was present in the left lower lobe. Another film on 25/3/47 showed that the collapse had become extensive, and bronchography on the same date demonstrated marked dilatation of the bronchi in the affected area. Again A.P. treatment was employed. On 30/4/47, straight x-ray suggested that the atelectasis was clearing, and it had disappeared by 8/5/47. The A.P. was allowed to absorb. Bronchography on 22/5/47 demonstrated that the bronchi had returned to normal. Bronchograms are shown opposite.

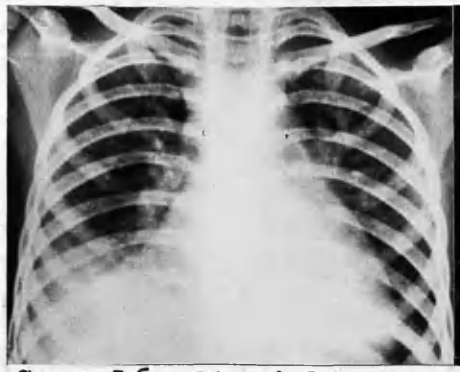
Case 13.

Male, aged 5 years. Illness noted on 26/3/47, and admitted to hospital on 9/4/47 with whooping cough. There was clinical evidence of broncho-pneumonia, and atelectasis of the left lower lobe. This was confirmed by x-ray on 18/4/47 which showed some broncho-pneumonic infiltration in both lungs, and extensive collapse of the left lower lobe. Bronchography on 13/5/47 revealed gross dilatation of the bronchi contained in the collapsed portion of lung. A left sided A.P. was induced on 21/5/47, and was maintained until 11/8/47, when x-ray indicated that the atelectasis was clearing up. The A.P. had completely absorbed by 25/8/47, and x-ray showed that the atelectasis had disappeared. The author was unable to carry out bronchography until 17/9/47, but when this was done it was seen that the bronchial tree was normal. Bronchography on 25/2/48 again showed that the bronchial tree was normal. Bronchograms are shown opposite.

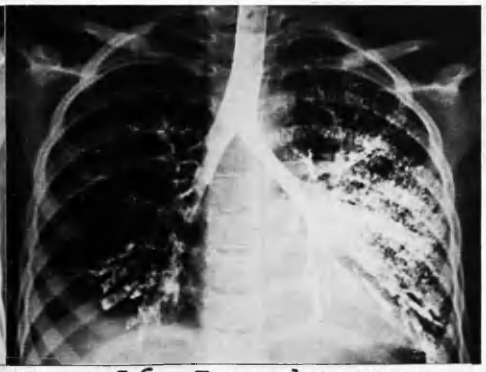
The above three cases will be discussed in detail in the next chapter.

Case 16.

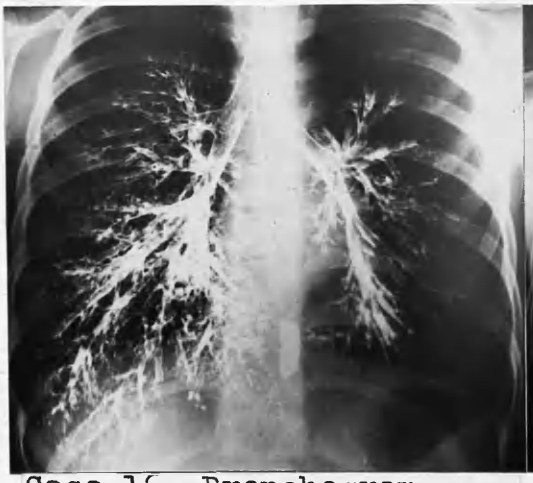
Female, aged 6 years. The patient became ill on 26/1/47, and was admitted to hospital with whooping cough on 7/2/47. Clinical examination revealed crepitations at both bases. Weekly radiograms demonstrated no abnormality until 11/3/47, when fairly extensive atelectasis of both lower lobes was noted. The P.N. was impaired at both bases, crepitations could be heard, and expiration was prolonged and somewhat blowing. Bronchographic investigation was therefore carried out on 18/3/47, and revealed that bronchial dilatation was present in the collapsed right and left lower lobes. A.P. treatment was not employed in this case; physiotherapy such as postural drainage and



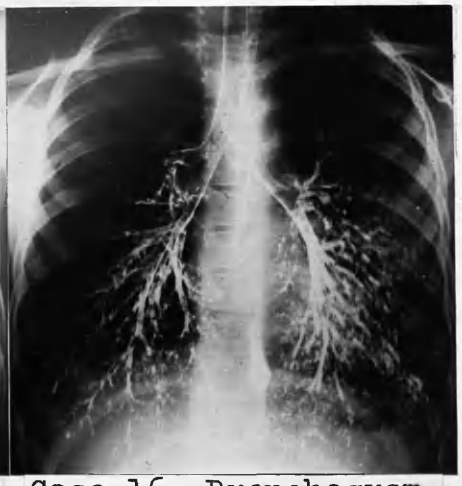
Case 16. Straight x-ray 11/3/47. Collapse of rt. and left lower lobes. The heart shadow has been shaded in blue to suggest the triangular collapsed area seen in the original plate, but not satisfactorily reproduced.



Case 16. Bronchogram, 18/3/47. Bronchial dilatation in right and left lower lobes. Note crowding of bronchi in collapsed areas.



Case 16. Bronchogram, 1/6/47. Re-expansion of the collapsed lower lobes has taken place. The right lower lobe bronchi are no longer crowded together, but slight dilatation still persists. The left bronchial tree has not been adequately filled with iodised oil.



Case 16. Bronchogram, 5/1/48. The rt. and left bronchial trees are normal.

squeezing the affected side of the chest was adopted in the hope of dislodging plugs of sputum from the bronchioles. By 6/5/47 the atelectasis of the right lower lobe had cleared up, and by 13/5/47 the atelectasis of the left lower lobe had also disappeared. A bronchogram taken on 1/6/47 demonstrated that the bronchi had almost returned to their normal calibre. There was, however, still slight but definite bronchiectasis in both lower lobes. The patient was then dismissed. For various reasons it was not until January 1948 that she could be brought back to hospital for bronchography. In November1947, the mother stated the girl could not come because she was confined to bed with a "bad cold", and it was feared on learning this news that the bronchiectasis had become permanent. Clinical examination on 5/1/48, however, revealed no abnormality of the chest, and the generalcondition of the patient was excellent. Bronchography on the same day demonstrated that the bronchial tree was normal. A straight x-ray and bronchograms are shown opposite.

Evidence of infection as indicated by temperature and production of sputum, varied in the four cases.

In Case 17, the temperature was not elevated at the onset of the atelectasis or during its course, and production of sputum was scanty.

In Case 18, the temperature was elevated to 100. F. on two occasions for two or three days during the course of the atelectasis, and production of sputum, which was at first moderate in amount, became scanty after a fortnight, and gradually dwindled to nothing.

Infection was severe in Case 13. On admission, the temperature was 103. F. Penicillin and sulphadiazine were administered, and after swinging for five days, the temperature settled down. Production of sputum remained extremely copious, however, and frequent and violent paroxysms of coughing harassed the patient. Loss of weight was also a prominent feature. A point of great interest was the striking manner in which the cough subsided after the induction of the artificial pneumothorax on the affec-

ted side. Before this operation was performed, the patient had been coughing persistently day and night; immediately afterwards, this symptom, though still remaining to a slight degree, ceased to be at all troublesome. Sputum production also rapidly declined. Both symptoms completely disappeared in two months.

Infection in Case 16, as in Case 18, was moderate in degree. During the course of the atelectasis the temperature was elevated on one occasion to 102. F., and settled in two days. Production of sputum was scanty. Cough was rather troublesome at first, but gradually disappeared.

The interpretation of cases such as the above, is still much in dispute. Lisa and Rosenblatt[34], for example, declare, "Despite Roentgen appearances of ectasia in these cases, there is no proof of its actual existence..... The ultimate disappearance of the alleged dilatation probably is presumptive proof to the contrary."

There appears, however, to be absolutely no grounds for statements such as these, and the authors do not put forward a shred of real evidence in support of their views. Bronchography has been performed on thousands of cases of atelectatic bronchiectasis, and the appearance of bronchial dilatation on the bronchogram has been shown at post mortem, or after lobectomy, to have indicated the actual state of affairs. If an author wishes to state that bronchography is misleading in this regard, then the onus is surely on him to produce proof substantiating his claims.

Blades and Dugan[108], who demonstrated four cases similar to the above, were unwilling to admit that the condition was really bronchiectasis. They did not doubt, however, that the bronchi were dilated; in fact they noted that bronchoscopic aspiration of secretion from the affected

bronchi did not, as in cases of permanent bronchiectasis, reduce their calibre to more normal dimensions.

Blades and Dugan[108] noted these instances of atelectasis in cases of "atypical pneumonia." They talk of "atelectasis and atypical pneumonia," but it is plain from their description that they mean that the "atypical pneumonia" was, in fact, infected atelectasis. It is necessary to state this in order to make the following quotation comprehensible to a reader not familiar with the complete article, as the term "atypical pneumonia" has been used by other authors in another sense.

"We have been able to demonstrate, however, that following atypical pneumonia, lipiodol bronchograms may occasionally be misleading, and give the erroneous impression that the condition is true bronchiectasis. ....Speculation on the possibility that in some instances permanent damage to the bronchi may result from the effects of atypical pneumonia would be premature. Campbell and his associates[109] in their excellent paper on atypical pneumonia describe a case in which the right lower lobe remained atelectatic for a period of 46 days. A bronchogram demonstrated not only atelectasis, but also localised fusiform bronchiectasis. Our experience suggests it may be another example of false bronchiectasis, and that it is possible that subsequent visualisation might reveal a normal bronchial tree..... It seems possible, however, that damage to the bronchi in some cases could result in irreversible changes. Fleischner[26] condemns the term pseudo-bronchiectasis because he considers reversible bronchiectatic cases represent an early stage of true bronchiectasis. We are sceptical of this conception of the disease, and have elected to employ the term pseudo-

bronchiectasis for our cases."

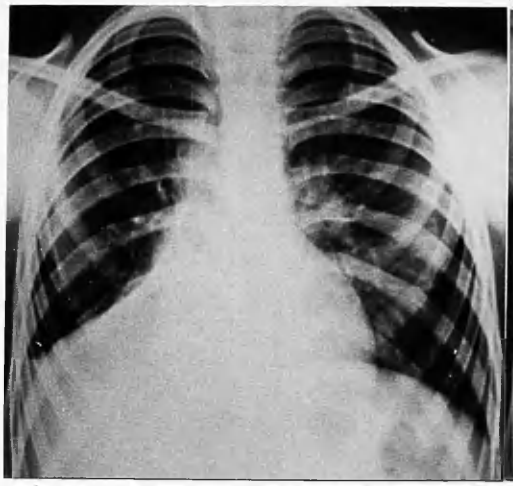
Blades and Dugan[108] go on to suggest that possibly pseudo-bronchiectasis may remain for as long as three months, though in most instances they are of the opinion that at least some of the bronchi will have returned to normal in four to six weeks. They do not give any reason for these views.

It is plain, therefore, that although Blades and Dugan[108] refuse to call these cases bronchiectasis, they are rather uneasy about the matter. They admit that if cases could be shown where permanent bronchiectasis resulted from "atypical pneumonias" they would have to alter their opinions, and agree with Fleischner's theory[24] that the dilatation of the bronchi contained in an atelectatic portion of lung represents an early stage of bronchiectasis. Two such cases follow.

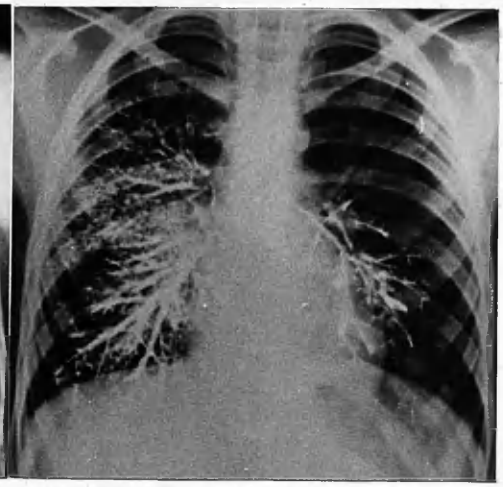
Case 50.

Male, aged 10 years, admitted to Ruchill Sanatorium on 20/1/46 with a history of troublesome productive cough, loss of weight, and poor appetite. The onset of the illness was approximately three months previously.

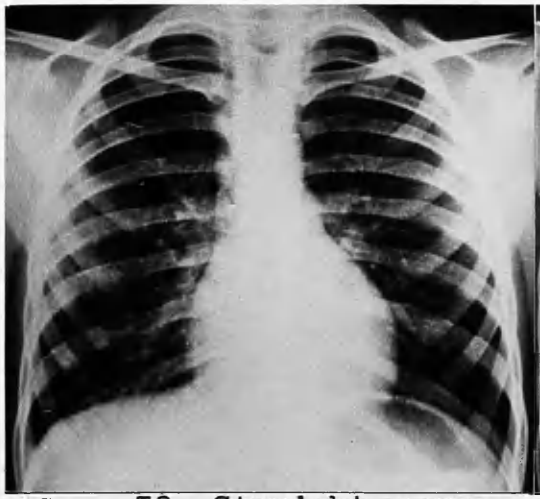
Clinical examination revealed impaired P.N. and R.M. at the right base. Expiration was prolonged and rather blowing in the same area, and inspiration was accompanied by a few moist crepitations. A radiogram on 5/2/46 showed atelectasis of the right middle and lower lobes. The presence of breath sounds over the affected area indicated it was due to bronchiolar obstruction, and hence it was deduced that the bronchi contained in the collapsed portion of lung would be dilated. By 7/5/46 the atelectasis had almost cleared up, but in order to make certain that the presumed bronchial dilatation had disappeared in the affected portion of lung in conformity with Blades' views on the subject, bronchography was carried out on the same date. The bronchogram, however, demonstrated that bronchial dilatation had persisted; there was cylindrical bronchiectasis of the right middle and lower lobes. The patient was dismissed and brought back for an x-ray on 7/8/46. There was no evidence of atelectasis, but there were increased bronchial markings at the right base. The



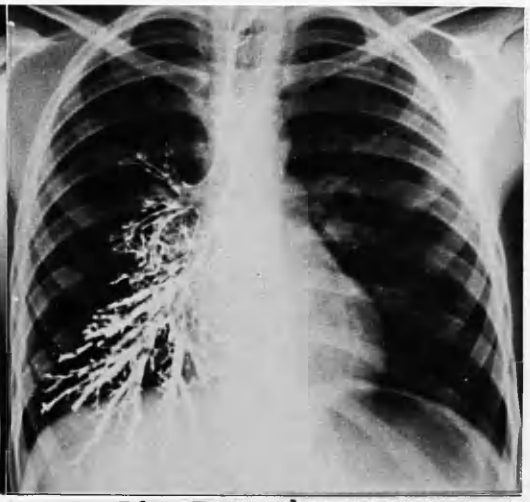
Case 50. Straight x-ray, 5/2/46. Collapse of rt. middle and lower lobes.



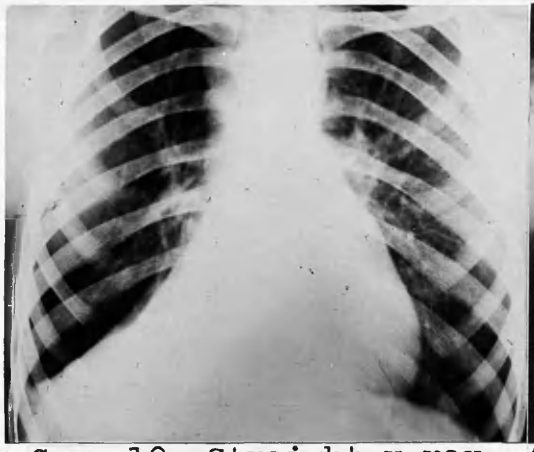
Case 50. Bronchogram, 7/5/46. Atelectasis has almost disappeared. Cylindrical dilatation of rt. middle and lower lobe bronchi.



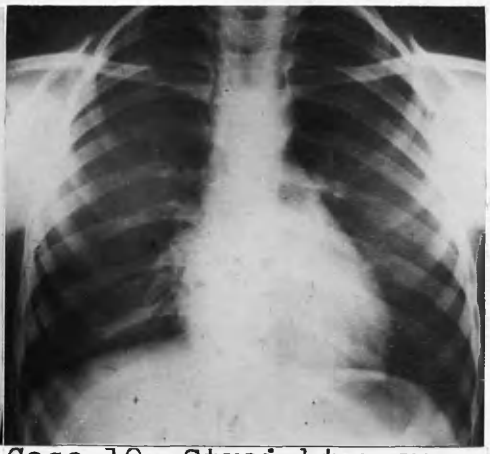
Case 50. Straight x-ray, 7/8/46. Rt. lung fully re-expanded. Slight increase in pulmonary striation in rt. cardiophrenic angle.



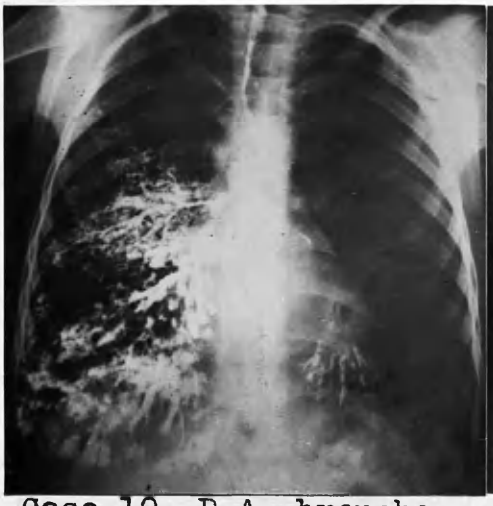
Case 50. Bronchogram, 16/1/47. Cylindrical dilatation of rt. middle and lower lobe bronchi, more marked than on 7/5/46.



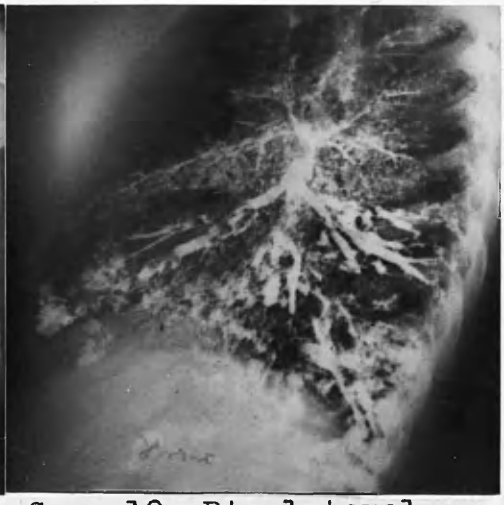
Case 10. Straight x-ray, 19/5/47. Collapse of rt. middle and lower lobes.



Case 10. Straight x-ray, 27/6/47. Re-expansion of collapsed portion of lung. Slight haziness in rt. cardio-phrenic angle.



Case 10. P.A. bronchogram, 28/7/47. Bronchial dilatation in rt. middle and lower lobes.



Case 10. Rt. lateral bronchogram, 28/7/47. Bronchial dilatation in rt. middle and lower lobes.

patient still had a troublesome productive cough, which became more marked in succeeding months, and the general condition deteriorated. He was re-admitted on 7/1/47. He had a productive cough, and the general condition was poor. Clinical examination revealed that expiration at the right base was prolonged and rather blowing, and crepitations could be heard over the right lower and middle lobes. Bronchography on 16/1/47 demonstrated bronchiectasis of the right lower and middle lobes, more marked if anything than in the bronchogram of 7/5/46.

The author did not see this patient until his re-admission to hospital on 7/1/47. The clinical findings and x-ray plates before that date were obtained from the records of Ruchill Sanatorium. Radiograms and bronchograms are shown opposite.

Case 10.

Female, aged 7 years, seen at a tuberculosis clinic on 7/5/47. There was a history of occasional pain in the right chest, troublesome productive cough, anorexia, and listlessness. Clinical examination revealed impaired P.N. at the right base, and weak tubular breathing in that area. A radiogram taken on 19/5/47 demonstrated atelectasis of the right middle and lower lobes. The patient was admitted to an observation ward of Ruchill Sanatorium on 25/6/47. On examination there was some prolongation of expiration at the right base, close to the spine, and crepitations over the right middle and lower lobes; it appeared that the atelectasis had now cleared up. This was confirmed by radiography on 27/6/47; the affected area appeared somewhat hazy, but the lung had re-expanded. Bronchography on 28/7/47 revealed bronchiectasis of the right middle and lower lobes. The productive cough and symptoms of general malaise did not improve, and the affected lobes were excised by Mr. R. S. Barclay on 2/10/47. Examination of the lobectomy specimen confirmed that the bronchi were infected and ectatic. Radiograms and bronchograms are shown opposite.

Reviewing the last six cases, some features of great interest arise.

In the four examples of "reversible" bronchiectasis, the onset of the atelectasis was known to within a few days. In all of them, bronchography revealed dilated bronchi in the atelectatic areas. This is exactly what would have been expected from the theoretical consider-

ations arising from a study of the experimental evidence previously cited, because since the iodised oil freely entered the bronchi, and a weak broncho-vesicular respiratory murmur could be detected over the affected areas, the atelectases were clearly of the bronchiolar obstruction type, experimentally and clinically demonstrated by Lander and Davidson.[17]

Since there was absolutely no reason to suppose that the bronchial tree was in any way abnormal before the occurrence of the collapse, these cases strengthen the evidence previously adduced, that in an atelectasis of this nature, the forces set in motion cause a mechanical dilatation of the contained bronchi.

The fact that the bronchial dilatation vanished with the disappearance of the atelectasis seemed to prove that bronchiectasis may, in some circumstances, be reversible.

Blades and Dugan[108] and other authors, as has been mentioned, have, however, hesitated to accept the bronchial dilatation seen in such cases as "true" bronchiectasis.

The last two examples in which the atelectasis cleared up, but in which permanent bronchiectasis remained, appear conclusively to set these doubts at rest.

The persistence of bronchial dilatation seen in these two cases after the atelectasis had disappeared was not, however, a state of affairs that Lander and Davidson[17] contemplated in 1938 after their investigations on the subject. It will be remembered that they declared that they were of the opinion that bronchiectasis was a reversible phenomenon "even after years." Clearly they had in mind only cases of bronchiectasis associated with atelectasis, and since their series of lobectomy cases proved to their satisfaction that infection was minimal and the

bronchial walls unscathed, they had no reason to suppose that the bronchial dilatation was other than a purely mechanical phenomenon which would disappear if the atelectasis cleared up.

Ogilvie,[18] in 1941, in the course of an essay on bronchiectasis, demonstrated a case in which there was atelectasis with associated bronchial dilatation, and in which the bronchial dilatation persisted after the disappearance of the atelectasis, but he apparently did not fully appreciate the significance of these phenomena. Lander,[15] however, must have done so, for in 1946 in a publication in which he noted Ogilvie's case, and was able to demonstrate a similar one, he had somewhat modified his views. Here he came to the conclusion, surely made obvious by these cases, and the two examples in the present publication, that the bronchial dilatation produced by an atelectasis will only disappear on the subsequent re-expansion of the lung, if, in the interval, the bronchi have not sustained damage which prevents them from reverting to their original calibre.

What factors are likely to produce such damage?

Lander[17] in his original article apparently assumed that mere prolonged mechanical distension would not be sufficient, but it seems highly improbable that any elastic material, using "elastic" in its ordinary unscientific sense, could be stretched for years without impairment of its power to revert to its original dimensions.

Whether or not this is the case, the most important factor would appear to be infection of the mechanically dilated bronchi. If this complication arose during the atelectasis, it is obvious that such damage might be inflicted on the bronchial walls, that their elasticity would be impaired, and the bronchi would not revert to

their normal calibre even when the abnormal distending stress to which they were exposed was removed by the re-expansion of the collapsed area of lung. The longer the duration of the atelectasis, of course, the greater the chance of a supervening infection.

The question now arises - what may properly be regarded as "bronchiectasis" at the present day?

Blades[108] stated that he was prepared to withdraw the term "pseudo-bronchiectasis" which he had applied to his cases of "atypical pneumonia" and call them "reversible bronchiectasis" if instances could be shown where permanent bronchial damage resulted. This has been demonstrated in the foregoing. Since he also declared that he regarded bronchiectasis as a "progressive disease marked by symptoms of infection," it is apparent that this opinion also would have to be altered. Kornblum[110] stated in 1944, "If reversible bronchiectasis is true clinical bronchiectasis, our concept of the disease must be revised," and a study of the material now available, makes it plain that this conclusion is irresistible.

In cases which have just been demonstrated, bronchography revealed atelectasis with dilated bronchi in the affected area. What, at that stage, should the condition have been called? It was clearly impossible to predict either whether the atelectasis would clear up at all, or whether, if it did disappear, the dilated bronchi would revert to normal.

If the condition were to be termed "pseudo-bronchiectasis," then in the event of the atelectasis remaining, at what point in time would it be justifiable to call the condition permanent or "true" bronchiectasis, and remove the lobe? This is a puzzling question which will later

be discussed more fully.

If evidence of infection is regarded as the criterion, should, then, the generally accepted term "dry bronchiectasis" be discarded in cases which appear to be absolutely "dry" and non-infected?

One such case (Case 27, p.68.), has already been shown. It may be recalled that the patient was a boy of nine years in which marked dilatation of the right upper lobe bronchi was suspected purely on account of the physical signs noted on routine examination. Over a period of fifteen months, no evidence of infection was noted. The probability is that the condition resulted from a collapse of tuberculous origin in early childhood, and although the bronchi remained permanently dilated, no doubt any infection there may have been eventually disappeared owing to the excellent drainage of the upper lobe.

In view of the considerations which have just been put forward, it is suggested that "bronchiectasis" should now mean any pathological dilatation of a bronchus. The word "pathological" would exclude the slight physiological dilatation of the bronchi which occurs in inspiration.

This use of the word "bronchiectasis" seems justified in view of the fact that bronchial dilatation appears to be the only factor common to all the conditions which, it appears, must now be included in the disease.

Case 17 (p.136.) showed bronchial dilatation without evidence of infection, and completely recovered.

Case 13 (p.137.) showed bronchial dilatation with evidence of gross infection (elevated temperature and abundant sputum), and completely recovered.

Case 50 (p.141) showed bronchial dilatation with evidence of infection, though less severe than in Case 13,

and did not recover.

Case 27 (p.68.) showed permanent bronchial dilatation without evidence of infection, and customary usage already classifies it as a case of "dry bronchiectasis."

The cases are all obviously stages of the same condition, and there appears no reason why the same term should not be applied to each.

Henceforth, "bronchiectasis" will therefore be used in the sense suggested above, as it is much less clumsy than the circumlocutions necessary to avoid it in cases as yet controversial. Where necessary, the word will be qualified by terms such as "reversible", "permanent", "compensatory" and so on.

It will have been noted that in the two cases in which bronchial dilatation remained after the atelectasis had cleared up, the bronchiectasis was looked upon as undubitably permanent.

This view was not adopted merely because the affected bronchi did not immediately revert to their normal condition on re-expansion of the collapsed portion of lung.

If, in an atelectatic area, the bronchial walls were quite unimpaired, it would, indeed, be expected that with the removal of the abnormal distending stresses consequent on the disappearance of the collapse, the bronchial calibre would at once become normal.

There remains, however, the possibility that during the course of the atelectasis the bronchial walls might sustain damage, but not irreparable damage, and therefore although on the re-expansion of the collapsed lung the bronchial elasticity might be impaired sufficiently to prevent a complete return of the bronchi to their normal calibre, ultimate recovery would not be out of the question.

This theory is supported by the findings in the whooping cough case in which artificial pneumothorax therapy was not employed.(Case 16, p.137). After the atelectasis had cleared up, bronchography revealed that although the bronchiectasis had practically disappeared, it was still to some extent in evidence. Complete recovery was, however, eventually achieved. In addition, it was noted in one of the "reversible" cases (Case 13, p.137) in which artificial pneumothorax was induced, that the introduction of air into the pleural cavity although it greatly reduced the bronchial dilatation, did not completely abolish it; this was also noted by Lander[17] in one of his cases.

The two cases in which the bronchiectasis was eventually deemed permanent, were therefore carefully observed for a period after the atelectasis had cleared up. In both of them the productive cough remained, and the general condition was poor. Lobectomy was performed on the girl, (Case 10, p.142), four months after the atelectasis had disappeared, and examination of the excised lobes showed that the bronchi were ectatic, infected,and damaged beyond hope of recovery.

Bronchography was carried out in the boy eight months after the disappearance of the atelectasis, and revealed that bronchial dilatation in the affected area was, if anything, worse than before. The symptoms of the disease had also become more marked, and the general condition had deteriorated. Lobectomy would have been carried out in this case, but the boy had to go with his family out of the area.

Once an atelectasis has cleared up, there is therefore no great difficulty in assessing the damage, if any, sustained by the bronchi.

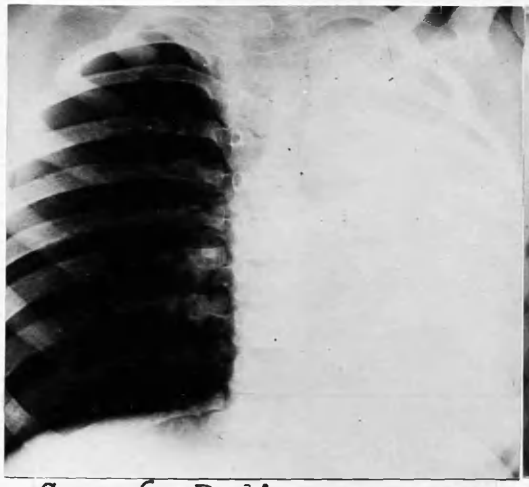
But suppose the case is seen at the onset of the atelectasis, and bronchography demonstrates the presence of bronchial dilatation in the area. How long can the atelectasis last, and hope still be entertained that the associated bronchiectasis is potentially reversible?

Campbell[109] recorded a case of lobar collapse associated with dilatation of the contained bronchi; re-expansion of the lung took place in 46 days, and there was apparently a complete return to normal. Blades,[108] commenting on this case, stated that he believed that had bronchography been carried out it would have revealed a normal bronchial tree. He also hazarded the opinion, though without any supporting evidence, that recovery of the bronchi might be possible in cases in which the atelectasis persisted for as a long a period as three months.

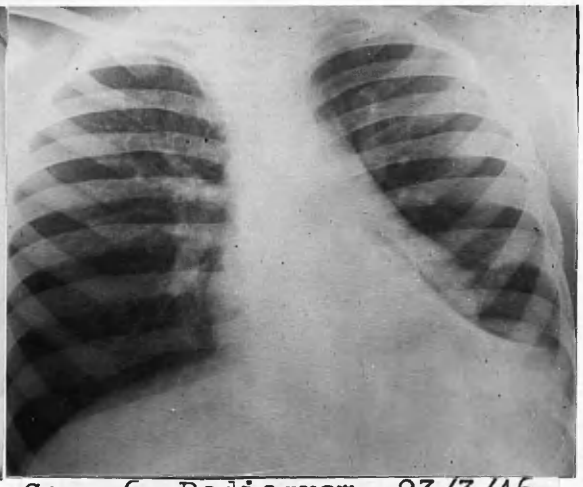
In a case already shown in this work, the bronchi returned to normal after re-expansion of a lobe which had been atelectatic for 18 weeks (Case 13, p.137), but an artificial pneumothorax was induced and maintained on the affected side, and it may be that this measure made conditions more favourable for recovery of the bronchi. This question will be discussed in the next chapter.

In the following case, however, where an atelectasis was left to itself, re-expansion of the affected area took place in three months, and subsequent bronchography revealed a normal bronchial tree.

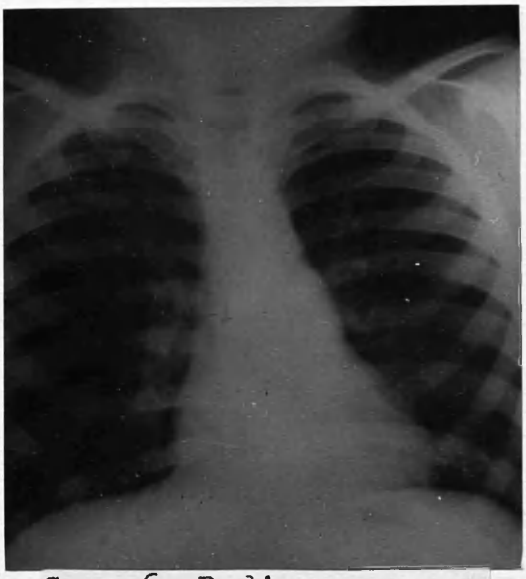
Unfortunately, however, bronchography was not carried out while the atelectasis was still present, and consequently bronchiectasis was never demonstrated. The significance of the case will therefore depend upon whether it is believed that bronchial dilatation is a necessary concomitant of this type of atelectasis, but the case is too



Case 6. Radiogram, 15/3/46. Complete collapse of left lung. Very marked shift of trachea and heart to the left.



Case 6. Radiogram, 23/3/46. Collapse of left lower lobe only.



Case 6. Radiogram, 10/6/46. Complete re-expansion of left lung.

interesting not to record.

Case 6.

Male, aged 8 years, admitted to Stobhill Hospital on 12/3/46 complaining of breathlessness, and pain in the left chest of a few days duration. Examination revealed complete collapse of the left lung, and this was confirmed by a radiogram taken on 15/3/46. By 23/3/46, the upper lobe had re-expanded, but the lower lobe remained atelectatic. The patient was seen by Mr. Bruce Dick, and transferred to Ruchill Sanatorium on 29/4/46. On examination, the P.N. was impaired over the left lower lobe, and weak tubular breathing accompanied by an occasional crepitation could be heard over the same area. There was little evidence of infection as judged by cough and spit. The atelectasis showed signs of disappearing by 25/5/46, and expansion of the lobe was complete by 10/6/46, Bronchography was not carried out until 14/1/47 when the left bronchial tree was seen to be normal. The patient did not come under the care of the author until after the atelectasis had cleared up, and the information on the previous findings was obtained from the records of Ruchill Sanatorium. Radiograms are shown opposite. Unfortunately the bronchograms of 14/1/47 are not available for reproduction.

It is clear from the cases which have been shown, and from the work of Blades and Dugan,[108] Campbell[109] and others, that atelectasis may persist for a considerable time without permanent damage to the bronchi resulting. But what the maximum period may be is quite unknown, judging from the literature on the subject. Wearing,[49] in a recent article called attention to this point, as he encountered a case of reversible bronchiectasis in which there was reason to believe that recovery had taken place after several months.

This matter, however, is of no small practical importance, because once permanent bronchiectasis is diagnosed, it may be said that in general the sooner lobectomy is performed the better, in order to minimise the risk of spread to unaffected parts of the lung. On the other

hand, failure to realise that atelectatic bronchiectasis may be potentially reversible in a particular case, may lead to the unnecessary excision of a lobe. Blades[108] pointed out this danger in the course of his article on "pseudo-bronchiectasis", and Barclay[111] encountered a case of atelectatic bronchiectasis sent to Mearnskirk Hospital for lobectomy, in which the collapsed area re-expanded, and in which subsequent visualisation of the bronchial tree revealed that the associated bronchiectasis had disappeared.

It would appear that six months would not be too long a period to await re-expansion of the lung before embarking on surgical measures.

When the onset of the atelectasis is unknown, and this is the usual state of affairs, the problem is still more complicated, for the collapse may have been present for months before the patient comes under observation. Generally speaking, it is rare that the history in such cases enables the investigator to ascertain the time of the occurrence of the atelectasis with any confidence, for the onset is commonly insidious. An account of symptoms such as productive cough and general malaise may be helpful in roughly assessing the duration, but the position is most unsatisfactory.

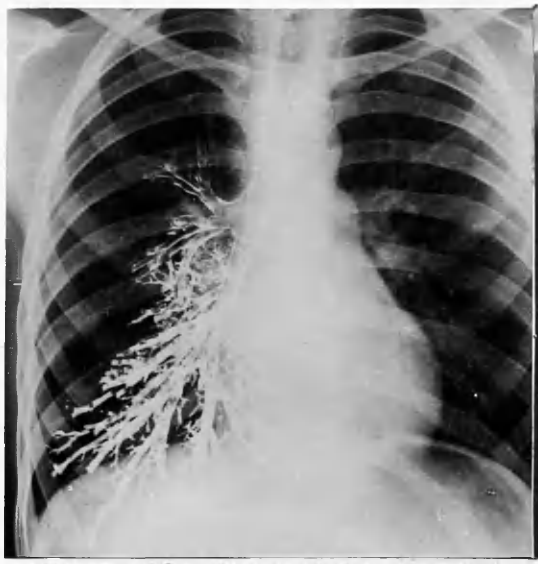
A method of determining whether or not the dilated bronchi in an atelectasis are capable of returning to normal in the event of re-expansion of the affected lung tissue is therefore highly desirable, but a review of the literature fails to reveal any attempt to solve the problem. A test for determining the potential reversibility of bronchiectasis in an area of pulmonary collapse is suggested in the next chapter.

Comments on the effect of obstruction of bronchioles or finer bronchi.

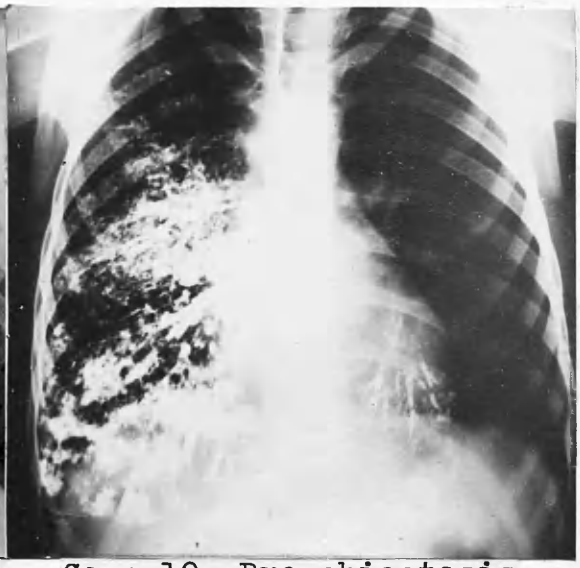
It is concluded from the experimental evidence discussed, and from a consideration of the cases which have just been studied, that obstruction of bronchioles or finer bronchi gives rise to atelectasis, and when the atelectasis involves at least the major portion of a lobe, ectasia of the contained bronchi is produced.

The bronchiectasis is at first purely a compensatory phenomenon, and will at this stage disappear if the lung re-expands. Should, however, the bronchial walls be severely enough damaged during the period when the atelectasis is present, the bronchi will lose their elasticity and remain dilated even if re-expansion of the lung takes place. The necessary damage in the vast majority of cases is inflicted by the combined action of two factors - mechanical dilatation and infection. It is believed that mechanical dilatation of the bronchi, if prolonged enough, may be alone sufficient. There is no direct evidence, however, that such is the case; still less what length of time is required. If infection is present when the atelectasis occurs, or supervenes after it has taken place, the bronchiectasis is obviously much more likely to become permanent.

It is considered that in some cases, although the elasticity of the bronchial walls may be impaired to such an extent that the bronchial calibre does not return to normal immediately after re-expansion of the collapsed portion of lung has removed the dilating stresses to which the bronchi have been exposed, the damage sustained by the bronchial walls may be such that ultimate recovery is possible. It therefore appears to be advisable, even in



Case 50. Bronchiectasis right middle and lower lobes.



Case 10. Bronchiectasis right middle and lower lobes.

cases in which bronchial dilatation persists after re-expansion of the lung has taken place not to resort to surgical measures until observation over a considerable period indicates that the bronchiectasis is permanent.

Evidence that Atelectasis is the Principal Factor in the Causation of Bronchiectasis.

So far, it has been claimed that when atelectasis caused by bronchiolar obstruction takes place, permanent bronchiectasis may result, and evidence has been put forward which, it is believed, proves this point beyond the shadow of a doubt. It will now be suggested that atelectasis is the principal factor in the production of the vast majority of cases of bronchiectasis. The contention, of course, cannot be proved, but it is believed that it is possible to adduce strong evidence as to its probability.

This theory is becoming ever more widely accepted, and is supported by Andrus[35], Coope[112], Lander[15] and others.

Yet a few years ago, such a postulate could scarcely have been put forward. Opponents of this view could merely have pointed to the fact that many cases of bronchiectasis do not have an associated atelectasis - evidence of infection is the predominant feature. Cases such as the two demonstrated in this work, and the other two published by Ogilvie[18] and Lander[15], in which atelectasis cleared up but bronchiectasis remained, show, however, that such an argument would be wide of the mark.

Looking at the bronchograms opposite this page and opposite pages 155 and 156, one would say that they were all typical examples of bronchiectasis without associated atelectasis; Ogilvie[18] would have placed them in his "broncho-pneumonic" group. Yet the first two broncho-

grams are of cases in which an atelectasis was previously demonstrated, and in which bronchiectasis remained after the affected area of lung had re-expanded.(Case 50, p.141; Case 10, p.142) The other bronchograms are from the following four cases in which it cannot be proved that the bronchiectasis originated in the same manner, as it was apparent from the histories that the disease had been present for some years. A most interesting feature of these latter four cases, however, was that the bronchiectasis almost certainly developed during the course of whooping cough.

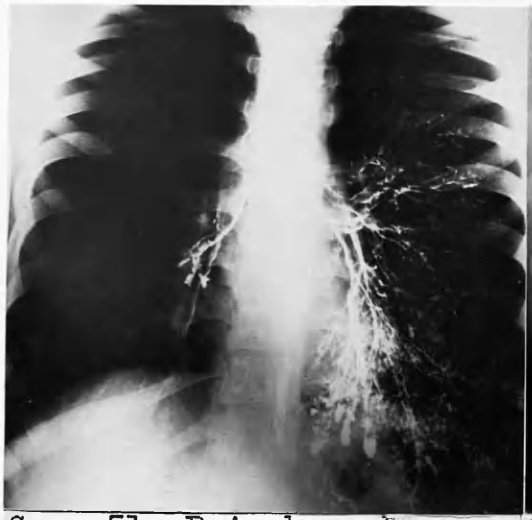
Great reluctance was always felt during the present investigations in attaching any weight to a history tending to impute the blame for bronchiectasis to a previous illness, since the impressions of patients and their relations are often extremely unreliable. For the most part, therefore, an account merely of having had measles, whooping cough, or other ailment was regarded as being of no real significance. After all, patients suffering from gall stones or pernicious anaemia probably at one time or another have had the same childhood diseases.

The evidence in the four cases under discussion, was, however, so striking and clear cut, that little doubt can be entertained as to its validity.

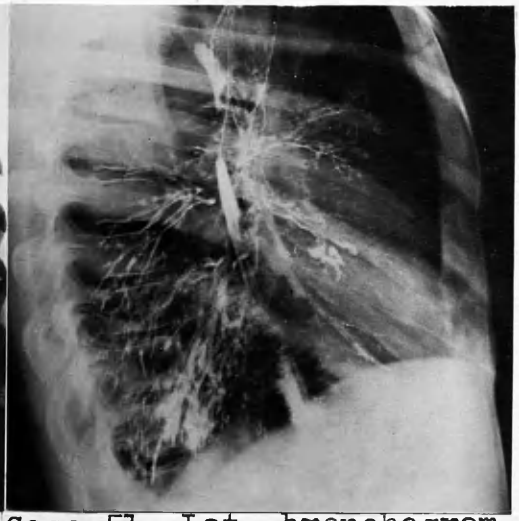
Case 51.

Male, aged 7 years, admitted to Ruchill Hospital on 7/8/47 with "pneumonia" of the left lower lobe. Examination revealed slight impairment of P.N. at the left base, and the R.M. in the same area, though not bronchial, had a tubular element with prolongation of expiration. Crepitations could also be heard at the left base. The temperature was 102. F. on admission, but settled after three days. There was a productive cough.

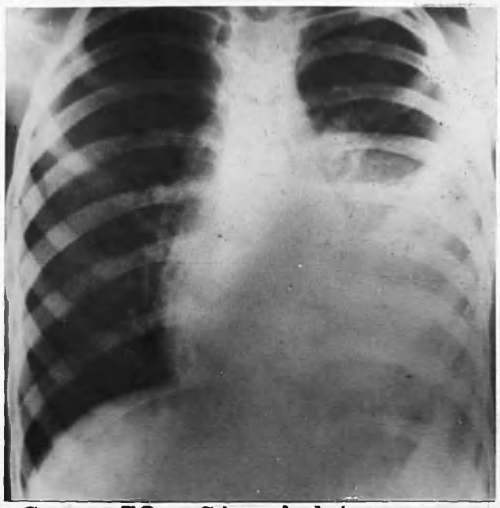
The patient's mother requested to see the ward doctor, as she was "worried about the boy's chest." She stated that the boy had had a persistent cough ever since he had



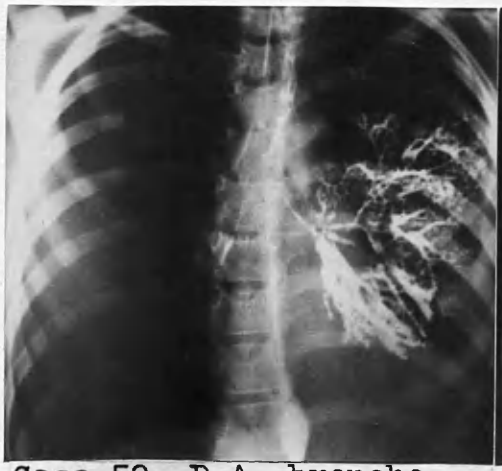
Case 51. P.A. bronchogram. Bronchiectasis left lower lobe.



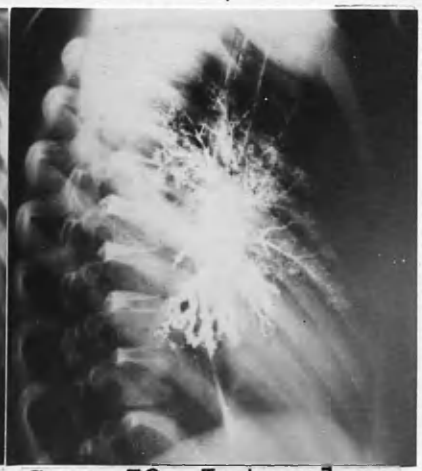
Case 51. Lat. bronchogram. Bronchiectasis left lower lobe. There is some radio-opaque material in the oesophagus.



Case 52. Straight x-ray, 14/8/47. Left pleural effusion.



Case 52. P.A. bronchogram, 9/9/47. Bronchiectasis affecting left lower lobe and lingula.



Case 52. Lateral bronchogram, 9/9/47. Bronchiectasis affecting left lower lobe and lingula.

whooping cough at the age of three years. She had taken him at various periods to her family doctor and several other physicians, including the School Medical Officer, but they had told her there was nothing to worry about. However, since the child had had five attacks of "pneumonia" in the three years preceding admission to hospital, she was still not satisfied that all was well.

The history, coupled with the fact that the productive cough and the abnormal physical signs at the left base did not clear up, pointed almost conclusively to bronchiectasis of the left lower lobe. The diagnosis was confirmed by bronchography on 26/8/47. Bronchograms are shown opposite.

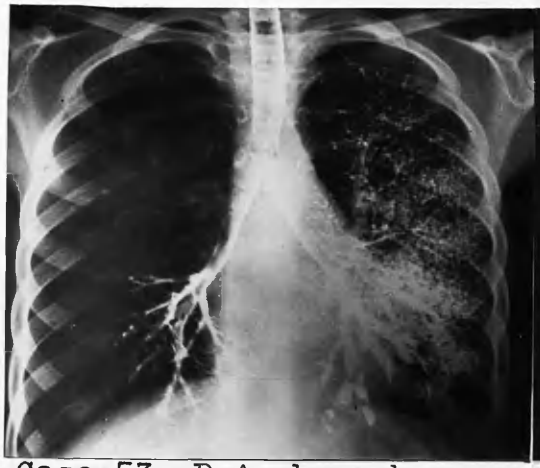
Case 52.

Female, aged 9 years, admitted to Ruchill Hospital on 6/8/47 with "pneumonia." The temperature was elevated to 103. F., and there was a productive cough. Physical examination pointed to a pleural effusion at the left base, with underlying pneumonia. 60 c.c. of thin but opaque fluid were aspirated; the cells were mainly polymorphonuclears, but there was no growth on culture. On examination of the blood, it was found that there was a polymorphonuclear leucocytosis of 13,000 per c.c.. The sputum was repeatedly negative for the tubercle bacillus. X-ray on 14/8/47 revealed that there was still fluid in the left pleural cavity. Absorption had taken place by 1/9/47, and physical examination then revealed impaired P.N., and a rather tubular R.M. with prolonged expiration at the left base. There were crepitations in the same area. A radiogram demonstrated pleural thickening over the left lung, and areas of consolidation in the left lower lobe. The temperature, which had dropped to normal a few days after admission in response to sulphadiazine therapy, remained settled.

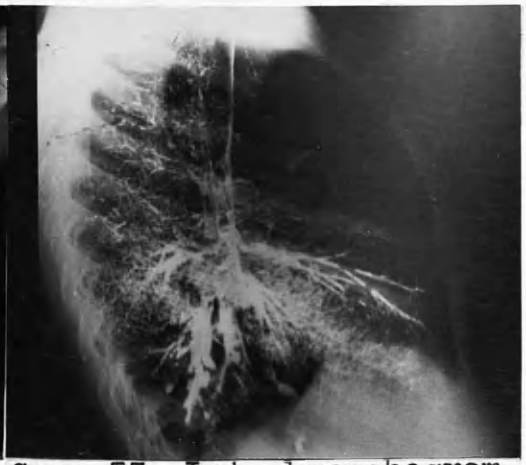
The mother was asked how the child had been faring before admission to hospital. She stated that the girl had had a persistent cough ever since contracting whooping cough at the age of five years. She had also had three attacks of "pneumonia". In view of this history, and the suspicious findings at the left base, bronchiectasis of the left lower lobe was diagnosed.

Bronchography on 9/9/47 revealed bronchiectasis of the left lower lobe and lingula. The patient was transferred to Mearnskirk Hospital where the affected lobes were excised two months later.

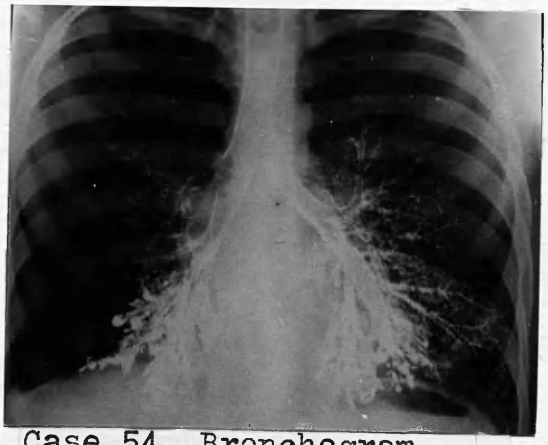
A radiogram and bronchograms are shown opposite.



Case 53. P.A. bronchogram. Bronchiectasis affecting left lower lobe.



Case 53. Lat. bronchogram. Bronchiectasis affecting left lower lobe.



Case 54. Bronchogram, 13/6/47. Bronchiectasis affecting both lower lobes. Lateral views would be necessary to exclude possible involvement of rt. middle lobe and lingula.

Case 53.

Male, aged 8 years, sent from a tuberculosis clinic to Ruchill Sanatorium as an out-patient for bronchography on 12/6/47 on account of a persistent cough and physical signs of bronchiectasis at the left base.

Examination confirmed the physical findings; crepitations could be heard over the left lower lobe, and expiration at the left base was prolonged and rather blowing.

The mother, who accompanied the child, was asked how long he had been troubled with his chest. She replied that he had had a persistent cough ever since he had whooping cough, about the age of four years. He had also suffered from repeated attacks of "pneumonia."

Bronchography demonstrated bronchiectasis of the left lower lobe.

The bronchograms are reproduced opposite.

Case 54.

Male, aged 12 years, sent from a tuberculosis clinic to Ruchill Sanatorium as an out-patient for bronchography on 13/6/47 on account of a persistent and markedly productive cough, and clinical evidence pointing to bilateral basal bronchiectasis.

On examination, numerous medium crepitations could be heard over both lower lobes, and expiration was prolonged and rather tubular at both bases. The boy was undersized, and his general condition poor, though like all the other children with bronchiectasis seen during the investigations, there was no clubbing of the fingers.

His mother volunteered the information that he had had a persistent cough ever since he had contracted whooping cough at the age of three years, and for the last year or more he had been "howkin' up an awfu' lot of spit." The patient coughed up a fair amount of sputum during the administration of local anaesthetic in preparation for bronchography, and copious quantities after the operation was over.

The bronchogram demonstrated extensive bronchiectasis of both lower lobes, which were visualised at the same time for convenience.

Bronchograms are shown opposite.

Without being unduly rash, it may be assumed that the bronchiectasis was, in the above four cases, produced during an attack of whooping cough. In the whooping cough series which was studied in the course of the present

investigations, it has already been pointed out that bronchiectasis was only seen in atelectatic portions of lung. It has also been mentioned that for one reason or another, bronchograms were carried out in twenty five of the cases in which atelectasis had never been detected or in which atelectasis of negligible extent had occurred and speedily cleared up, and in none of them was bronchiectasis noted, nor were any symptoms of the disease such as persistent cough present when the patients left the hospital, or during the subsequent observation. This suggests that in whooping cough at least, bronchiectasis only arises as a consequence of atelectasis. The chances are, therefore, that when the disease originated in the four cases in question, atelectasis was present, and subsequently disappeared.

It is freely admitted that this postulate is mere deduction, and far from being satisfactory proof, but when bronchiectasis has been present for years, months, even weeks, estimates of the originating causes must of necessity be conjectural.

The longer the period which elapses between a study of the disease and its beginning, the more tenuous become the evidential links connecting the established condition with the prime causative factors. In the first two of the previous six cases it is known that bronchiectasis was preceded by atelectasis. In the next four cases, the evidence is weaker; there is good reason to suppose that the disease originated during the course of whooping cough, a malady in which atelectasis has been shown to be frequent.

Suppose that none of the cases had been seen until a further period of years had elapsed: what might the

position have been then?

If similar examples are any criterion, spread of the disease to other lobes, clubbing of the fingers, foul sputum, and advancing cavitation might be expected. (The last case shown, incidentally, was in a fair way to reaching this stage.). Doubtless pulmonary fibrosis and patchy broncho-pneumonia would be radiologically in evidence. The last four patients might well have been unaware by that time that they ever had whooping cough at all.

The time for pathological study is now drawing near, and if ever carried out, examination would enable the confident statement to be made that there was little evidence of atelectasis, but overwhelming evidence of inflammatory processes in the shape of destruction of the bronchial wall, parenchymal scarring, and so on.

It is for this reason that it is believed that pathological studies on bronchiectasis are apt to be misleading, if inferences are drawn from the findings relative to the causation of the disease. Supervening and increasing infection alters the picture, and obscures evidence of the earliest changes.

The best method of studying the origins of the disease is believed to be the method employed in the whooping cough series, that is to say, endeavouring to pick up the malady at its outset in conditions with which it is known to be associated, and tracing the subsequent course.

Even when bronchiectasis has long been established, however, there are often phenomena which indicate that at one time atelectasis was present, and there are many features of the disease which can only be satisfactorily accounted for on that basis.

Indirect evidence which tends to show that atelectasis is the main factor in the causation of bronchiectasis will be discussed under the following headings.

1. Pathological features.
2. Cylindrical bronchiectasis.
3. Mode of progression of bronchiectasis.
4. Incidence of atelectasis in diseases associated with bronchiectasis.
5. Incidence of atelectasis and bronchiectasis in chronic bronchitis.
6. Association of bronchiectasis with nasal sinusitis.
7. Lobar incidence of atelectasis and bronchiectasis.

1. Pathological features.

The pathological findings have been shown to be extremely complex and confusing, but if it is accepted that bronchiectasis originates in a collapsed portion of lung which may later re-expand, all the pathological observations can easily be reconciled, and indeed are precisely what would be expected.

Ogilvie,[18] it will be remembered, divided his specimens into two groups - a larger one in which atelectasis was the predominant feature, and a smaller one in which evidence of infection was the leading finding.

The "atelectatic group" he believed was due primarily to abnormal stresses and strains on the bronchial walls, and the "broncho-pneumonic" group to infective processes. It may be noted, however, that the line of demarcation was extremely arbitrary, for he pointed out that in the first group there was often marked evidence of infection, and in the second group often patchy atelectasis.

There is no difficulty in accepting the postulate that in the first group the causative atelectasis had remained, and the bronchial tree and parenchyma had been more or less damaged by supervening infection.

It is submitted that in the second group there had been atelectasis of the affected lobe with a supervening infection which converted the original compensatory bronchial dilatation into permanent bronchiectasis. It is further suggested that the atelectasis eventually cleared up, but the bronchiectasis remained, as in the cases which have been demonstrated. Extension of the infective processes either continuously, or as is so commonly observed in bronchiectatic subjects, by repeated exacerbations, would account for the varying degrees of damage found in the bronchi and parenchyma. It may be objected that however many considerations there may be in favour of this theory, there is nothing in the pathology of this group of cases which can be construed as direct evidence in its favour.

On the contrary, even although little or no atelectasis was noted when the cases were examined, there was one important finding which strongly suggests that it had occurred at some previous time; this was the observation that emphysema was present in the affected areas in all the specimens just as it was in the "atelectatic" group.

Ogilvie[18] believed that the association of emphysema with atelectasis and bronchiectasis had not hitherto been stressed in the literature. He was almost, but not quite, correct, as the following quotation from an article which Andrus[35] wrote four years before Ogilvie's publication, shows.

"The association of emphysema with bronchiectasis and

atelectasis is referred to by a number of authors, but as above noted the relationship is not customarily stressed. One of these(Hewlett) states, "Bronchiectasis also arises in connection with emphysema, and is merely an extension of that condition," and another (Green), "The causation of bronchiectasis is in great measure analogous to that of emphysema." With these statements, we are in full agreement. Thus although bronchiectasis means only dilatation of the bronchi, bronchiectatic disease as it actually occurs, is so frequently a combination of bronchial dilatation with severe air-cell dilatation of common origin, that we propose that the term"Pulmonectasis" be used as more accurately descriptive of the basis of this type of disease."

It has already been seen that all observers of atelectasis have pointed out that compensatory over-distension of the alveoli is a constant concomitant of the condition, and that it may lead to emphysema.

The localised emphysema constantly noted in Ogilvie[18]'s group of "broncho-pneumonic" cases could therefore be simply explained on the basis of a previous atelectasis, and there does not appear to be any other satisfactory way of accounting for it.

Another important pathological finding is that in many instances there is gross dilatation of the bronchi with a minimal degree of infection and bronchial damage; the bulk of the cases in Lander[17]'s lobectomy series were of this nature, and some examples were also noted by Ogilvie[18].

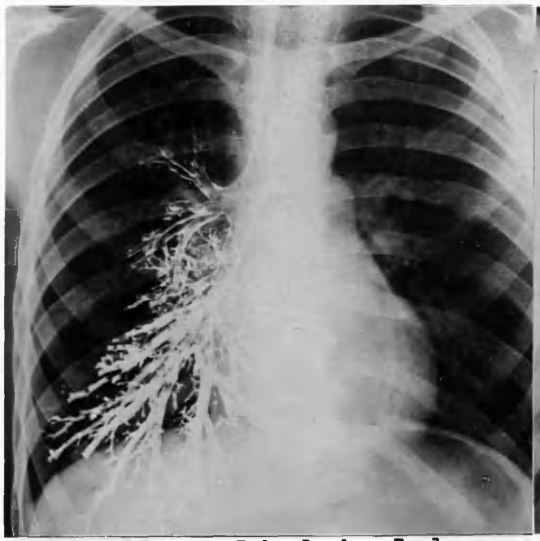
If an initial atelectasis is postulated, a feasible explanation at once presents itself, for when the bronchi are mechanically dilated for some weeks or months, no doubt a comparatively minor degree of infection might cause them

to lose their elasticity to a degree sufficient to prevent reversion to normal even if the atelectasis cleared up. Indeed, as has been suggested, it is quite possible that mere prolonged mechanical dilatation may be sufficient to produce permanent bronchiectasis. At any rate, it appears to be impossible to account for the marked dilatation of almost undamaged bronchi in any other way.

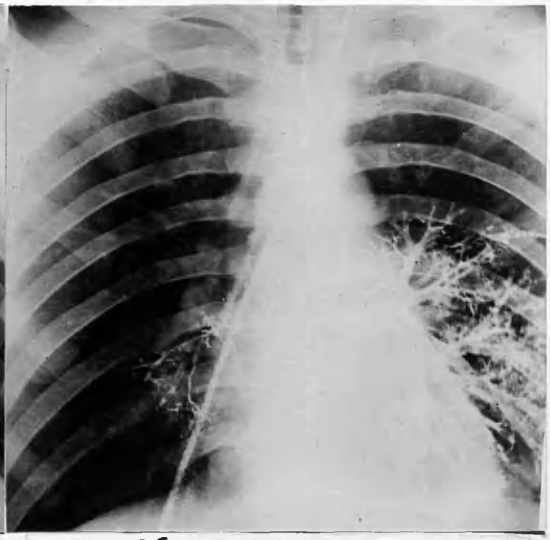
Certainly it cannot be explained on the basis that bronchiectasis is caused by infection plus the ordinary dilating forces of inspiration. It has already been pointed out that from a consideration of the dynamics of respiration, it is extremely unlikely that dilatation would be induced even if the elements of the bronchial walls were largely replaced by fibrous tissue, and where they are almost intact, such an event appears even more improbable. In any case, chronic bronchitis is a disease which enables the theory to be put to the test, since here there are chronically inflamed bronchi subjected to the normal dilating forces of respiration for years. As has already been stated, however, the careful and prolonged observation of 2,500 cases reported by Andrus[35] showed that bronchiectasis did not occur in uncomplicated cases. This appears to prove conclusively that moderate bronchial damage at least is not normally sufficient to give rise to bronchiectasis.

Once more, therefore, the atelectatic theory is the only one which can satisfactorily explain the pathological findings.

Incidentally, Andrus[35] noted that bronchiectasis developed in a few cases of chronic bronchitis in which the disease was complicated by an attack of "penumonia." It is suggested that the "pneumonia" in these instances was,



Case 50. Cylindrical bronchiectasis of rt. middle and rt. lower lobes.



Case 26. Cylindrical bronchiectasis of left lower lobe. Another photograph of the bronchogram reproduced here in which the exposure used has better displayed the dilated bronchi behind the heart, is shown opposite p.68.

in actual fact, an infected atelectasis.

2. Cylindrical bronchiectasis.

Two examples of cylindrical bronchiectasis are shown opposite. A consideration of the regular character of the dilatations prompts the inference that they were produced by the uniform application of a distending force to the affected areas, and the occurrence of an atelectasis at some time would appear to offer the only satisfactory explanation as to how it could have been effected.

In point of fact, in the first instance a previous atelectasis was demonstrated, and the case has already been discussed (Case 50, p.141.). It is therefore tempting to explain the appearance of the second bronchogram on the same basis, though here nothing is known of the origin of the condition.

The features of the case (Case 26, p.67.), an example of "dry bronchiectasis", have already been outlined. The patient was a healthy man of twenty two years of age, admitted to hospital with a trivial infection of the left lower lobe which speedily cleared up. He had recently been demobilised from a front line unit, and there was no history of previous respiratory trouble. Bronchography was carried out mainly because of suspicious prolongation of expiration at the left base.

If the occurrence and subsequent disappearance of an atelectasis at some time in the past is discounted, what alternative theories can be advanced to explain the cylindrical dilatation of the left lower lobe bronchi?

Fibrosis of the lung in the affected area has already been shown (p.p. 79,80.) to be a postulate totally unacceptable on several grounds, and in any case, there was no

radiological evidence of its existence.

Broncho-pulmonary necrosis, already considered on pages 84 to 94, is another theory purporting to explain the causation of bronchiectasis. The general condition of the patient, and the disappearance of cough and sputum production a few days after admission, puts this out of the question. Besides, pulmonary cavities would not remain cylindrical; they would become spherical because of the elasticity of the lung tissue, as has already been pointed out.

The only explanation left is the theory that at one time, the bronchi of the lobe were uniformly "weakened" by some agency, presumably infection, and were dilated by the normal forces of respiration. This possibility has already been discussed, (p.p. 84-94.) and all that need be said here is that direct evidence in its favour has so far never been produced, and the balance of indirect evidence is strongly against it.

The only feasible explanation for cylindrical bronchiectasis, therefore, appears to be that it is brought about by the forces arising when pulmonary collapse takes place.

3. Mode of progression of bronchiectasis.

In the past it was generally supposed that spread occurred by the "spill over" of secretions. It is, however, common knowledge that the disease is often limited to one lobe for very prolonged periods, and extension to other lobes, if it does occur, is sudden. This mode of progression by periods of apparent confinement and abrupt advance can best be explained by postulating further atelectases from aspiration of secretions.

4. Incidence of atelectasis in diseases associated with bronchiectasis.

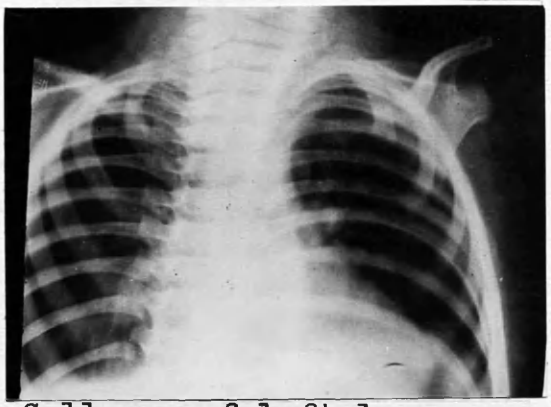
Various diseases have been accused of giving rise to bronchiectasis, but the ones most commonly implicated are the pneumonias, acute bronchitis, whooping cough, and measles. Infection is certainly present in all these conditions, but there is a wide variety in the nature of the causative organisms, and it would be difficult to explain why they should all, on occasion, give rise to bronchial dilatation localised to certain lobes or bronchopulmonary segments. Moreover, Lander and Davidson's[17] experiments on cats showed that, in the absence of infection, the aspiration of viscid material into the peripheral bronchioles brought about atelectatic bronchiectasis.

As sputum production is a feature common to all the diseases mentioned above, the mechanism for causing atelectasis is available, and if the occurrence of collapse could be shown to be a not infrequent event in these illnesses, an explanation for the association of bronchiectasis with these conditions would be at hand.

Whooping cough was the only one of these diseases systematically studied in this work, but a few comments will be passed on the others also.

Erwin[58] stated in an article in 1939, "In whooping cough... the tenacious nature of the sputum bespeaks collapse."

During the present investigations this theoretical expectation was seen to be fully borne out in practice, as atelectasis occurred in no less than forty three per cent of the one hundred and fifty cases examined. In most instances, the collapse was small in extent and comparatively ephemeral in character, but in eight cases in which



Collapse of left lower lobe. The triangular shadow due to the atelectasis can be seen lying in the heart shadow. The condition occurred in a patient suffering from polio-encephalitis.

the atelectasis was extensive and persistent, bronchial dilatation was demonstrated in the collapsed areas. The greater the extent of collapse, the greater was the bronchial dilatation, and in four examples the latter condition was shown to be well marked. In all of the cases, the affected part of the lung eventually re-expanded, though in one instance only after eighteen weeks. Reversion of the bronchi to normal was demonstrated as an event coincident with the disappearance of the atelectasis.

Further, bronchography was carried out for various reasons in twenty five patients in whom no atelectasis could be demonstrated, and in these cases bronchial dilatation was never seen. This series served to some extent as a control, and is supporting evidence that in whooping cough atelectasis is the only factor which gives rise to bronchial dilatation.

Broncho-pneumonia was demonstrated clinically and radiologically in thirty cases. An analysis in chapter 2 shows, however, that atelectasis did not occur more frequently in this group than in the group without broncho-pneumonia. This indicates that the atelectasis was caused by aspiration of sputum into the bronchioles or finer bronchi, and not by bronchiolar occlusion due to localised infective processes. This theory is supported by the fact that Runciman[113] detected atelectasis of the left lower lobe in a case of bulbar paralysis occurring in the course of polio-encephalitis; he has kindly given the author permission to reproduce the relevant radiogram which appears opposite this page. As there was no evidence of pulmonary infection, the atelectasis was almost certainly due to aspiration of sputum accumulating in the respiratory passages as a result of

the bulbar paralysis.

Bronchial dilatation did not occur in any of the thirty cases of broncho-pneumonia except in an atelectatic area.

The results of the whooping cough investigation, even remembering the comparatively small number of cases, appears to show fairly conclusively that in this disease at least, bronchiectasis only arises as a result of atelectasis, and although infection is doubtless a potent factor in determining whether the condition will be permanent, it cannot by itself give rise to it.

It is submitted that the evidence is so strong in favour of this contention, that it could only be rebutted by demonstrating in a whooping cough study of comparably close radiological control, the onset of bronchiectasis without associated atelectasis.

It is believed that no example of such an occurrence, whether in whooping cough or any other disease, has ever appeared in the literature. It is emphasised that radiological examination, to be of value, must be frequent. It would be of no significance to show a normal lung, and then bronchiectasis without atelectasis after even a few weeks' interval; the atelectasis might have cleared up.

The approximate incidence of atelectasis in the other diseases commonly implicated in the genesis of bronchiectasis, is apparently so far unknown, as, judging from the literature, investigations on the lines on which the whooping cough series was carried out are rare; no similar study was noted.

However, in the pneumonias at least, the work of Coryllos and Birnbaum[91], Coryllos[92,114,115], Sells[76], Warner[81], Wearing[49] and others indicates that atelectasis cannot be uncommon.

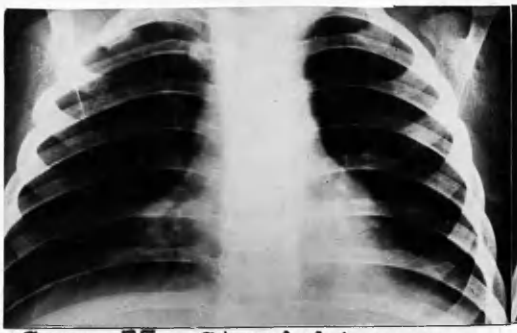
Wearing[49] states that patchy atelectasis is a common feature of all forms of pneumonia other than lobar pneumonia. With regard to lobar pneumonia, he points out that according to Hadfield and Garrod[116] when a person has been sensitised to the pneumococcus, a further infection leads to an allergic reaction in which fluid is poured into the pulmonary alveoli, and consequently collapse cannot take place. Nevertheless, during the process of resolution, there is no reason why aspiration of sputum should not lead to atelectasis.

Many so-called "atypical pneumonias" are, of course, infected aspiration atelectases. The cases of Blades and Dugan[108] which have been discussed, clearly fall into this category.

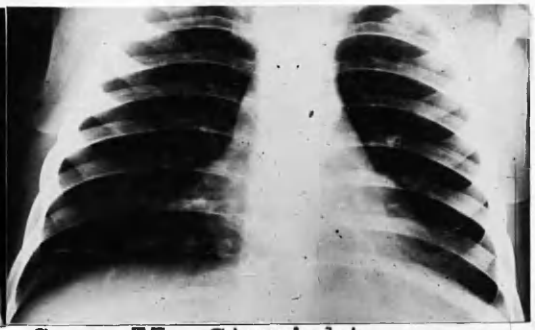
There is also no doubt at all that in many instances, an "unresolved pneumonia" is really an atelectasis, and it is significant in that regard that Coryllos[114] promoted resolution in some cases by bronchoscopic aspiration of plugs of mucus from the peripheral bronchi.

No attempt was made in the present study to estimate systematically the frequency of atelectasis in the diseases other than whooping cough under discussion, as the radiological department of the hospital could not have coped with the work; but the examination of routine x-rays of patients suffering from broncho-pneumonia, acute bronchitis, and measles showed that atelectasis was by no means uncommon in these conditions.

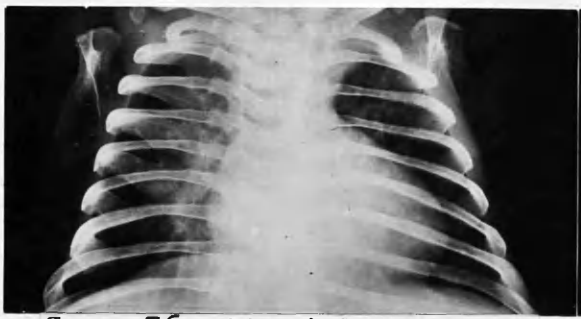
Only general impressions were gained, but it appeared that atelectasis was a much more frequent complication of pneumonia in young children than it was in adults, probably because in children the broncho-pneumonic form is more common, and also because in children the bronchial calibre is narrower and hence more likely to be obstructed by



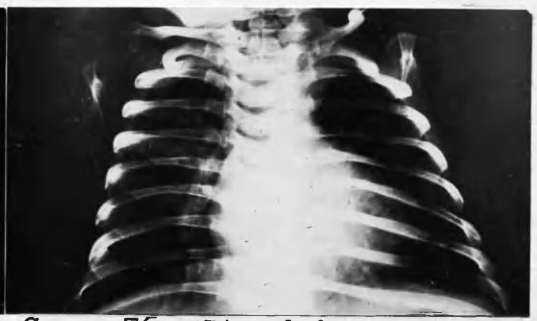
Case 55. Straight x-ray, 4/11/47. Atelectasis affecting rt. middle and rt. lower lobes.



Case 55. Straight x-ray, 8/12/47. The atelectasis has disappeared apart from a small area in the rt. middle lobe.



Case 56. Straight x-ray, 4/11/47. Atelectasis rt. upper lobe. (Not well reproduced).



Case 56. Straight x-ray, 12/12/47. Re-expansion of collapsed area.

sputum. Atelectasis seemed to be less common in measles than in whooping cough; perhaps this is because the sputum is less viscid in the former disease.

A few examples of atelectasis occurring in broncho-pneumonia, acute bronchitis, and measles follow.

Broncho-pneumonia.

Case 55.

Female, aged 2 years, admitted to Ruchill Hospital on 15/10/47 with broncho-pneumonia. Examination revealed crepitations in the lower part of both lungs, but the percussion note was not impaired. The R.M. was broncho-vesicular at the bases. On admission, the temperature was 103. F. but settled in a few days in response to sulphadiazine. After a week, the patient appeared well, but a radiogram on 4/11/47 demonstrated an area of atelectasis in the right middle and lower lobes. Clinically, there was perhaps some impairment of P.N. at the right base and prolongation of expiration, but the condition would probably have been missed but for the x-ray.

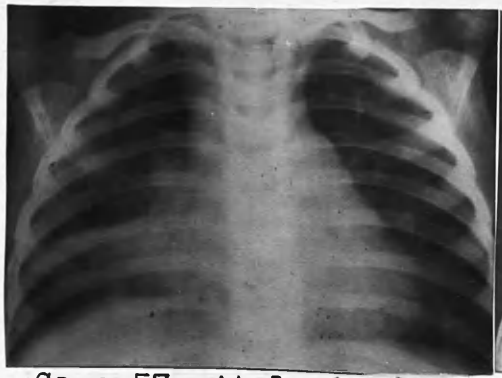
By 8/12/47, the atelectasis had practically disappeared. The child was dismissed, and an x-ray a month later revealed no abnormality.

The radiograms are shown opposite.

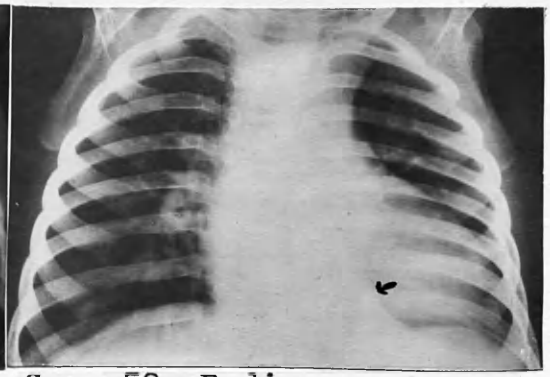
Case 56.

Male, aged 10 months, admitted to Ruchill Hospital with broncho-pneumonia on 17/10/47. Examination revealed scattered crepitations in both lungs. The P.N. was not appreciably impaired and the R.M. harsh and vesicular. The temperature was elevated on admission to 102 F., but after the administration of sulphadiazine, settled on 22/10/47, and did not rise again. On 1/11/47, it was noted that the P.N. was impaired over the right upper lobe, and the R.M. in that area differed from the other side, in that it was louder and more tubular. A radiogram on 4/11/47 showed collapse of the right upper lobe. Re-expansion was almost complete by 12/12/47, and the parents were requested to take the child home and bring him back as an out-patient.

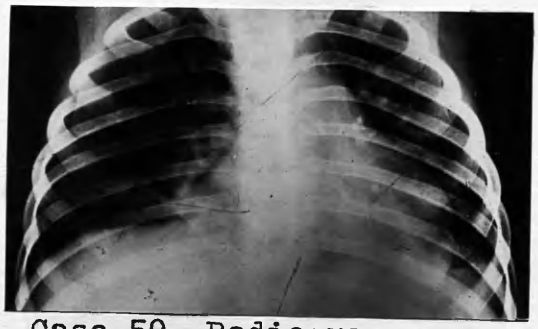
It is difficult to say in this case, in the absence of bronchography, whether the collapse was due to medial obstruction of the upper lobe bronchus, possibly due to glandular pressure or whether it was the "peripheral" type of collapse under discussion. It is true that the R.M. could be heard over the affected lobe which would



Case 57. Atelectasis affecting rt. lower lobe and probably also rt. middle lobe.



Case 58. Radiogram, 6/4/48. Complete collapse of left lower lobe.



Case 59. Radiogram, 1/10/47. Atelectasis involving rt. lower lobe.

point to the latter alternative, but as it was stronger and not weaker than normal, the sounds were, in all probability, conducted from the trachea by the collapsed tissue, and therefore the presence of R.M. over the affected area was, in this instance, no indication of the patency of the bronchi.

The radiological appearance (not well brought out on the photograph) and the absence of an elevated temperature, appeared to exclude pneumonic consolidation.

In view of the tender age of the patient, the good general condition, negative Mantoux, normal temperature, and the comparative rapidity with which the atelectasis cleared up, aspiration of sputum appeared to be the likeliest causative factor.

The radiograms are reproduced opposite page 169.

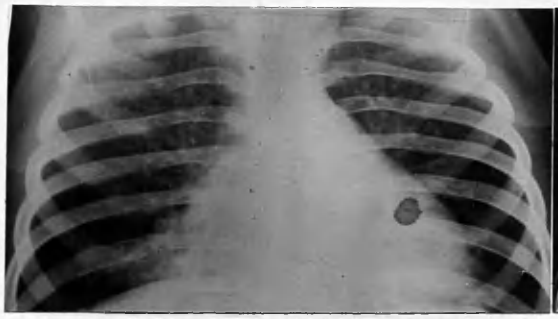
Case 57.

Female, aged 4 years, admitted to Ruchill Hospital on 12/10/47 with broncho-pneumonia. There were numerous crepitations in the lower part of both lungs, but the P.N. was not appreciably affected,and the R.M., while harsh, was not tubular. The temperature was 103 F. on admission, but settled in 5 days in response to the administration of sulphadiazine. On 18/10/47 it was noted that the P.N. was impaired at the right base, and the R.M. was weak and tubular in character. A radiogram on 22/10/47 revealed atelectasis affecting the right lower and also apparently the right middle lobe, though a lateral view was not taken. The author did not see this patient until a month later by which time the atelectasis had cleared up. Bronchography revealed a normal bronchial tree.

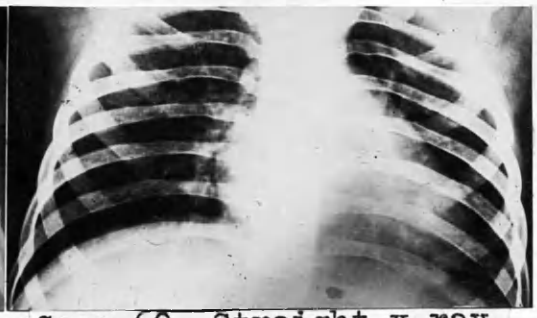
A radiogram is shown opposite.

Case 58.

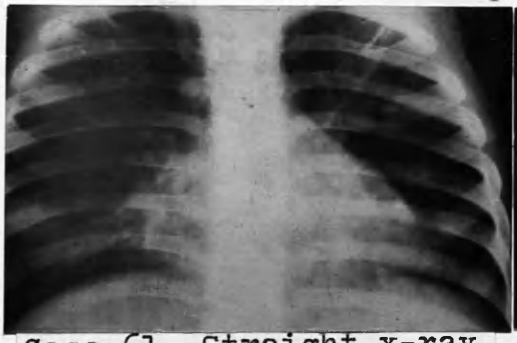
Male, aged 16 months, admitted to Ruchill Hospital on 2/4/48. The temperature was 102 F., and there were numerous medium crepitations at both bases, though the P.N. was not appreciably impaired. Sulphadiazine was administered. On 5/4/48 the temperature was 99 F.. Physical examination on this date revealed a very well marked and clearly defined triangular area of dullness at the left base close to the spine. Over the dull area, well marked tubular breathing and bronchophony could be detected. The remainder of the lung fields was clear. The mediastinum had moved to the left. Apart from the fact that the R.M. was rather loud tubular than weak tubular in character, the signs were the classical ones of complete collapse of the left lower lobe. The diagnosis was confirmed by a radiogram on



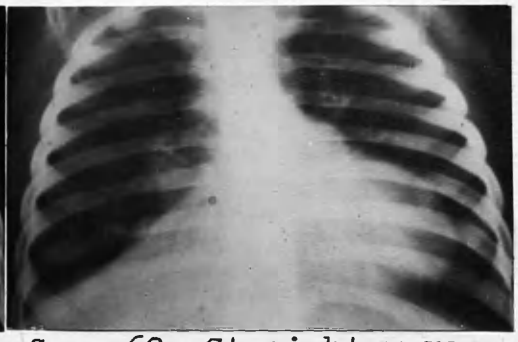
Case 60. Straight x-ray, 21/11/47. Atelectasis involving rt. lower lobe.



Case 60. Straight x-ray, 2/12/47. The collapsed portion of lung has re-expanded.



Case 61. Straight x-ray, 27/1/48. Atelectasis affecting rt. middle lobe.



Case 62. Straight x-ray, 7/11/47. Collapse of rt. lower lobe.

6/4/48. Percussion postular drainage was instituted, but at the time of writing (June 1948) the collapse has not cleared up.

A radiogram is shown opposite page 170.

Acute Bronchitis.

Case 59.

Male, aged 2 years, admitted to Ruchill Hospital on 25/9/47. No history of whooping cough or other respiratory infection. The temperature was 100 F. on admission, and was normal the next day. Examination revealed rhonchi and a few scattered crepitations in both lung fields. The P.N. was not impaired, and the R.M. was normal. Routine x-ray on 1/10/47 demonstrated an area of atelectasis in the right lower lobe. Save for a few crepitations at the right base there was little to be discovered clinically. The condition cleared up in a fortnight.

A radiogram is shown opposite page 170.

Measles.

Case 60.

Male, aged 18 months, admitted to Ruchill Hospital with measles on 11/11/47. Examination revealed numerous crepitations at both bases, and impaired P.N. at the right base. The left base had cleared by 14/11/47, but there were still a few crepitations in the right lower lobe, and, close to the spine, expiration appeared to be prolonged. X-ray on 21/11/47, revealed a slight degree of atelectasis of the right lower lobe. The condition had cleared up clinically and radiologically by 2/12/47.

Radiograms are reproduced opposite.

Case 61.

Female, aged 14 months, admitted to Ruchill Hospital with measles on 7/1/48. Examination revealed scattered crepitations at both bases. The crepitations persisted at the right base, and the child was x-rayed on 27/1/48. A degree of atelectasis of the right middle lobe was noted. The condition cleared up in a fortnight.

A radiogram is shown opposite.

Case 62.

Female, aged 3 years, admitted to Ruchill Hospital on 2/11/47. There were scattered crepitations in both lungs, and the P.N. was impaired at the right base. The R.M. in the same area was weak, and tubular in character. X-ray on 7/11/47 revealed atelectasis at the right base. The

condition cleared up clinically and radiologically in a month. Bronchography was not carried out.

A radiogram is shown opposite page 171.

Tuberculosis is not much discussed as a disease giving rise to bronchiectasis, because here bronchiectasis is over-shadowed by the gravity of the malady of which it is a complication.

Erwin,[58] however, in 1939, in a fascinating article, demonstrated that bronchiectasis is of fairly frequent occurrence, and showed that here also atelectasis is the inducing cause. He considered it beyond doubt that in the vast majority of cases the atelectasis was caused by aspiration of sputum into the bronchioles or finer bronchi.

Postulating an analogous mechanism, he was able to put forward an interesting and convincing explanation of "total excavation of a lobe or lung" in tuberculosis. He suggested that if a cavity were present in an atelectatic lobe, its exit might, at first, be blocked by one of the plugs of sputum. The air in the cavity would commence to be absorbed, and the gas pressure in the cavity would fall. The plug of mucus would thus be likely to be sucked into the cavity. When this happened, the cavity would be in communication with the atmospheric air, and being in an atelectatic area, would suddenly expand, like the patent bronchi, as a compensatory measure. In this way, a sudden "excavation" of the lobe could be produced. This observation tends to confirm the correctness of the principle underlying the atelectatic theory of the origin of bronchiectasis.

As well as demonstrating that bronchiectasis was a constant accompaniment of atelectasis in tuberculosis where the bronchi in the affected area were patent, Erwin[58] also performed bronchographies on non-atelectatic cases in

which fibrosis was extensive. He found that distortion of the bronchi, not bronchiectasis, was present.

5. Incidence of atelectasis and bronchiectasis in chronic bronchitis.

Since sputum is abundant in this disease, one would have expected that atelectasis would be a common complication, yet surprisingly enough, according to Erwin[58], Andrus[35], and others, this is not the case.

Erwin[56] attributes the rarity of its occurrence to the loose character of the sputum, and states that when it does take place, it is generally seen in the more acute phases of the disease when the sputum is more viscid.

It has already been mentioned that the impression was gained in the course of this study, that atelectasis in pneumonia appeared to be much commoner in children than in adults, and that possibly the larger calibre of the bronchi in adults made obstruction by sputum less likely.

If this is so, since chronic bronchitis is a disease of the older age groups, this factor would militate against the frequent occurrence of atelectasis in the disease.

It has already been mentioned that Andrus[35] found bronchiectasis was also rare in chronic bronchitis.

This is in keeping with the atelectatic theory of the genesis of bronchiectasis. If, on the other hand, it is suggested that the condition is caused by infection weakening the walls of the bronchial tubes, the rarity of the condition in chronic bronchitis is surprising.

6. Association of bronchiectasis with nasal sinusitis.

Much interesting work has been done on the relation of infection of the nasal sinuses to bronchiectasis, and

perhaps, on the whole, it may be said that an association has been established. The proviso must be made at once that it cannot be the prime cause of the condition as some authors have suggested, since bronchiectasis occurs in many cases in which the sinuses are normal.

A short review of the literature on the subject follows.

In 1916, Sargent[117] pointed out the connection between infections of the upper respiratory tract and those of the lower, and Mullin[118] later showed that infection could spread from the naso-pharynx and sinuses to the bronchi of rabbits by direct aspiration, or by the lymphatics and blood stream.

Quinn and Meyer[119] performed the striking experiment of introducing iodised poppyseed oil into the nasal passages during sleep, and showing its presence in the bronchi the next morning by radiological examination. McLaurin[120] confirmed this finding, and also demonstrated that iodised oil could similarly be aspirated from the nasal sinuses into the bronchial tree.

A mechanism of bronchial infection has therefore clearly been established.

Many careful investigations of the sinuses of bronchiectatic patients have been carried out, and the results of several of these studies will now be given in tabular form.

	Cases of Bronchiectasis.	No. with Infected sinuses.	%
Quinn and Meyer.[119](1929)	38	22	58
Clerf.[121](1934)	200	164	82
Hodge.[122](1935)	37	28	75
Boyd.(1935)[123]	12	10	83
Goodale.(1938)[124]	75	46	61
Walsh and Meyer.[125](1938)	217	145	67
Chipman and Collins.[126](1939)	58	26	45
	637	441	69%

In 129 gastric cases used as a control series, Martin and Berridge[48] found 56 cases had infected sinuses i.e. 43.4%.

Even making due allowance for the facility of the ear, nose and throat specialist in discovering sinus infection, the enthusiasm of the advocates of a theory, the prevalence of sinus infection and other factors, this evidence, if it does not prove there is a connection between nasal sinusitis and bronchiectasis is at least strongly suggestive.

The problem arises once more, however, as to the manner in which bronchiectasis arises once the infection is introduced into the bronchial tree.

Enough has already been said in the course of this article to indicate that atelectasis is a fairly common phenomenon. It has been shown, also, that even after months it may disappear without giving rise to any apparent inconvenience or permanent after effects.

It is submitted, however, that should a patient in whom an atelectasis is present, also suffer from sinus infection, the chances of permanent bronchiectasis developing will be greatly increased, since infected material will constantly be entering the dilated bronchi in the atelectatic area. Under those circumstances, even should re - expansion of the lung subsequently take place, it obviously need not lead to a disappearance of the bronchial dilatation.

This theory would explain why the prevalent, and usually comparatively innocuous, chronic sinus infection may be associated with gross localised bronchial dilatation.

7. Lobar incidence of atelectasis and bronchiectasis.

In the whooping cough series investigated in this work, an analysis was made of the order of frequency in which the different lobes were affected by atelectasis. The figures will be compared below with the figures for the frequency with which the different lobes were affected by bronchiectasis in Tudor Edwards'[50] series of 166 lobectomies, until recently the largest of its kind appearing in the literature. In March, 1947, Meade[127] published an account of 196 lobectomies, but unfortunately he did not give the necessary details.

Tudor Edwards[50] gives the following details of his lobectomy series:-

"Table 1. Distribution of 166 Lobectomies.

Left lower lobe	78
Left lower lobe and lingula	14
Left upper lobe	7
Lingular process of left upper lobe	1
Right lower lobe	42
Right middle and lower lobes	15
Right upper lobe	4
Right middle lobe	4
Right upper and middle lobes	1"

From this information it is easy to deduce the number of times the various lobes were involved in bronchiectasis except in the case of the lingular process of the left upper lobe, which, since it is the homologue of the right middle lobe has been regarded as a separate lobe in this work. According to the table above, left upper lobe lobectomy was carried out seven times. The lingular process of the left upper lobe may have been bronchiectatic on every occasion, but on the other hand it may not,

and have been removed with the rest of the lobe merely for operative convenience. Accordingly, in the analysis given below of the frequency with which the various lobes were involved in bronchiectasis in Tudor Edwards'[50] series, the number of times the lingula was involved assuming it was affected with the remainder of the left upper lobe on the seven occasions in question will be stated in plain figures, and the number of times the lingula was involved assuming it was not affected on these seven occasions will be stated in brackets.

Lobe.	No. of Times Affected by Bronchiectasis.
Left lower lobe	92
Right lower lobe	57
Right middle lobe	20
Right upper lobe	5
Left upper lobe (excluding lingula)	7
Lingula	22(15)

Details of the lobar incidence of atelectasis in the whooping cough series of one hundred and fifty cases investigated in this work have already been given on pages 27 and 28.

The lobar incidence per cent of atelectasis in the whooping cough series will now be compared with the lobar incidence per cent of bronchiectasis in Tudor Edwards'[50] lobectomy series. The figures for the lobar incidence per cent in Tudor Edwards' series will be given on the assumption that the lingular process of the left upper lobe was involved in bronchiectasis on the seven occasions on which the left upper lobe was removed, and figures calculated on the assumption that the lingula was not involved on these occasions will be given in brackets afterwards.

Lobe	Incidence of atelectasis per cent in wh. cough series.	Incidence of bronchiectasis per cent in T. E.'s lobec. series.
Left lower	50.5	45.3 (46.9)
Right lower	36.5	28.1 (29.1)
Right middle	9.4	9.9 (10.2)
Right upper	2.4	2.5 (2.5)
Left upper	1.2	3.4 (3.6)
Lingula	-	10.8 (7.7)

It will be observed that the correspondence of the figures is amazingly close, and the obvious inference is, of course, that if the lobar incidence of atelectasis in respiratory infections other than whooping cough is approximately the same as in that disease, then the lobar incidence of bronchiectasis could be easily explained on the basis that bronchiectasis is caused by atelectasis.

With regard to the lingula, where there is a considerable discrepancy in the incidence of atelectasis and bronchiectasis, it may be noted that in Tudor Edwards'[50] series the lingula was only once alone affected, and fourteen times affected in combination with the left lower lobe. This would suggest that once bronchiectasis is established in the left lower lobe, subsequent aspiration of infected secretion into the lingular process with consequent atelectasis and bronchiectasis, is not uncommon. The discrepancy could also be explained, if, in the whooping cough series on some occasions, atelectasis of the left lower lobe alone was diagnosed, when actually the lingular process was also involved. This is by no means impossible, as the mistake is easy to make, especially if the extent of atelectasis is small. Lordotic views,

however, were taken in doubtful cases, and it is thought that not many cases could have been overlooked.

It is believed that a comparison between the lobar incidence of atelectasis in one of the diseases commonly associated with the genesis of bronchiectasis and the lobar incidence of bronchiectasis has not hitherto appeared in the literature, but the work of Erwin[58] on atelectasis and bronchiectasis in tuberculosis supports the findings. Here, as is well known, bronchiectasis is seem mainly in the upper lobes, contrary to the usual form of the disease in which the lower lobes, predominantly the left, are affected. Erwin's[58] figures for the incidence of atelectasis were:-

Upper lobes	-	Right, 24; Left, 5.
Lower lobes	-	Right, 6; Left, 2.
Combinations	-	Both upper, 1; Upper and lower, 2; Both middle, 1; Middle and lower, 3.
Total unilateral	-	Right, 20; Left, 21.

He did not employ the method used above of stating the number of times a lobe was affected whether in combination or not, and he does not give enough data to enable this to be done. But the point is clearly made that atelectasis, like bronchiectasis, occurs in pulmonary tuberculosis predominantly in the upper lobes.

What factors are responsible for the selective lobar incidence of atelectasis? It is suggested that the site of sputum production is important, and that the influence of gravity also plays a part.

If sputum is being produced wholly or mainly in one lobe, as is common for example in tuberculosis, then it is likely that it may accumulate in the main bronchus of the lobe, fail to be completely expelled by coughing and succeed in blocking completely either the main bronchus of the lobe or a secondary bronchus of the lobe, and consequent-

ly be "sucked" to the periphery of the bronchial tree in the manner described by Lander.[16] As the sputum of tuberculosis is commonly viscid, and the disease chiefly affects the upper lobes of the lung, the high upper lobe incidence of atelectasis can perhaps best be accounted for by this mechanism. Respiratory infections other than tuberculosis predominantly affect the lower lobes of the lung, and the fact that sputum is produced mainly in these lobes no doubt favours their involvement in atelectasis.

In cases in which sputum has been expelled from the affected part of the lung into the trachea, the influence of gravity no doubt plays a major role in deciding into which lobe sputum will be aspirated. If the subject is standing or sitting up the sputum will tend to be directed into the bronchi supplying the lower lobes, and since the left lower lobe bronchus is more directly in line with the trachea than is the right lower lobe bronchus, the sputum will be more likely to find its way into the left lower lobe than into the right, thus accounting for the higher incidence of left lower lobe atelectasis. Brock[7] has made the important observation that if the subject is lying down, the lobe into which material is likely to gravitate from the trachea depends on whether he is lying on his back or on his side. If he is lying his back, secretion will tend to gravitate into the apical segments of the lower lobes, and also into the posterior part of the sub-apical segments of the upper lobes. If he is lying on his side, secretion will gravitate into the dependent upper lobe bronchus, and not into the lower lobe bronchus which is not dependent in this position. This fact would explain the occasional involvement of an upper lobe in atelectasis even when sputum is being produced in

the lower lobes.

The correspondence of the incidence of atelectasis and bronchiectasis in the different lobes of the lung, is therefore further evidence of the association between the two conditions.

There is another interesting point which arises from a consideration of the above figures. Apart from tuberculosis, atelectasis and bronchiectasis have been seen to affect the lower lobes, predominantly the left. Advocates of the congenital theory of atelectatic bronchiectasis, such as Sauerbruch[63], are at pains to propound ingenious theories of embryological maldevelopment in order to demonstrate that the left lower lobe is likely to be atelectatic at birth, thus explaining its predominant position in bronchiectasis. The figures show that this lobe, in whooping cough at least, is anyhow the commonest seat of atelectasis in post natal life, and thus speculation on esoteric congenital defects seems scarcely necessary, particularly as in addition is is now known that atelectasis can take place in the early weeks of life from aspirated sputum. Many of these cases have undoubtedly been classed as congenital by observers adhering to Sauerbruch's theory. Examples of atelectasis occurring in children a few months old, were encountered in the whooping cough investigations, as the figures in the second chapter indicate.

Conclusions.

A consideration of the evidence put forward in this chapter leads to the following conclusions:-

1. When atelectasis of a lobe or lung occurs, a mechanical dilatation of the contained bronchi always results, providing that the factor causing the collapse is obstruction of the bronchioles or finer bronchi.

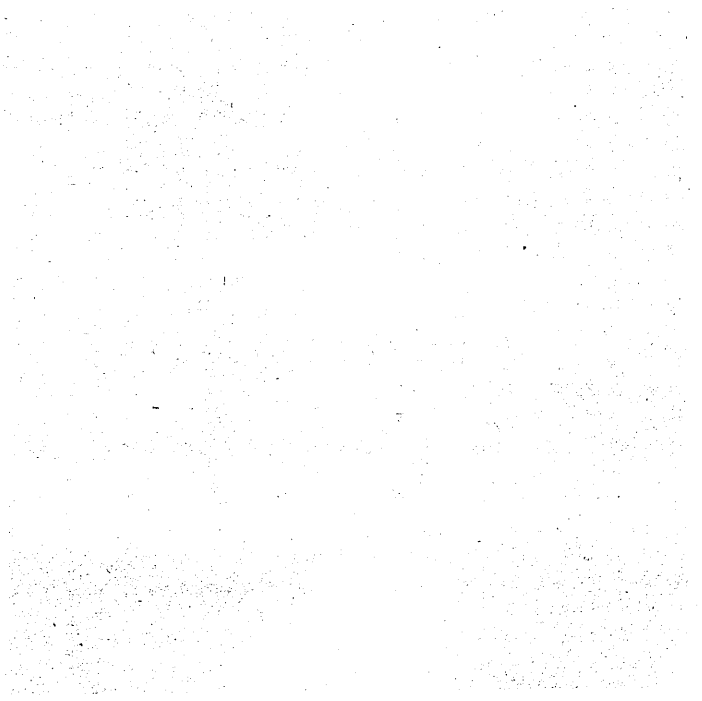
2. If the atelectatic area does not re-expand, the bronchiectasis is permanent.

3. If the atelectasis does resolve, the bronchiectasis may or may not disappear, depending on the degree of damage sustained by the bronchial walls when the atelectasis was present. It is possible that the elasticity of the bronchial walls may be permanently impaired by prolonged mechanical overdistension alone, but in the vast majority of cases the damage results from a combination of mechanical overdistension and infection.

4. The minimum degree of atelectasis which will cause significant bronchial dilatation is so far unknown, but presumably a considerable portion of a lobe would have to be affected.

5. When the bronchus supplying an area of lung is proximally occluded, atelectasis of the pulmonary tissue results. Air is also absorbed from the bronchial tree distal to the obstruction, and the bronchi in the atelectatic system therefore collapse as far as the nature of their walls permits. If infection supervenes, bronchiectasis may ensue; this is due to the damming up of infected secretions, and not to the mechanical stresses set up by atelectasis. Proximal bronchial obstruction, however, can only account for a small proportion of cases of bronchiectasis.

6. As regards the causation of bronchiectasis in general, it appears that the vast majority of cases originate in atelectasis brought about by the occlusion of bronchioles or finer bronchi. As the atelectasis in many instances disappears before investigation is possible, evidence of its former existence must necessarily be indirect, but no other theory appears capable of supplying a satisfactory explanation for the causation of the disease.



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CONTENTS.

VOLUME 3.

CHAPTER 4.

It is convenient to divide this chapter into three sections. In the first section the dynamics of atelectasis is discussed, in the second a test for distinguishing potentially reversible from permanent atelectatic bronchiectasis is suggested, and in the third artificial pneumothorax as a therapeutic agent in potentially reversible atelectatic bronchiectasis is considered.

SECTION 1.

THE DYNAMICS OF ATELECTASIS.

Preliminary Considerations.

A review of the literature on atelectasis revealed that one of the most striking accompaniments of the condition is a marked lowering of the intrapleural pressure in the affected side of the chest. In the earlier cases reported, there was complete or almost complete collapse of the lung, and the intrapleural pressures observed in the affected hemithorax were often very low indeed. Farris[1] for example, noted readings of -16,-10 cms. of water, Elkins[2] recorded readings of -20,-16 cms. of water, and Habliston[3] returned readings of -43,-33 cms. of water. The latter writer accounted for the extreme lowering of the pressure in his case by suggesting that the extent of the collapse was greater than in the other cases mentioned. Lander and Davidson[4] in cases of lobar atelectasis due to bronchiolar obstruction, demonstrated a similar lowering of the intrapleural pressure, their figures in one case being -30,-24 cms. of water, and in another case -22,-8 cms. of water. (It has already been noted that in the text of their

publication millimetres not centimetres of water appeared, but this, from the context, was obviously a mistake).

When collapse was extensive as in some of the earlier cases appearing in the literature, and the mediastinum had moved markedly to the affected side, cardiac embarrassment and cyanosis often ensued. Farris[1] and Habliston[3] were convinced that this mediastinal shift was due to the lowered intrapleural pressure, and in order to restore it to normal, introduced air in several cases into the intrapleural space in the affected hemithorax. The gratifying result was that the mediastinum moved back to its former position, and symptoms of distress were at once alleviated. Lander and Davidson[4] were of the opinion that the low intrapleural pressure so often observed in cases of pulmonary collapse, was responsible not only for the shift of the mediastinum to the atelectatic side, but for the dilatation of the bronchi in the collapsed area. In one case, as has already been mentioned in the previous chapter, they demonstrated dilated bronchi in an atelectatic lobe, and recorded an isolateral intrapleural pressure of -30.-24 cms. of water. Three hundred and twenty five c.c.s of air were introduced into the pleural space raising the pressure to -10, zero. A subsequent reading revealed a pressure of -16,-4,and on a further five hundred c.c.s of air being given, the pressure became -4,+4 cms. of water. Bronchography was performed, and demonstrated that the bronchial dilatation, like the mediastinal shift, had disappeared. Further, when absorption of the air which had been introduced caused the intrapleural pressure to revert to its former low level, these phenomena again became evident.

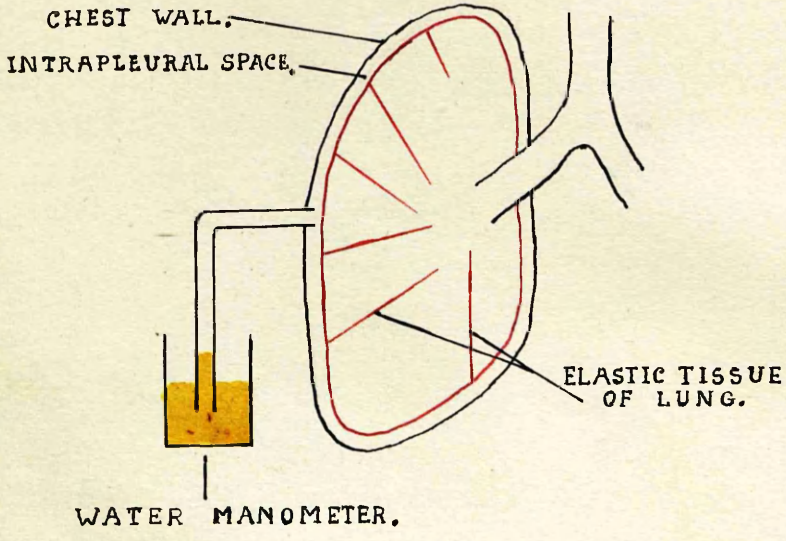
The publications just considered are interesting and important contributions to the literature, and Lander and Davidson's[4] in particular suggested to the author that artificial pneumothorax might be a valuable therapeutic agent in cases of potentially reversible atelectatic bronchiectasis which it was thought were likely to be encountered in the course of the whooping cough investigations. A study of the literature, however, made it apparent that great confusion has arisen as to the nature and effects of intrapleural pressures, not only in cases of pulmonary collapse, but also in normal subjects, and it is therefore proposed to consider this problem in some detail.

It is well known that intrapleural readings in healthy subjects record sub-atmospheric pressures, and apparently on this account it is commonly believed that it is a sub-atmospheric gas pressure in the intrapleural space which prevents the lung from collapsing owing to its inherent elasticity. In considering the dynamics of respiration it is important to remember that this is not the case; there is no gas in the normal intrapleural space, and consequently no sub-atmospheric gas pressure. Intrapleural readings really record the gas pressure of a pneumothorax which is created when the needle is introduced into the intrapleural space in the course of the procedure. In the normal chest the visceral pleura is closely applied to the parietal pleura, and only separated from it by a thin film of liquid. The pleural surfaces are thus held together by the cohesion of the molecules of the liquid, and to overcome this cohesion would obviously require a force enormously greater than that which could be exerted under any conceivable circumstances by the elastic tissue of the lung.

Because of the fallacy just referred to, it has been stated that during inspiration there is a lowering of the intrapleural pressure, and that as a consequence of this, expansion of the lung takes place. What actually happens is that owing to muscular effort the walls of the thorax move outwards, and this expansive force is transmitted to the periphery of the lung through the film of liquid in the intrapleural space, with resultant expansion of the organ. When the intrapleural space is completely obliterated by adhesions it is obvious that there can be no low gas pressure in a space filled with solid material, yet expansion of the lung on inspiration still takes place.

The conception that there is normally a sub-atmospheric gas pressure in the intrapleural space has led to errors with regard to pulmonary tensions which would not have been made had it been remembered that the space is occupied by a film of fluid. It is submitted that Cameron[5] in a recent article on pulmonary oedema has been misled through overlooking this fact, and it is proposed closely to examine the relevant passage of his publication at this point in order to avoid repetition later in refuting statements by other writers embodying what is considered to be the fallacy he propounds.

Cameron[5] states, "Now, the colloid osmotic pressure for pulmonary blood is so far as we know, that of the blood in general, which is variously stated as 18.3 to 25 mm. Hg (Verney, 1926; Landis, 1930; Muntwyler et al., 1933; Kerkhof, 1937). This means that conditions favour retention of fluid within the pulmonary capillaries even when we take into consideration the sub-atmospheric ("negative") intrapleural pressure fluctuating about a mean of 5 cm. of water (Printzmetal and Kountz, 1935;


CHEST WALL.
INTRAPLEURAL SPACE.
ELASTIC TISSUE OF LUNG.
WATER MANOMETER.

Drinker, 1945), which favours outflow from the capillaries."

Cameron is obviously suggesting that there is a sub-atmospheric gas pressure in the intrapleural space, and that this sub-atmospheric gas pressure tends in some way to "suck" fluid out of the capillaries of the lung. If it is remembered that in fact there is a tension on a film of liquid in the pleural cavity due to the pull of the elastic tissue of the lung, it is seen at once that this tension cannot have any effect in inducing fluid to leave the pulmonary capillaries.

It is instructive to consider what takes place, however, when a marginal pneumothorax is created by the insertion into the pleural space of a needle connected to a water manometer. A simple diagram is shown opposite.

Suppose the subject inspires, holds his breath, but does not close the glottis. The air in the pleural space, and in the manometer tube above the level of the liquid, will be at a pressure lower than that of the atmosphere, and the difference will be indicated by the rise of the water in the manometer tube. According to Myers and Blades[6], the pressure in the intrapleural space in an average case will be eight cms. of water less than atmospheric pressure. But what is the pressure of the air in the alveoli in which run the pulmonary capillaries? It is at atmospheric pressure; otherwise air would move into or out of the thorax. Therefore, although there is a sub-atmospheric pressure in the intrapleural space, the gas pressure in the lung itself is at atmospheric pressure, and hence there is no negative pressure applied to the pulmonary capillaries tending to induce fluid to move through their walls and produce pulmonary oedema.

Let it now be supposed that expiration has taken

place, and the glottis is again kept open as it is during normal respiration. The intrapleural pressure will now approximate more nearly to the atmospheric pressure; according to Myers and Blades[6] in an average case it will be four cms. of water less than atmospheric pressure. Again the gas pressure in the alveoli must be at atmospheric pressure or air would move out of or into the thorax. Once more there is no negative pressure inducing fluid to leave the pulmonary capillaries.

Next let the position be considered during the expiratory phase. At the beginning of expiration the intrapleural pressure was eight cms. of water less than atmospheric, and at the end of expiration four cms. of water less than atmospheric. During the expiratory phase the intrapleural pressure must therefore lie between eight cms. and four cms. of water less than atmospheric pressure. What is the pressure of the air in the pulmonary alveoli during this period? It is greater than atmospheric pressure, otherwise air would not move out of the thorax. Thus, although during expiration the intrapleural pressure is sub-atmospheric, the gas pressure in the pulmonary alveoli is greater than atmospheric pressure, and far from inducing fluid to leave the pulmonary capillaries actually tends to prevent it from leaving them.

During inspiration the intrapleural pressure will fall from four cms. of water below atmospheric pressure to eight cms. of water below atmospheric pressure. The gas pressure in the pulmonary alveoli will also be sub-atmospheric, otherwise air would not move into the lung. During this period, therefore, the gas pressure in the pulmonary alveoli is such that it would tend to induce fluid to leave the pulmonary capillaries.

It is clear from what has been said that a sub-atmospheric pressure per se does not have any effect on the pulmonary capillaries. It is change in the intra-pleural pressure, which, by causing alterations in the gas pressure in the pulmonary alveoli, has an effect on the pulmonary capillaries. But the change in inspiration which tends to cause fluid to leave the pulmonary capillaries is exactly counterbalanced by the change in expiration which tends to keep fluid in the pulmonary capillaries.

In the hypothetical case in which intrapleural pressureswere taken it was postulated that at the end of inspiration the intrapleural pressure was -8 cms. of water, and at the end of expiration -4 cms. of water. It is worth noting that during inspiration, when the intrapleural pressure is -6 cms. of water, the pressure in the pulmonary alveoli is also sub-atmospheric, but when the intra-pleural pressure is -6 cms. of water during expiration, the pressure in the pulmonary alveoli is greater than atmospheric pressure. This emphasises the fact that it is change in the intrapleural pressure which influences intra-alveolar pressure. If the intrapleural pressure does not change, the fact that it is sub-atmospheric has no influence on the intra-alveolar pressure. If a low intrapleural pressure really tended to produce pulmonary oedema, one would expect that this complication would be frequent in cases of pulmonary collapse when the whole lung was not involved. When the greater part of a lung collapses it has been seen that very low intrapleural pressures have been recorded. The non-atelectatic part of the lung would therefore be exposed to the effects of this very low intrapleural pressure, but if this tends to cause pulmonary oedema the fact has certainly been so far

overlooked in the literature on atelectasis. Experience confirms that Cameron[5]'s reasoning is fallacious. It may be observed that Drinker[7], whom Cameron[5] quotes, also confuses intrapleural pressures with intra-pulmonary pressures.

The significance of the intrapleural pressures in healthy subjects may now be further examined. Suppose a needle, connected with a water manometer, has been inserted into the intrapleural space. The position is diagrammatically represented opposite page 188. Now suppose the subjects inspires and holds his breath at the end of inspiration. The outer thoracic wall is prevented from moving inwards by muscular action. The periphery of the lung is also motionless. What is the balance of forces which prevents the periphery of the lung from moving either outwards towards the thoracic wall, or inwards away from the thoracic wall?

There are two forces tending to cause the periphery of the lung to move inwards; first, the pressure of the air in the intrapleural space and the manometer tube above the water level, and second, the elastic tension of the lung. There is one force tending to cause the periphery of the lung to move outwards; the pressure of the air in the pulmonary alveoli which is the same as that of the atmosphere. Since the periphery of the lung is motionless, equilibrium has been attained, and the balance of forces can be expressed by the following equation :-

Intrapleural Pressure + Elastic Tension of Lung =
Atmospheric Pressure.

It follows that :-

Elastic Tension of the Lung = Atmospheric Pressure -
Intrapleural Pressure.

The difference between atmospheric pressure and the intrapleural pressure is measured by the extent of the rise of the water in the manometer tube. It is therefore possible to express the elastic tension of the lung in terms of centimetres of water; thus if the reading is recorded in the usual way as -8 cms. of water, it means that the intrapleural pressure is atmospheric pressure in centimetres of water minus eight centimetres, and the elastic tension of the lung, expressed in the same way is eight centimetres of water.

The equation may be written in yet another way :-
Intrapleural Pressure = Atmospheric Pressure - Elastic Tension of the Lung.
In other words, the greater the elastic tension of the lung, the lower the intrapleural pressure will be.

During respiration, the equilibrium of the forces noted above is constantly being disturbed, with the result that the periphery of the lung moves outwards and inwards. In the inspiratory phase, muscular effort causes the thoracic walls to move outwards, and if intrapleural readings are taken it is seen that the intrapleural pressure falls. The combined forces of the intrapleural pressure and the elastic tension of the lung are now less than the pressure of the alveolar air, and consequently the periphery of the lung moves outwards. As the lung expands the pressure of the alveolar air also falls and air thus moves into the thorax from the atmosphere. At the end of inspiration equilibrium is again attained, but the forces now in balance are different from what they were at the beginning of inspiration. The alveolar air is still at atmospheric pressure, but the intrapleural pressure is lower than it was at the beginning of inspir-

ation, and the elastic tension of the lung is correspondingly greater than it was at the beginning of inspiration. The changes in the relevant forces during the expiratory phase are the reverse of those in the inspiratory phase.

The subject of intrapleural pressures has been dealt with at some length, in order to avoid the confusion which seems often to arise when the dynamics of atelectasis are discussed. The forces set in motion when pulmonary collapse occurs may now be considered.

When a portion of a lung collapses, air is absorbed from the alveoli of the affected lung tissue. Consequently, part of the lung, as it were, disappears. The part remaining has, however, to accomodate itself to a hemithorax almost the same size as before, since the surface of the lung cannot retract from the chest wall owing to the cohesion of the molecules of the thin layer of liquid in the intrapleural space. The remaining part of the lung is therefore now stretched out to occupy a space practically the same as that formerly occupied by the whole lung, and its elastic tension is consequently correspondingly increased. If a marginal artificial pneumothorax is created by the insertion into the isolateral intrapleural space of a needle connected with a water manometer, the intrapleural pressure will be found to be lower than normal, because, as has been explained, the greater the elastic tension of the lung, the lower the intrapleural pressure.

The overstretching of the affected lung is somewhat mitigated by various compensatory mechanisms. The walls of the affected hemithorax may be pulled inwards slightly, thus somewhat diminishing the size of the thoracic cavity, but owing to the rigidity of the thoracic cage the relief

to the overstretched lung is extremely limited. The thoracic cavity is more appreciably diminished in size by elevation of the diaphragm, which, being mobile, is pulled upwards by the overstretched affected lung, thus relieving the tension of the lung to some extent. Within the thoracic cavity itself another compensatory mechanism may be at work. If the mediastinum is mobile, it will be pulled to the affected side of the chest, thus diminishing the elastic tension of the lung involved in collapse. If the mediastinum is pulled across, the contralateral lung will be slightly overstretched, its elastic tension will increase, and if a needle connected with a water manometer is inserted into the contralateral intrapleural space, the intrapleural pressure will consequently be found to be lower than normal. If the mediastinum is not mobile, however, no relief will be afforded to the overstretched affected lung, and the elastic tension of the contralateral lung will remain the same.

As a result of the overstretching of the lung affected by pulmonary collapse, it is plain that any remaining airbearing alveoli will become distended, and if the collapse is due to obstruction of bronchioles or finer bronchi, then, since the bronchi in the collapsed zone proximal to the obstructing plugs are patent and in communication with the atmosphere, they will be distended just as are the alveoli in the unaffected part of the lung.

Lander and Davidson[4] and many writers acquainted with their work, for example, Ogilvie[8] and Wearing[9], have suggested that the low intrapleural pressure associated with pulmonary collapse is responsible for the alveolar distension, dilatation of the bronchi in the collapsed zone, and mediastinal shift to the affected side which have frequently

been observed. It is obvious, however, that it is not gas pressure differences between the intrapleural space and the intrapulmonary air which produce these phenomena, for it has been pointed out that there is no gas in the intrapleural space. It is true that when intrapleural readings are taken, it is seen that the small amount of air which has entered the intrapleural space is at a pressure further below atmospheric pressure than would have been observed had pulmonary collapse not taken place. This is the result of the increased elastic tension of the lung brought about by the "loss of space" which occurs when air is absorbed from the alveoli of the collapsed portion of lung. If this "lost space" is restored by resolution of the collapse, the elastic tension of the lung will return to normal, and consequently an intrapleural reading would show that the small amount of air introduced into the intrapleural space in the course of the procedure was at the normal sub-atmospheric pressure. It is clear, therefore, that the importance of the low intrapleural gas pressures recorded in cases of pulmonary collapse consists merely in the demonstration that the elastic tension of the lung increases owing to the overstretching to which it is subjected.

It is obvious that the increased elastic tension of a lung involved in pulmonary collapse, and the associated compensatory phenomena, would disappear if the lung were allowed to occupy a smaller volume. This could be done in two ways - by reducing the capacity of the thoracic cavity, or by allowing the periphery of the lung to recede from the chest wall by introducing liquid or gas into the intrapleural space.

The size of the thoracic cavity could be reduced by thoracoplasty, and in a case of pulmonary tuberculosis complicated by collapse in which this operation was performed, Lander and Davidson[4] demonstrated the disappearance of all the associated compensatory phenomena - the shift of the mediastinum, the rise of the diaphragm, the alveolar distension in the airbearing portion of lung, and the bronchial dilatation in the collapsed zone. The thoracoplasty was, of course performed on account of the tuberculosis, and its effect on the tensions set up by the collapse was incidental.

The introduction of liquid into the intrapleural space in order to reduce the overstretching of a collapsed or partially collapsed lung is only of theoretical interest for it would be much more convenient to introduce a gas, and Elliot and Dingley[10] as long ago as 1914, suggested that intrapleural injections of oxygen might relieve cyanosis and dyspnoea occurring in pulmonary collapse by allowing the displaced mediastinum to return to its normal position. It has been mentioned that Farris[6] and Habliston[3] adopted this procedure with gratifying results, and it has also been noted that Lander and Davidson[4] demonstrated in a case of pulmonary collapse that the introduction of air into the intrapleural space not only abolished shift of the mediastinum, but abolished alveolar distension in the airbearing part of the lung, and bronchial dilatation in the collapsed part. It has previously been explained that the difference between the pressure of a gas in the intrapleural space and the atmospheric pressure is a measure of the elastic tension of the lung. Lander and Davidson[4] found that the gas pressure in the intrapleural space rose as they introduced more air, and when the media-

stinal shift and the other compensatory phenomena had disappeared, the difference between the pressure in the intrapleural space and the atmospheric pressure was approximately that found in subjects without pulmonary collapse. From what has been said, it is seen that this meant that the affected lung had been allowed to occupy such a volume that its elastic tension had returned to normal. As the air in the intrapleural space was absorbed, the affected lung once more became overstretched, and its elastic tension, as indicated by the intrapleural pressure, increased. The compensatory phenomena once more appeared. When all the air was absorbed from the intrapleural space, and the lung was once more compelled to fill the whole hemithorax, the effects of the overstretching were as obvious as they were before air had been introduced, and the lung relaxed.

Experimental Studies of Intrapleural Pressures.

Having discussed the effects of the respiratory movements on the intrapleural pressure (i.e. on the gas pressure of a very small artificial pneumothorax) in healthy subjects, and the influence on the intrapleural pressure of the increased elastic tension of the lung arising as a result of atelectasis, experimental work on intrapleural pressures carried out in the course of these investigations may now be considered.

1. Intrapleural pressures in healthy subjects.

In view of the interest aroused by the demonstration of low intrapleural pressures in cases of atelectasis, it was decided to investigate this matter, but before doing so, it was essential to find out the average intrapleural

pressure in healthy subjects. Most estimates of the average intrapleural pressure are based on the findings in patients suffering from pulmonary tuberculosis, but, of course, unless cases are carefully selected, using only those in which the disease is minimal, false impressions may be gathered regarding the normal level. Much will obviously depend on the elasticity of the lung tissue. The greater this is, the lower will be the intrapleural pressure. If a considerable portion of the lung were diseased, elasticity would be diminished, and the intrapleural pressure less negative than usual, providing pulmonary fibrosis was not present. Some conditions also will affect not only initial intrapleural pressure readings, but subsequent readings if air is introduced in order to relax the lung. It is well known, for example, that in fibrous tuberculosis, the initial reading may record a very low intrapleural pressure, which rapidly rises on the introduction of a small quantity of air. In an actual case of fibrous tuberculosis with a free pleura, the induction readings were -30,-18 cms. of water, and after a mere 150 c.c. of air had been given, the readings fell to -4,-1 cms. of water. This phenomenon has been noted by Myers and Blades[6], Andrus[11] and others. It is suggested that the contracting fibrous tissue exerts a strong pull lowering the intrapleural pressure, but the introduction of a comparatively small quantity of air allows the lung to diminish in volume sufficiently to permit relaxation of the fibrous strands. When this happens, the introduction of more air produces a sudden rise in the intrapleural pressure, and as the normal elasticity of the lung is reduced, a few more c.c.s of air speedily brings the intrapleural pressure to that of the atmosphere.

A similar rapid rise in the intrapleural pressure on

the introduction of air may occur if the pleura is partially adherent, and the needle is introduced into a free area. Under these circumstances, the introduction of a few c.c.s of air may obviously bring the pressure to that of the atmosphere.

Again, if the visceral pleura is partially adherent, but capable of being stripped from the chest wall when air is given, the induction reading may be less negative than if the pleura were completely free.

It is seen, therefore, that unless a careful selection of tuberculous cases is made, erroneous estimates as to the normal intrapleural pressure may be arrived at. The ideal case would be one in which there was a very small tuberculous focus, not large enough to interfere with the elasticity of the lung tissue as a whole, and not involving the pleura.

However, making due allowance for the fallacies mentioned, a large series of induction readings in cases of tuberculosis should give an approximation to the normal, and since in any particular case, owing to instrumental variation and differences in the extent of respiratory excursion, there will anyhow be a fair margin of error, the results of such a series are no doubt good enough for practical purposes. Myers and Blades[6] and Andrus[11] on the basis of induction readings in cases of tuberculosis, estimated the average intrapleural pressure as varying between -8, and -4 cms. of water.

The induction readings of one hundred cases of artificial pneumothorax performed in cases of tuberculosis at Ruchill Sanatorium were considered. In forty five of these cases, the procedure was carried out by the author. Wide variations were noted, but the average readings closely approximated to those already given. The figures were -7 cms. of water on inspiration, and -3.1 cms. of water on

INTRAPLEURAL PRESSURES IN NORMAL SUBJECTS.

	Age of subject in years.	Pressure in cms. of water.	
		Inspiration.	Expiration.
1.	35	-14	-10
2.	21	- 9	- 5
3.	27	- 8	- 4
4.	18	- 8	- 4
5.	17	- 8	- 4
6.	15	- 8	- 4
7.	8	- 8	- 3
8.	45	- 7	- 4
9.	28	- 7	- 4
10.	19	- 7	- 4
11.	9	- 7	- 4
12.	5	- 7	- 4
13.	28	- 7	- 3
14.	25	- 7	- 3
15.	6	- 7	- 3
16.	26	- 6	- 4
17.	23	- 6	- 3
18.	33	- 6	- 3
19.	14	- 5	- 2
20.	22	- 4	- 2
Average Readings		- 7.3	- 3.85

expiration.

After all, however, this method of estimating the normal intrapleural pressure is only second best. The ideal is to test it in lungs, which so far as is known, are free from disease.

Twenty subjects with lungs clinically and radiographically normal, were therefore investigated by the author. The average intrapleural pressure was found to vary between -7.3 cms. of water on inspiration, and -3.85 cms. of water on expiration, an observation in keeping with that of Aron,[12] who in a study of thirty six normal individuals discovered that the average intrapleural pressure varied between -8 cms. of water on inspiration and -3.4 cms. of water on expiration.

The table shown opposite indicates the readings in individual cases, and the ages of the subjects, who were all males. It will be noted that the variations are considerable. One adult had readings of -14,-10 cms. of water. As compared with the others, the intrapleural pressure was therefore exceptionally low. This could be accounted for by the patient taking a fairly deep inspiration, and then only allowing a comparatively small respiratory excursion, but this was not noted at the time. It could scarcely be explained by postulating fibrosis of the lung, for there was no evidence of this in a radiogram, and besides, when there is fibrosis, although the inspiratory and expiratory readings are both low, there is usually a very marked difference between them. This is because during inspiration the comparatively rigid lung does not readily expand. It appears, therefore, that in some normal subjects the elastic tension of the lung may be unusually high, and the intrapleural pressure consequently unusually low. In another case, it will be observed that the reverse was found.

These wide variations agree with the findings of Evans[13] who states that intrapleural readings for human subjects vary between -10,-5 mm. of mercury (-13.6,-6.8 cms. of water) for inspiration and -5,-3 mm. of mercury (-6.8,-4.1 cms. of water) for expiration.

It was interesting to observe, in view of the following statement by Andrus," that there was no appreciable difference in the readings of adults and children, though no child younger than five years of age was investigated. "A further weighty argument against the probability of the normal elastic tension of the lung customarily producing bronchial dilatation, is, in fact, that a large percentage of cases arises during childhood and infancy. At birth, however, the lung has no elastic tension, and we are told that an average of seven years, and a maximum of fourteen years, passes before the adult development of the lung is attained. This means that the lung has normally an unusually low elastic tension during the very period in which bronchiectasis is common."

The number of cases which were investigated is, of course, so small that no comment on the general accuracy of the statement quoted can be made. It can be inferred, however, that the lungs of some children at least, appear to have just as great an elastic tension as those of the average adult. It seems strange, however, that Andrus" should cite the fact that development of the lung proceeds up to the age of seven and even fourteen years, as evidence that the elastic tension should be low, as it does not necessarily follow that the two factors are inseparable.

With reference to the allegedly low intrapleural pressure in infancy, the opportunity was taken of studying the intrapleural pressure in two infants, aged seven and

three months respectively, where closed drainage for empyema had been instituted. The average readings were -7,-4 cms. of water in both cases. It could not be claimed, of course, that such a method of estimating the intrapleural pressure was ideal or accurate, but the figures do not suggest that the elasticity of the lung even at that early age is low. If this were indeed the case, it would mean that the insertion into the intrapleural space of a needle communicating with the atmosphere, would not cause extensive collapse of the lung in an infant. This appears to be almost incredible, and, indeed, in the treatment of empyema in infants, open drainage is avoided just as it is in adults. Therefore, although Evans[13], for example, states that the lungs of newly born mammals do not collapse if the thorax is opened, it would appear that this state of affairs does not last for long.

The main point, however, which arises from a contemplation of the variations in the intrapleural readings in those apparently normal individuals, is that it would not be at all easy to say in a case of pulmonary collapse, in the absence of knowledge of the normal intrapleural pressure of the case, whether or not there had been a reduction due to the atelectasis.

2. Intrapleural pressures in cases of atelectasis.

Pending the arrival of cases in the whooping cough series in which the onset of atelectasis would be known to within a week, it was decided to investigate cases of atelectatic bronchiectasis admitted to the hospital in which the duration of the condition was problematical.

The first patient studied was a man of twenty five years with well marked atelectasis and cylindrical bronchiectasis of the right lower lobe.

The intrapleural pressures recorded in the affected side were -6,-4 cms. of water. This was an astonishing finding. Nothing in the literature had indicated that the intrapleural pressure in cases of well marked atelectasis was not constantly decidedly reduced. Yet here was an example where the intrapleural pressure was apparently well within normal limits. Intrapleural pressures taken on four other similar patients gave the following results:- -5,-3; -8,-4; -5,-3; -7,-5 cms. of water. The cases will be more fully discussed later. These results were rather astonishing in view of the fact that all theoretical calculations had led to the expectation of an unduly low intrapleural pressure. It was concluded that there must have been some difference between the cases appearing in the literature, and the cases which had been investigated. A difference in extent of the atelectasis might have accounted for the disparity in the figures, but in a case quoted by Lander[4], the atelectasis involved only a lobe. Besides, the bronchograms of the cases studied showed dilated bronchi, and if the theory advocated above were correct, one would have expected the increased elastic tension of the lung to be reflected in a lowered intrapleural pressure.

It was observed, however, that the cases quoted in the literature were cases in which the atelectasis was almost certainly of recent origin. The cases under investigation, on the other hand, were probably, though not certainly, of comparatively long duration. It seemed possible that this factor might account for the difference in results. Colour was lent to this view by the fact that in two cases of proximal obstruction of the main bronchus of a lung, in which the atelectasis resulting was demon-

strated radiographically within a few days of onset, the intrapleural pressure in the affected side was so low that the water was almost sucked out of the manometer before the needle could be withdrawn. That is to say, it was lower than -16 cms. of water, the lowest reading the instrument employed recorded. These cases have already been discussed in detail in the previous chapter. (Case 42, p.121 and Case 2, p.124).

The results of the above investigations suggested that the intrapleural pressure might be a valuable guide in determining whether or not bronchiectasis was reversible. It seemed probable that in established cases, unlike early cases, the intrapleural pressure would not be much, if at all, lowered. It appeared that as the compensatory changes became established, the intrapleural pressure reverted to normal. Continuous overdistension of any still airbearing alveoli in the atelectatic area, and continuous overdistension of alveoli peripheral to the atelectatic area, for example, would eventually cause disintegration of the elastic tissue of their walls with resultand emphysema, and this, in fact, is what Ogilvie[8] discovered in all the specimens of atelectatic bronchiectasis which he examined. Once the elastic pull of the alveolar walls had thus disappeared, the intrapleural pressure would be correspondingly less negative. The bronchi in the atelectatic area, as has been seen, are also dilated, because the periphery of the lung is maintained in its position close against the chest wall, and hence the walls of the bronchi are pulled apart. As they are elastic, they will maintain at first a corresponding pull in the opposite direction, which is reflected in the diminished intrapleural pressure. But if, in the course of time, the bronchial walls lose

their elasticity, their inward pull will diminish, and the intrapleural pressure will tend to revert to normal. It is as if one end of a rubber band attached at its other end to a fixed point is seized, and pulled out a certain distance. A corresponding pull from the rubber band is felt. If the band loses its elasticity, its pull will gradually diminish. So it is suggested, the pull of the dilated bronchi will decrease as their elasticity disappears, and this diminution will be reflected by the gradual reversion of the intrapleural pressure to normal.

These considerations suggested that the isolateral intrapleural pressure might be a valuable guide as to whether atelectatic bronchiectasis was potentially reversible or not; if it was found to be lowered, the indications would be that bronchial elasticity had been retained, whereas if it were not lowered, the presumption would be that bronchial elasticity had been lost, and even if the atelectasis cleared up the bronchial dilatation would remain.

Intrapleural readings in normal subjects, however, had shown such wide variations that it was clear it would be difficult to be certain in a particular case whether the intrapleural pressure had been lowered or not, but it seemed that this problem might be solved if intrapleural pressures were taken simultaneously on both sides of the chest. It would be significant if the intrapleural pressure of the atelectatic side of the chest were markedly lower than the intrapleural pressure of the contralateral side.

Adams and Escudero[14] found that in normal dogs the intrapleural pressure ranged between -6 and -8 cms. of water. When atelectasis of the complete left lung was

produced, the pressure was found to be -14,-18 cms. of water on the left side, and -10,-12 cms. of water on the right. Habliston,[3] on the other hand, reported of a case of complete atelectasis of the left lung due to occlusion of the left main bronchus by an aneurysm occurring in the human subject, that whereas the intrapleural pressure on the left side was -33.7,-43.2 cms. of water, the pressure on the right side was only -4,-8 cms. of water, which is an average reading in healthy subjects. In a case already referred to (Case 2, p.124), in which there was complete atelectasis of the left lung, not only was the intrapleural pressure so low in the affected side that the needle had to be promptly withdrawn to avoid aspiration of the water from the manometer into the pleural cavity, (i.e. the pressure was much less than -16 cms. of water, the maximum mark on the instrument) but the intrapleural pressure in the right side was -12,-10 cms. of water. This was lower than the average, and appears to confirm the findings of Adams and Escudero,[14] though as has been pointed out above, the intrapleural pressure in an apparently normal subject may occasionally be as low as this.

The balance of evidence seemed to indicate that if the mediastinum were mobile when extensive atelectasis occurred in one lung, the intrapleural pressure on the contralateral side would be somewhat reduced, but not to anything like the extent it would be on the affected side. If the mediastinum were fixed, of course, the intrapleural pressure in the contralateral side would not be lowered at all. It therefore seemed that intrapleural readings taken at approximately the same time on both sides of the chest, might give a reasonable indication whether bronchial dilatation in an atelectasis were still potentially revers-

ible, or whether it had become permanent.

It was desirable, in order to test this theory, to procure cases in which the onset of atelectasis was known, and ascertain the intrapleural pressures within a short time of the occurrence of the condition.

Extensive atelectasis was noted in four children over three years of age in the whooping cough series. The date of onset of the atelectasis was known to within a few days, and these cases were therefore excellent material for investigation.

It had been decided, however, to determine the effects of artificial pneumothorax on such cases, and one was left alone in order to serve as a control. Intrapleural pressures might well have been ascertained in this case, even though, since both the right and left lower lobes were atelectatic, the information would not have been of much value in the present connexion, but this was not done. Details of the case have already been given. (Case 16, p.137).

A second case had a partially adherent pleura, and therefore the readings had to be disregarded. Another case developed a spontaneous pneumothorax, and thus the initial intrapleural pressure was not available. Only the fourth case was uncomplicated and valuable in the present line of investigation.

This case, details of which will later be given, showed extensive collapse of the left lower lobe with marked dilatation of the contained bronchi. The intrapleural pressure was -8,-4 cms. of water on the left side, and -5,-3 cms. of water on the right. The latter reading was taken immediately after the former, factors such as the position of the patient, the place of insertion of the needle etc., being kept as similar as possible.

The small reduction of the intrapleural pressure on the atelectatic side was a somewhat astonishing finding; in fact had the pressure of the contralateral intrapleural space not been ascertained, it might have been thought that the intrapleural pressure on the affected side had not been reduced at all, as a reading of -8,-4 cms. of water was well within normal limits. Even as it was, the difference was by no means striking, and might well have been accounted for by instrumental error, or variation in the depth of respiration, though it is unlikely that these factors actually came into play, as the same instrument was used for both sides of the chest, and the child was of such a placid nature that accurate recording was possible.

It has already been noted that such studies on intrapleural pressures in cases of recent atelectasis as have appeared in the literature suggest that the readings on the affected side are markedly low, and that two cases which were subsequently available for investigation supported this view. These findings were therefore in complete accord with the atelectatic theory of bronchiectasis expounded in the previous chapter. Yet to find even one case which did not fulfil the theoretical expectations was disconcerting, and cast doubt on the whole conception of the mechanism producing bronchial dilatation in an atelectatic area.

How was it to be reconciled with a theory which hitherto had seemed so well supported by the facts?

The cases in which low intrapleural pressures had been recorded were reviewed, in order, if possible, to discover some point of difference capable of explaining the disparate findings.

Habliston's[3] cases, and Case 42, page 121, and Case 2, page 124, were examples of complete collapse of a lung, and

the difference in extent of the atelectasis was therefore capable of accounting for the discrepancy in the degree of lowering of the intrapleural pressure.

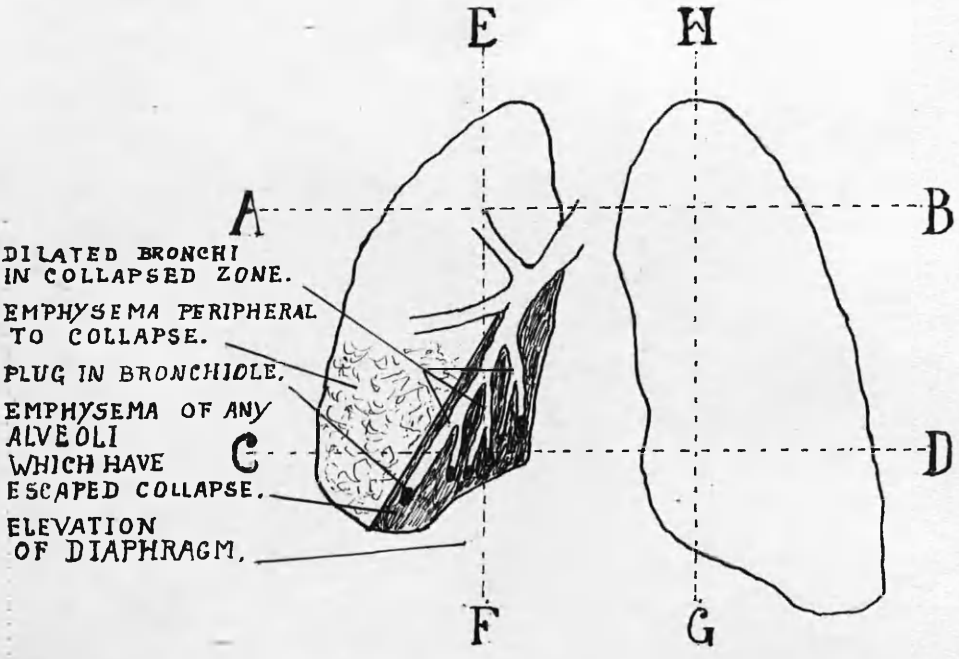
Lander[4], however, in 1938 demonstrated a case of complete lower lobe collapse in which the intrapleural pressure on the affected side was -22,-8 cms. of water. The case under discussion was one of extensive, but not quite complete collapse of a lower lobe, so that the difference in extent, coupled with the individual variation in intrapleural pressures (which, as was shown earlier, may be considerable), might again satisfactorily explain the difference in the figures.

The fact remained, however, that the bronchi in an atelectasis of known recent origin had been shown to be markedly dilated, and if this were due to the overstretching of the remaining and still elastic tissue in the affected hemithorax as has been suggested, it seemed strange that a force capable of so dilating the bronchi was not evidenced by an intrapleural pressure lower than -8,-4 cms. of water.

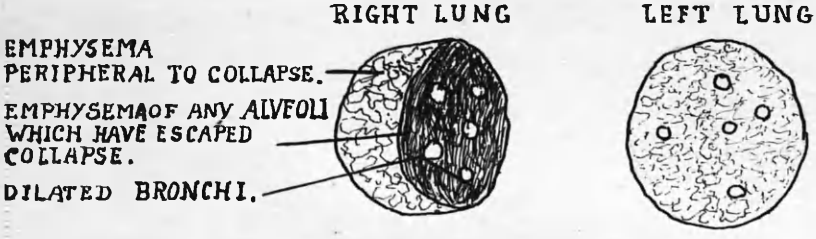
Upon reflection, it appeared that there could only be one explanation capable of reconciling the apparently contradictory facts - the stresses set up by the atelectasis must be concentrated in the affected area, and dissipated to some extent in the intrapleural space.

The discovery of this possibility led to a more exact consideration of the dynamics of atelectatic bronchiectasis.

LUNG STRESSES IN ATELECTASIS



HORIZONTAL SECTION AT C D



Lung Stresses in Atelectatic Bronchiectasis.

As has already been stated, when atelectasis occurs, there is a loss of volume in the contents of the thoracic cavity, and because of the comparative rigidity of the chest wall, internal stress is set up. The increased tension of the contents of the affected hemithorax is reflected in a lowering of the intrapleural pressure.

If an intrapleural pressure is taken, as the intrapleural space is continuous, the pressure at every point in it will be lowered by approximately the same amount. The usual assumption in the literature is that likewise in the interior of the lung the new tensions set up will be everywhere the same. The suggestion is that the lung is like a balloon with a few air filled compartments enclosed in a rigid outer container, and if the air in one of the compartments disappears, the others will all equally expand to make good the loss of volume.

But, of course, the structure of the lung is far from being of so simple a nature, and although such a facile conception gives a rough idea of what happens when atelectasis takes place, it is far from being an accurate guide.

The lung is a highly complicated fibro-elastic structure, traversed by blood vessels and bronchial tubes. These elements have all a considerable mass, and they will not respond to increased tension in a manner at all comparable to a volume of pure gas.

The dynamics of atelectatic bronchiectasis can be more easily discussed by referring to the diagram opposite.

Let it be assumed that atelectasis of the right lower lobe has occurred, and that the shaded area in the diagram represents the collapsed portion.

Since the pleural space is occupied only by a thin

film of liquid and not by a gas, the surface of the lung must remain closely applied to the chest wall and diaphragm.

The stresses to which various parts of the lungs are subjected may now be considered.

At the level AB there will be no increased stress in a lateral or antero-posterior direction as the quantity of airbearing lung tissue filling the thoracic cavity at that level is the same after the atelectasis as before it.

It is far different at the level CD; here part of the lung has, as it were, disappeared. Since the surface of the lung cannot shrink away from the chest wall, either the chest wall must be pulled inwards, or what remains of the pulmonary tissue must be stretched in the lateral and antero-posterior directions in the horizontal plane at CD in order that it should occupy practically the same space as before, because, since the thoracic cage is comparatively rigid, the area cannot be much diminished by contraction of its walls.

In the lateral direction in the plane CD the degree of stress set up in the atelectatic area will vary according as the mediastinum is fixed or mobile. If the mediastinum is fixed the whole strain of compensation will be thrown on the pulmonary tissue in the atelectatic area. If there are any remaining air filled alveoli in this zone, their walls will be subjected to gross overdistension, and emphysema will result. Emphysema of the alveoli peripheral to the collapsed area will also result. Assuming that the bronchi in the atelectatic zone are plugged by mucus at their terminal parts, the portions proximal to the blockage will be patent and in communication with the atmosphere. They will therefore also be subjected to a distending force in the lateral direction. If the media-

stinum is mobile the heart will be drawn to the affected side, and the alveoli in the contralateral lung will become slightly distended, and take part of the strain; but it is obvious that although the lateral stress in the atelectatic area may be somewhat reduced in this manner, the mediastinal shift cannot abolish it. The movement of the heart, in fact, is an indication that a lateral stress is present, and greatest in the affected side. The lateral stress in the contralateral lung produced by the mediastinal shift, will clearly be slight.

In the antero-posterior direction in the horizontal plane CD the whole strain of compensation will fall on the pulmonary tissue in that part of the affected hemithorax, and the contralateral lung will be unaffected. The bronchi in the atelectatic area will once more be subjected to a distending stress.

Owing to the loss of airbearing pulmonary tissue in the atelectatic zone there will be increased tension in the line EF, but this will be somewhat diminished because the diaphragm is mobile, and can be pulled upwards. Again, although the elevation of the diaphragm reduces the strain, it cannot abolish it, and its appearance indicates that the increased tension is present. The stretching force will once more be felt most intensely in the atelectatic zone, as owing to the inertia of the pulmonary tissue it will progressively diminish in an upward direction. Distension of the alveoli in the middle and upper lobes will, however, afford some relief, and the stretching forces experienced by the atelectatic tissue will be less in the vertical than in the horizontal plane.

There will be no increased strain on the pulmonary tissue of the contralateral lung in a vertical direction along the line HG.

It will be seen that in collapse of the lower lobe, the strongest distending forces are in the horizontal plane, and as they are approximately at right angles to the bronchi, which in collapse are in the main vertically disposed, they will be particularly effective in causing dilatation.

In the case of middle lobe collapse, or collapse of the lingular process of the left upper lobe, similar considerations will apply. The increased stress will here be chiefly lateral as in the antero-posterior and vertical directions much of the tension will be relieved by emphysema of the upper and lower lobes. Since the middle lobe bronchi run roughly in a postero-anterior direction, vertical and lateral stresses would be most effective in causing dilatation. As even the lateral stress must be greatly reduced by compensatory emphysema, it would be expected that when the middle lobe is alone affected, the dilatation of the contained bronchi would be less than that seen in extensive collapse of the lower lobe. This is, generally speaking, borne out in practice.

In the upper lobe, the main stresses set up when atelectasis occurs will be in the horizontal plane. Expansion of the sound lung tissue below will partly relieve the vertical stresses. As the bronchi when the lobe is collapsed, are in the main vertically disposed, the forces most effective in dilating them will lie roughly in the horizontal plane, since they will act at right angles to the bronchial walls, and as has been noted, these are the strongest forces.

In the discussion above only the forces acting in an antero-posterior and lateral direction in the horizontal plane, and forces acting in the vertical planes have been considered. This selection of a few components of the

forces at work is, of course, only for convenience in description, and forces acting in all directions actually come into play.

The theory outlined above has been derived from the crude application of the principles of physics to the problem of pulmonary atelectasis.

What other evidence is there in support of the contention that the stresses arising when collapse takes place are concentrated in the manner described?

In the first place, there is the fact, pointed out by Erwin[15] among many others, that in atelectasis of the upper lobe, displacement of the trachea to the affected side is marked, whereas in lower lobe collapse, it is the shift of the heart which is the prominent radiological feature. If the increased tension in the affected lung were everywhere the same, these localised signs should not be observed.

Most striking of all perhaps, is the emphysema which is so constantly noted in the peri-atelectatic and atelectatic zone. Ogilvie,[6] as has been seen, observed this phenomenon in every one of his lobectomy specimens. Why should this condition occur only in and immediately adjacent to the collapsed zone? The obvious answer is that it is here that the strain is greatest. The alveoli in the other lobes of the lung are somewhat overdistended, but the strain on the remaining airbearing lung tissue in the atelectatic area and on the adjacent lung tissue is so great that the walls of the alveoli eventually rupture, and emphysema is the result. Even when no atelectasis is present, emphysema is often marked in a bronchiectatic area. In fact, the ring forms so often seen in straight x-rays of this condition were formerly mistaken for the

dilated bronchi themselves until bronchography demonstrated the error. This localisation to the affected lobe and adjacent lung tissue can only be satisfactorily explained on the basis of a previous atelectasis causing a concentration of stresses in the manner described above.

Lastly, there is the marked dilatation of the bronchi in the atelectatic lobe, which can only be satisfactorily explained by postulating a concentration of the distending forces in the affected area.

Warner[16] has also pointed out a contributory factor in causing the ectasia - the bronchi have largely lost their buffering surround of air-filled alveoli, and the dilating stress will be more directly applied to their walls through the atelectatic tissue.

The theory of the dynamics of atelectatic bronchiectasis, which has been advanced above, was evolved in seeking an explanation for the surprisingly slight reduction in the intrapleural pressure of a case of fairly extensive atelectasis of the left lower lobe. At that time, it was believed that the theory of the concentration of forces had not so far been expounded in the literature. It was later discovered that Andrus[11], in 1937, had come to exactly similar conclusions in an exhaustive study of dilating forces in bronchiectasis, though he had approached the problem from quite a different standpoint. The clinical feature which drew his attention to the concentration of stresses in an atelectatic area, was the constancy with which emphysema was found in bronchiectatic lobes whether there was atelectasis or not. He concluded that in all cases, atelectasis must have been present at one time, as this was the only satisfactory way of accounting for a localised dilating force powerful enough to cause the

emphysema and bronchiectasis. He suggested that the latter condition should be called "pulmonectasis" instead of "bronchiectasis."

The original problem, why the intrapleural pressure was not much reduced in a case in which there was extensive atelectasis of the left lower lobe with well marked dilatation of the contained bronchi, may now be considered in the light of the theory which has been worked out above.

It has been shown that when atelectasis occurs, the internal stress which is set up in the affected lung is largely concentrated in the collapsed area. As indicated in the diagram, the area of lung surface which tends to be most powerfully retracted from the wall of the thoracic cavity, is that lying between the horizontal plane at the upper limit of the collapse and the diaphragm. The lung surface applied to the diaphragm would also tend to be retracted, but to a lesser extent, as the elevation of the diaphragm would somewhat diminish the internal stress in a vertical direction. The forces tending to cause retraction of the remainder of the lung surface from the walls of the thoracic cavity, are, for reasons which have been already given, much weaker.

Since the pleural space is continuous, however, the intrapleural reading (which is really the measure of the gas pressure in a marginal pneumothorax) will only give an indication of the <u>mean</u> elastic tension of the whole lung, and this may not be much greater than normal. The fall in the intrapleural pressure may therefore not be marked, but when it is remembered that the increase in the elastic tension of the lung is concentrated mainly in the atelectatic zone in the manner described, the strength of the dilating forces in this area may be quite sufficient to

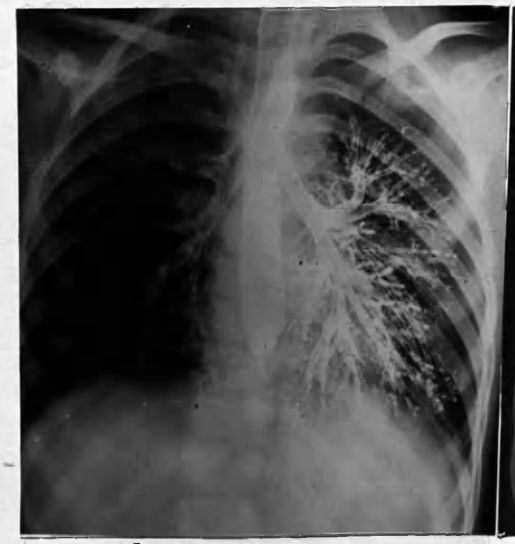
cause dilatation of the contained bronchi.

Extent of atelectasis required to produce bronchiectasis.

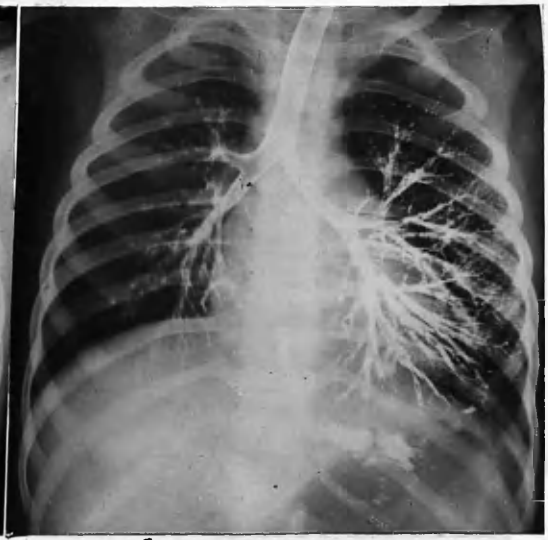
It has already been shown that when there is complete collapse of a lobe, or almost complete collapse of a lobe, dilatation of the contained bronchi will result, if the atelectasis is due to obstruction of the bronchioles or finer bronchi of the lobe. If the atelectasis is trifling in extent, however, it is obvious that no appreciable internal stress would be set up in the affected area of lung, as the normal pulmonary tissue lying between the area of collapse and the chest wall could easily expand and compensate for the lost space; hence bronchiectasis would not result.

The minimum extent of atelectasis necessary to produce bronchial dilatation is, judging from the literature, so far unknown, and there are so many variable factors to be taken into account in forming an estimate, that the problem is a formidable one. An investigation into the matter was beyond the scope of the present inquiry, but as a matter of interest, bronchography was carried out on ten patients in the whooping cough series who showed evidence of a moderate degree of lobar collapse. The meaning attached to the word "moderate" has already been explained in the first chapter, and some examples shown.

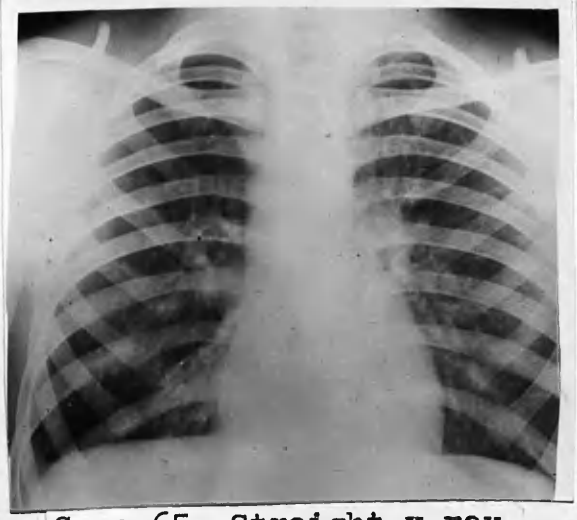
In six of the cases no definite bronchial dilatation could be discerned, but in four, slight cylindrical bronchiectasis was observed. These latter cases are noted below and bronchograms shown opposite. A straight x-ray illustrating the extent of the atelectasis in one case is demonstrated, but the straight x-rays of the other cases are not shown as the collapse was in the left lower lobe



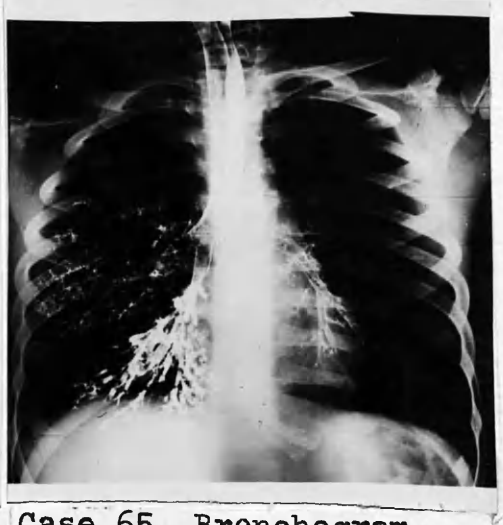
Case 63. Bronchogram showing very slight cylindrical dilatation of left lower lobe bronchi.



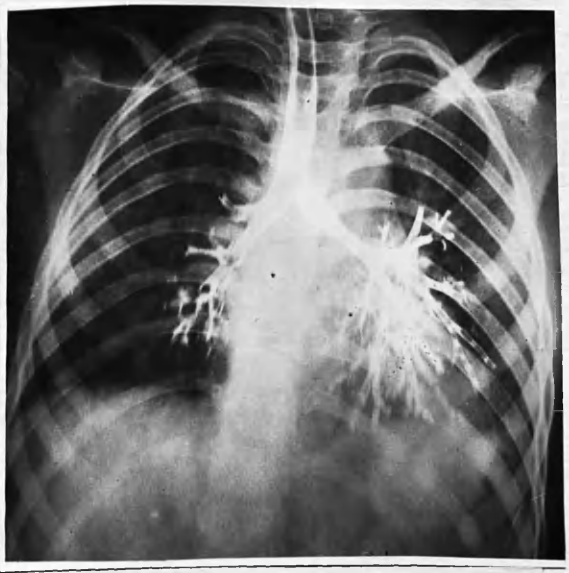
Case 64. Bronchogram showing very slight cylindrical dilatation of left lower lobe bronchi.



Case 65. Straight x-ray showing partial collapse of rt. lower lobe.



Case 65. Bronchogram showing that there is slight cylindrical dilatation of the rt. lower lobe bronchi.



Case 66. Bronchogram showing slight cylindrical dilatation of left lower lobe bronchi.

and the shadow behind the heart could not be satisfactorily reproduced.

Case 63.

Female, aged 3 years. Became ill on 17/2/47, and whooping cough was later diagnosed. In the fifth week of illness there was radiological evidence of a moderate degree of atelectasis of the left lower lobe. There were the usual signs of slight impairment of theP.N., prolonged, rather tubular expiration, and crepitations at the left base. Bronchography revealed very slight cylindrical dilatation of the bronchi in the left lower lobe. The atelectasis disappeared in four weeks from the time it was diagnosed. Subsequent bronchography revealed a normal bronchial tree. A bronchogram is shown opposite.

Case 64.

Female, aged 4 years. Became ill on 6/3/47 and was admitted to hospital with whooping cough a few days later. A moderate degree of atelectasis of the left lower lobe was noted radiographically in the third week of illness. Bronchography demonstrated very slight cylindrical bronchiectasis in the affected area. The atelectasis cleared up in four weeks from the time it was diagnosed, and subsequent bronchography revealed a normal bronchial tree. A bronchogram is shown opposite.

Case 65.

Male, aged 4 years. Became ill on 21/1/47, and whooping cough was later diagnosed. In the fourth week of illness, a straight P.A. x-ray showed an area of atelectasis in the right lower or middle lobe. A lateral view demonstrated that it was in the lower lobe. Bronchography revealed slight but definite cylindrical bronchiectasis of the right lower lobe bronchi. The atelectasis disappeared in five weeks from the time it was diagnosed, and subsequent bronchography showed a normal bronchial tree. A radiogram and bronchogram are shown opposite.

Case 66.

Male, aged 5 years. Became ill on 7/2/47, and whooping cough was later diagnosed. A moderate degree of atelectasis of the left lower lobe was diagnosed clinically and radiologically in the fourth week of illness. Bronchography revealed slight but definite cylindrical bronchiectasis in the left lower lobe. The collapse cleared up in five weeks from the time it was diagnosed and subsequent bronchography revealed a normal bronchial tree. A bronchogram is shown opposite.

SECTION 2.

A TEST FOR DISTINGUISHING POTENTIALLY REVERSIBLE FROM PERMANENT ATELECTATIC BRONCHIECTASIS.

In view of the theory of the dynamics of atelectatic bronchiectasis which was eventually evolved, the idea that a study of intrapleural pressures would be of practical value in determining whether or not atelectatic bronchiectasis was reversible had to be abandoned. It is considered, nevertheless, that the underlying principles were correct; that is to say that whereas the isolateral intrapleural pressure would be lowered if the pulmonary and bronchial elements subjected to the abnormal stresses still retained their elasticity, it would be normal if their elasticity had disappeared due to prolonged overstretching alone or to prolonged overstretching with the additional factor of infection. A lowered isolateral intrapleural pressure would indicate the potential ability of the bronchi to revert to their normal calibre in the event of the atelectasis clearing up, while a normal intrapleural pressure would indicate that the bronchial dilatation had become permanent.

It has been shown above, however, that owing to localisation of the internal stresses set up by an atelectasis, bronchial dilatation may be produced without a marked lowering of the intrapleural pressure; in fact, if the intrapleural pressure on the affected side were ascertained it might be discovered to be within normal limits, which, as has also been demonstrated, appear to be somewhat wide. In addition, as the atelectasis would, in the vast majority of cases, be present when the patient was

first examined, his normal intrapleural pressure would not be known.

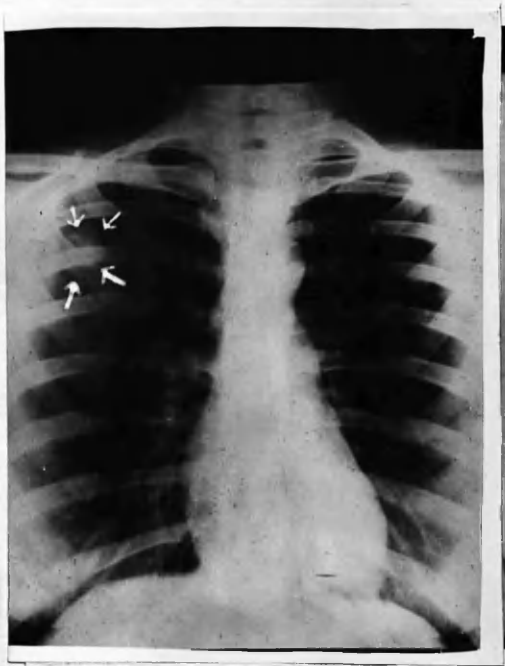
Simultaneous readings of the intrapleural pressures on both sides of the chest, as has been suggested, might help to solve this problem, because the internal tension of the contralateral lung is never raised, and consequently, the intrapleural pressure of the contralateral hemithorax is never lowered, to anything like the same degree as on the affected side. A difference in the two readings would be theoretically significant. However, as has been seen, the fall in intrapleural pressure on the affected side, even in a recent case of atelectasis, may not be great, and the difference in the two sides might therefore be too small to be of practical value. The instruments used for artificial pneumothorax work, and for recording intrapleural pressures are commonly somewhat crude. If the same instrument were used to take the pressure on one side immediately after taking it on the other, it would be impossible to ensure that the respiratory excursion on both occasions had been exactly the same. If different instruments were used, and the pressures recorded simultaneously, the instrumental error would probably be fairly large. Moreover, the position of the patient would have to be such that both sides of the chest moved equally, and the site chosen for insertion of the needle would have to be the same. In the case of children, it would be extremely difficult to ensure accuracy. Only in instances, therefore, where the difference in the intrapleural pressures was marked, would the procedure be of value; it might then be considered that the tissues subjected to the abnormal stresses still retained their elasticity. On the other hand, because of the possible fallacies considered

above, it would not be safe to assume in cases where the intrapleural pressures were much the same, that elasticity of the tissues in question had been lost.

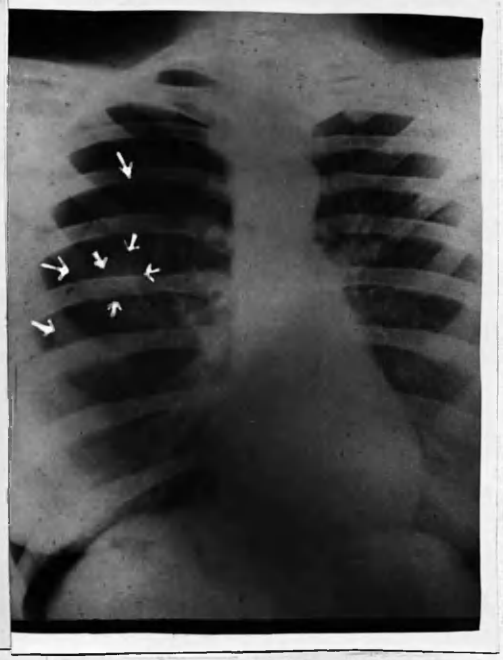
Even in cases where the difference in intrapleural pressures was unequivocal, the conclusion that the tissues in the atelectatic area still retained their elasticity might not in all instances be accurate. If, for example, the collapse involved a very large portion of the lung, compensation might not be complete even though the tissues had been stretched out for a period long enough permanently to impair their elasticity, and hence the intrapleural pressure would still be low; or to put the matter in another way, the equilibrium attained following the atelectasis might still be unstable even after the tissues in the affected zone had been overdistended sufficiently permanently to impair their elasticity, and the instability in the equilibrium would be manifested by a low intrapleural pressure which might be erroneously interpreted.

Again, it is conceivable that the pulmonary tissue in the atelectatic zone might retain its elasticity and cause a lowering of the intrapleural pressure even though the elasticity of the bronchial walls had permanently disappeared. Probability is lent to this speculation by the fact that the bronchi in an atelectasis are peculiarly exposed to the risk of infection, since expulsion of secretion from the lumen is not so easy as under normal circumstances for reasons which will be mentioned later.

These considerations are sufficient to show that the level of the intrapleural pressure is an unsatisfactory criterion from a practical point of view as to whether bronchiectasis is reversible or not, and there are others which indicate that the behaviour of the intrapleural



Case 67. Focus of tuberculosis indicated by arrow.



Case 67. Right A.P. present. Outline of lung and tuberculous focus indicated by arrows.

pressure is not yet always predictable.

It has already been pointed out that the intrapleural pressure depends on the elastic tension of the lung, and Blades[6] states, "It may be assumed that the difference between expiratory and inspiratory intrapleural pressures approximates the distending force applied to the lung during inhalation. Pulmonary distensibility (elasticity), might therefore be expressed as a ratio of the volume of air inspired to the distending force or change in intrapleural pressure."

Christie and McIntosh[17], in fact, measured pulmonary elasticity by simultaneous estimations of the tidal air volume and intrapleural pressures.

It must not be forgotten, however, that since the lung is composed of living tissue, its reactions to changing circumstances cannot yet be guaged with mathematical precision. The elasticity of the lung, and consequently the intrapleural pressure, may vary from time to time in the same person, and when a pneumothorax has been induced, the relationship between the intrapleural pressure and the pulmonary elasticity is much more complex than appears at first sight. The following case illustrates the point.

Case 67.

Female, aged 19 years. Admitted to Ruchill Sanatorium on 1/2/47, with an early, well defined, tuberculous focus in the right upper lobe, too small to interfere to any appreciable extent with the elasticity of the lung as a whole. Radiograms are shown opposite. A right artificial pneumothorax was induced on 9/2/47. The induction readings were -7,-4 cms. of water, and after 250 c.c. of air had been introduced into the pleural cavity, the readings were -6,-3 cms. of water. Artificial pneumothorax treatment was continued, and on 20/2/47 the lung was relaxed to the extent shown in the x-ray opposite. Readings taken after the x-ray on the same day were -8,-5 cms. of water, and after 500 c.c. of air had been introduced, -3,-1 cms. of water.

It is seen, therefore, that the induction readings in the case just outlined, were -7,-4 cms. of water, and when the lung was partially relaxed the readings were -8,-5 cms. of water. One would have expected that since the lung was occupying a smaller volume, its internal elastic tension would have been less, not more, than it was at induction, and would only have regained its original level when the air had been absorbed from the pleural cavity and the lung had once more been stretched out to occupy the whole hemithorax. It would appear, therefore, that either the elastic tension of the lung adjusts itself after a time to more or less its original level when the lung is allowed to occupy a smaller volume, or the diffusion of gases from the pleural cavity and other complicated factors make the intrapleural pressure no longer a true measure of the pulmonary elasticity. The problem is obviously extremely complex. Many findings similar to the above are encountered in artificial pneumothorax work.

It is also difficult to explain the not infrequent experience that two or three hundred c.c. of air can be introduced into the pleural cavity without appreciably lowering the intrapleural pressure, though insensitivity of the instruments used may partly account for the phenomenon.

Again, on rare occasions artificial pneumothorax treatment for cases of tuberculosis has to be abandoned because the lung obstinately and illogically insists on "coming up."

Therefore, though in general the behaviour of the intrapleural pressure and the elastic tension of the lung can be predicted with some confidence, there are many extremely complex problems yet to be solved.

Although, therefore, the study of intrapleural pressures led to a more exact conception of the dynamics of atelectasis, it appeared to be a difficult approach to the problem of finding a method of determining whether or not atelectatic bronchiectasis was potentially reversible.

A very simple, and once it had been thought of, a very obvious test, was eventually evolved. This was to visualise the bronchi in the atelectatic area by introducing iodised oil, induce an artificial pneumothorax in the affected hemithorax, once more carry out bronchography, and then ascertain if the dilatation of the bronchi in the collapsed area had been diminished. The introduction of air into the pleural cavity, by allowing the surface of the lung to retract from the chest wall, would diminish the internal pulmonary tension. If the bronchi retained their elasticity, therefore, the reduction of the abnormal tension to which they had been exposed, would permit them to revert to their normal calibre; if, on the other hand, their elasticity had been impaired, they would remain dilated.

The ideal volume of air to introduce into the pleural cavity would appear at first sight to be one equivalent to that lost from the alveoli on account of the atelectasis. On reflection, however, it is seen that this would not lead to an exact counterbalancing of the tensions set up by the collapse. It has been shown above that when atelectasis occurs, the increased internal stress is largely localised to the collapsed area of the lung, and therefore it would be essential selectively to relax that portion of lung. This could not be done merely by introducing air into the pleural cavity, as the non-atelectatic part of the lung would obviously also diminish in volume, though

not to the same extent as the collapsed portion. Thus, even if the necessary calculations could be made, the introduction into the intrapleural space of a volume of air equal to that of the air absorbed from the alveoli would not necessarily exactly restore the original balance of pulmonary tensions, though it would probably come near to doing so.

The essence of the matter is, however, that if the lung, including the atelectatic area, were thoroughly relaxed, a marked reduction in the bronchial dilatation would be expected if bronchial elasticity were unimpaired.

It appeared that the test could be satisfactorily conducted on this empirical basis. If an artificial pneumothorax of any extent were present, and the bronchi remained dilated, it would be extremely unlikely that they would revert to their former calibre in the event of the atelectasis clearing up. Of course, if a very large artificial pneumothorax were induced, no doubt some diminution in the calibre of permanently damaged bronchi might be expected, but the change would presumably not be anything like the dramatic alteration that could be anticipated where the elasticity was unimpaired.

In order to discover whether the test was of a practical nature, and whether the principles underlying it were correct, it was necessary to study the effects of an artificial pneumothorax on the bronchial calibre in cases of atelectasis in which it was known that the bronchi retained their elasticity, and in cases of atelectasis in which it was known that the bronchi had lost their elasticity.

Evidence as to the state of the bronchial elasticity when the artificial pneumothorax was induced might be of first class or second class nature.

If it could be shown at a subsequent period that the artificial pneumothorax had absorbed, the atelectatic area of lung had re-expanded, and the dilated bronchi had regained their former calibre, then it would be known for certain that when the artificial pneumothorax had been induced, the bronchial walls were elastic.

Similarly, if, when an artificial pneumothorax had been induced, lobectomy were performed within a short period thereafter, and fibrous replacement of the myo-elastic tissue of the bronchial walls demonstrated, it would be first class evidence that the bronchial walls had lost their elasticity when the artificial pneumothorax was present.

The evidence as to the state of the bronchial walls at the time when the artificial pneumothorax was induced could be of a less direct nature, but still good. If the time of onset of an atelectasis were known, and an artificial pneumothorax were performed within a short period thereafter, there would be a very strong presumption that the bronchial walls still retained their elasticity, providing that infection of the atelectatic system was slight or absent. Again, if an artificial pneumothorax were induced in a case with a history of persistent productive cough for years, it might safely be assumed that permanent damage to the bronchial walls was present, and this deduction would be strengthened if subsequent observation demonstrated that the bronchiectasis was permanent.

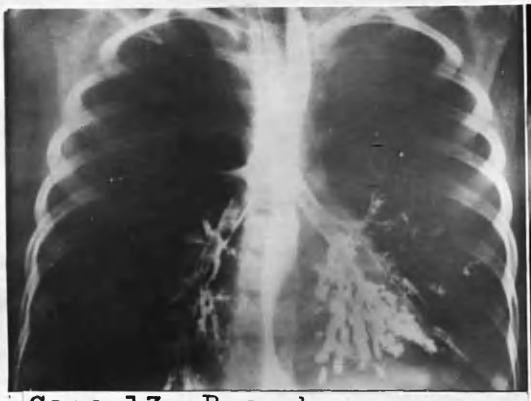
It is therefore seen that means are available for ascertaining if calibre changes in the dilated bronchi in response to the induction of an artificial pneumothorax are a reliable index of the elasticity of their walls.

Evidence of the nature described above will now be

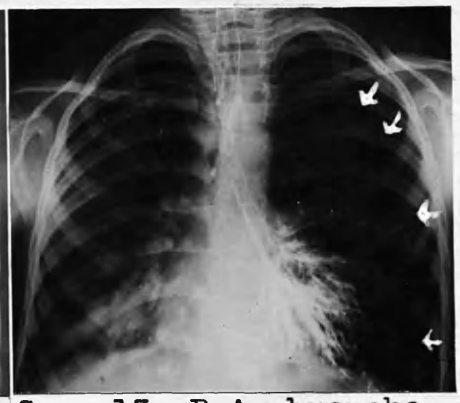
brought forward to show that if the dilated bronchi in an atelectasis retain their elasticity a marked reduction in calibre is seen if an artificial pneumothorax is induced.

Four cases of well marked reversible atelectatic bronchiectasis occurred in the whooping cough series, and in three of these an artificial pneumothorax was induced and maintained as a therapeutic measure. The importance of performing bronchography after the artificial pneumothorax had been induced was unfortunately not appreciated in the first two of the latter three cases. It was only by the time the third case came to be treated that the full value of the procedure, in this, and in other connexions, was realised.

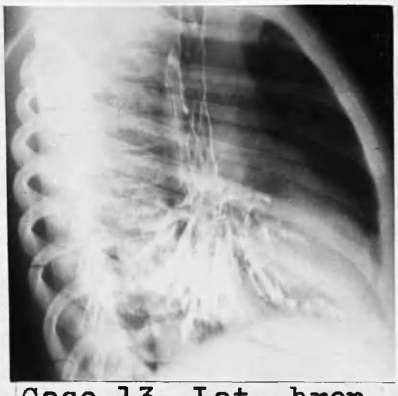
As this case will be fully discussed when artificial pneumothorax as a therapeutic agent is considered (Case 13, p.249), it is only necessary to give here such details as are necessary for the present purpose. The patient, a boy aged five years, became ill on 26/3/47 and was admitted to hospital with whooping cough on 9/4/47. Atelectasis and bronchiectasis of the left lower lobe were diagnosed clinically. A radiogram on 18/4/47 confirmed the finding that there was extensive collapse of the left lower lobe, and a bronchogram on 13/5/47 demonstrated marked dilatation of the contained bronchi. An artificial pneumothorax was induced on 21/5/47. Bronchography was carried out on 7/6/47 under general anaesthesia, but the bronchogram was unsatisfactory. The procedure was repeated under local anaesthesia on 26/6/47, and the affected bronchi were clearly outlined; it was observed that the dilatation had almost, though not quite, disappeared. A radiogram on 4/8/47 showed that the atelectasis in the partially relaxed lung was almost as marked as before, but another on 11/8/47



Case 13. Bronchogram, 13/5/47, showing collapse of left lower lobe with marked dilatation of the contained bronchi.



Case 13. P.A. bronchogram, 26/6/47, showing the presence of a left A.P. of moderate extent. The outline of the left lung is indicated by arrows. There has been a dramatic reduction in the calibre of the left lower lobe bronchi.



Case 13. Lat. bronchogram, 26/6/47, confirming the fact that the bronchial dilatation in the left lower lobe has almost disappeared.

demonstrated that very considerable re-expansion of the collapsed area had taken place. The artificial pneumothorax was allowed to absorb, and subsequent bronchography demonstrated a normal bronchial tree. Bronchograms are shown opposite.

The fact that the bronchiectasis was eventually shown to be reversible proves that the bronchial walls had never lost their elasticity. A comparison of the bronchograms before and after artificial pneumothorax demonstrates that when the bronchi in an atelectasis are in this condition, a relaxation of the abnormal distending stresses to which they are subjected will produce a striking reduction in the bronchial calibre.

The bronchial dilatation did not, however, completely disappear with an artificial pneumothorax of the extent seen in the bronchogram. It appears that there could be two explanations for this; either the artificial pneumothorax was not large enough to secure a complete negation of the abnormal stresses set up by the atelectasis, or alternatively the artificial pneumothorax had successfully achieved this, but the bronchial elasticity was slightly though temporarily impaired. The first possibility could have been explored by increasing the extent of the artificial pneumothorax and visualising the bronchi, but for reasons which will be explained in the section dealing with artificial pneumothorax as a therapeutic agent, this was not done. No categorical statement can therefore be made, but as infection in the atelectatic system was severe in this case, it is believed that the bronchial elasticity, while largely retained, may have been temporarily slightly impaired.

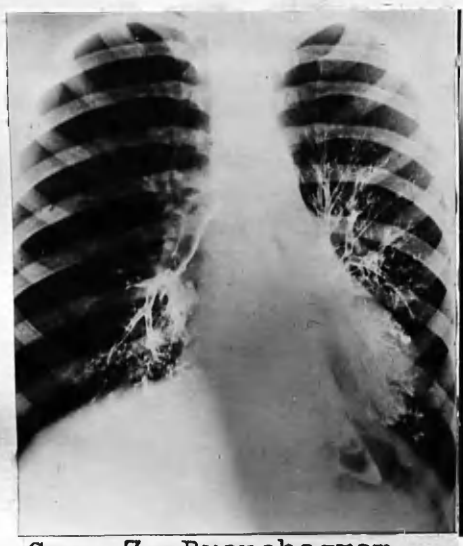
Since the elasticity of the bronchi was proved in

this case by the eventual complete disappearance of the bronchiectasis, it afforded first class evidence that the dilatation of elastic bronchi in an atelectatic area is markedly reduced by the induction of an artificial pneumothorax. It was obviously desirable to corroborate this finding by reference to the work of other authors, but although Franklin[18], Fleischner[19], Lander[4], Blades[20], and Ogilvie[8] among others, demonstrated cases of proven reversible atelectatic bronchiectasis, no instance was discovered in which an artificial pneumothorax was performed while the atelectasis was present, and the effects of the procedure on the bronchial calibre ascertained by bronchography.

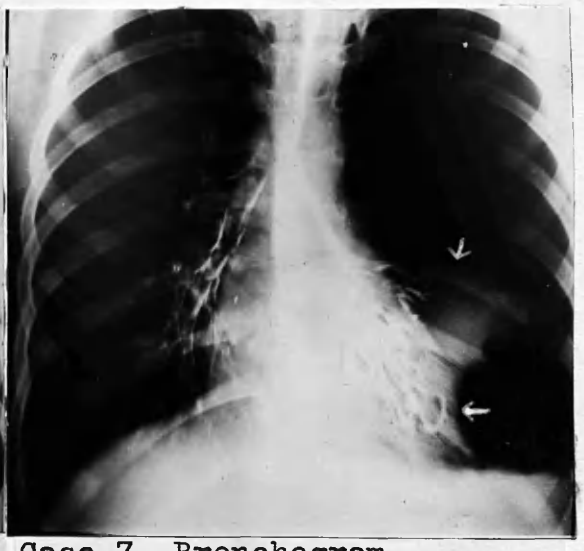
The next best thing was to study cases of atelectatic bronchiectasis in which an artificial pneumothorax had been induced shortly after the occurrence of the atelectasis; the elasticity of the bronchi could then be presumed. Lander[4] demonstrated two such examples in 1938.

One was a case of tuberculosis in which atelectasis of a lobe occurred during artificial pneumothorax treatment. Immediately after the onset of the collapse, it was found that the intrapleural pressure on the affected side was markedly lowered, indicating increased internal tension of the lung. The bronchi in the atelectatic area were shown to be markedly dilated. The artificial pneumothorax was increased in extent until the lung was thoroughly relaxed, and bronchography was again performed. The dilatation of the bronchi was seen to have practically disappeared.

The other was a case of complete collapse of the left lung, in which bronchography demonstrated a gross dilatation of the bronchi. After an artificial pneumothorax had been performed, the bronchi were again visualised;



Case 7. Bronchogram, 11/3/47. Gross bronchial dilatation in left lower lobe.



Case 7. Bronchogram, 24/8/47. Complete relaxation of left lung by artificial pneumothorax. Gross bronchial dilatation still present.

they had almost reverted to their normal calibre.

Theoretical considerations, therefore, suggest that if the dilated bronchi in an atelectatic area retain their elasticity the induction of an artificial pneumothorax will effect a marked reduction of the ectasia, and such practical experience as there is points to the same conclusion.

The effects of an artificial pneumothorax on the calibre of the bronchi contained in an atelectatic portion of lung when elasticity has been lost is illustrated by the following cases.

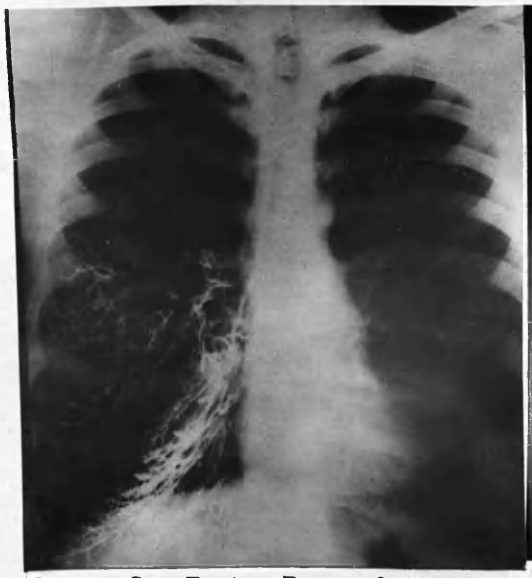
Case 7.

Female, aged 5 years, admitted to Ruchill Sanatorium on 10/1/47. There was a history of persistent cough following an attack of whooping cough at the age of three years. For several months before admission it had become very troublesome, and was associated with the production of a fair quantity of sputum. Physical examination revealed impairment of the P.N., and tubularity of the R.M. over a triangular area at the left base. Crepitations could be heard in this zone, and also over the lingula. Straight x-ray showed extensive collapse of the left lower lobe. Bronchography on 11/3/47 revealed gross dilatation of the bronchi in the collapsed area, and also slight dilatation of the bronchi in the lingula. The bronchi of the right lung were visualised on 4/4/47, and found to be normal.

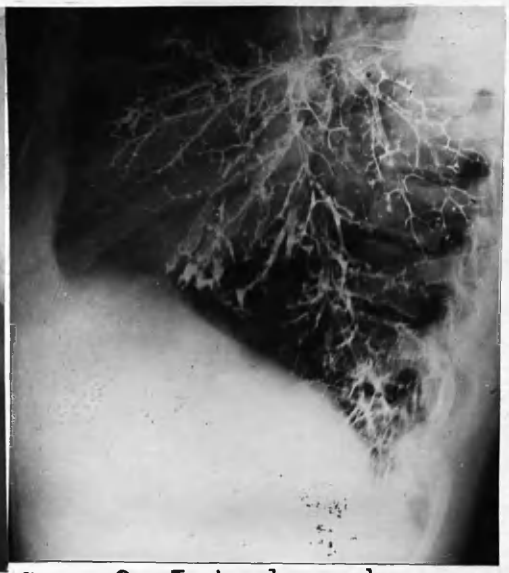
A left artificial pneumothorax was induced on 4/4/47. The induction readings were -5,-3 cms. of water, and after 200 c.c. of air had been introduced, -3,-1 cms. of water. The A.P. was maintained. On 20/6/47, x-ray revealed that the lung was roughly half relaxed, but bronchography showed no apparent reduction in the bronchial dilatation.

The lung was subsequently relaxed completely, but bronchography on 24/8/47 demonstrated that the bronchiectasis was little if at all reduced. The A.P. was allowed to absorb, and the left lower lobe and lingula were removed at Mearnskirk Hospital a few weeks later.

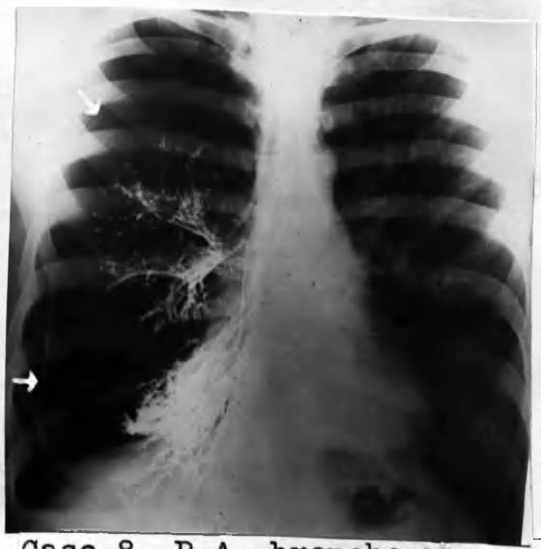
Examination of the left lower lobe showed that the bronchi were grossly dilated, and contained infected secretion. Their walls were fibrous and inelastic. There were also permanent changes in the walls of the bronchi of the lingula, though dilatation was not marked. Bronchograms are shown opposite.



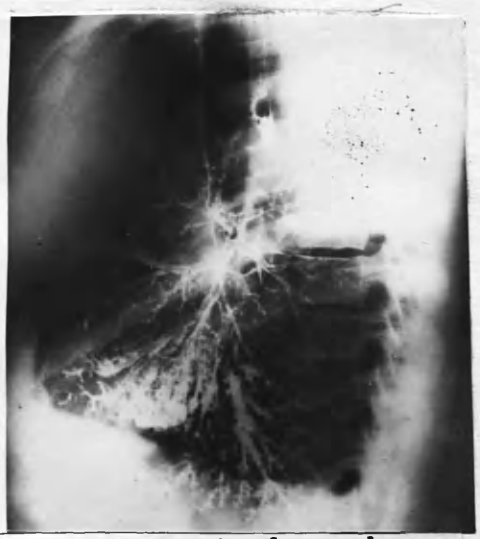
Case 8. P.A. Bronchogram, 25/4/48, showing cylindrical dilatation of bronchi in partially collapsed rt. lower lobe.



Case 8. Lat. bronchogram, 25/4/48, showing that the atelectasis and bronchial dilatation chiefly affects the ant. basal segment of the rt. lower lobe.



Case 8. P.A. bronchogram, 20/6/48, demonstrating extent of rt. A.P. present.



Case 8. Lat. bronchogram, 20/6/48, showing that the bronchial dilatation has not been reduced by the relaxation of the rt. lung.

Case 8.

Male, aged 25 years, admitted to Ruchill Hospital on 22/4/47. The history was vague, but for some months he had noted some breathlessness on exertion, and had been troubled with a slight productive cough. He could not remember any illnesses in childhood.

Physical examination revealed impaired P.N., and diminished V.R. amd R.M. at the right base. On some occasions, a few crepitations could be heard in the same area.

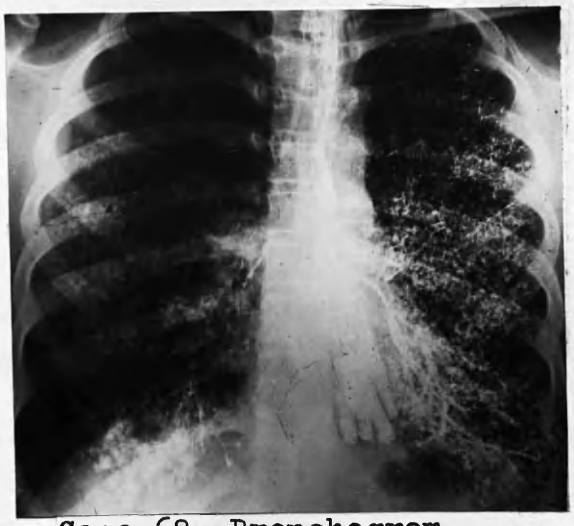
Straight x-ray on 17/3/47, before admission, had already revealed that there was extensive collapse of the right lower lobe. Bronchograms on 25/4/47, reproduced opposite, showed collapse of the right lower lobe with cylindrical dilatation of the contained bronchi.

A right sided artificial pneumothorax was induced on 12/5/47. The induction reading was -6,-4 cms. of water, and after 300 c.c. of air had been introduced, -4,-2 cms. of water. An x-ray on 6/6/47 showed that by this time the lung was half relaxed. Bronchography was carried out on 20/6/47. The bronchograms shown opposite demonstrate the extent of the A.P. at the time, and also that the calibre of the affected bronchi had not been reduced by its presence. The A.P. was continued until 7/10/47 when it was allowed to absorb. The right lower lobe was excised at Mearnskirk Hospital on 23/10/47. There was cylindrical dilatation of the bronchi,and their walls were fibrous and inelastic.

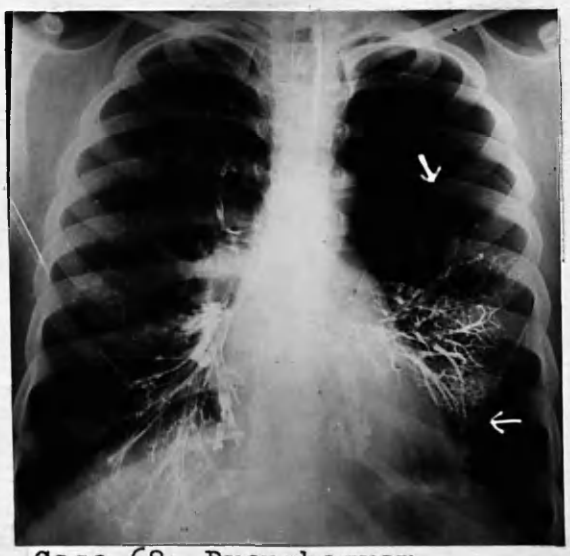
Case 68.

Female, aged 12 years, admitted to Ruchill Sanatorium on 16/6/47. There was a history of persistent productive cough for two years at least. She had whooping cough and measles in childhood, but the mother could not remember if the cough was a sequel to either of these diseases. Physical examination revealed impairment of P.N., and tubular breathing at the left base, and moist crepitations could be heard in the same area. Straight x-ray showed atelectasis of the left lower lobe, and bronchography on 19/6/47 demonstrated that the contained bronchi were dilated. A left artificial pnemothorax was induced on 2/7/47. Induction readings were -7,-5... 300 c.c. of air.. -6,-3.

It is interesting to note in view of what was previously said regarding the impossibility of looking on intrapleural readings as a measurement of the elastic tension of the lung when an A.P. is present that the readings two days later were -7,-5... 300 c.c. of air...



Case 68. Bronchogram, 19/6/47, showing atelectasis and bronchiectasis of left lower lobe.



Case 68. Bronchogram, 1/8/47, showing large left A.P. with selective relaxation of left lower lobe owing to adhesions in mid-zone. Considerable bronchial dilatation persists in the left lower lobe, but its extent has been reduced.

-5,-2, and in a further two days -7,-4... 400 c.c. of air... -4,-1.

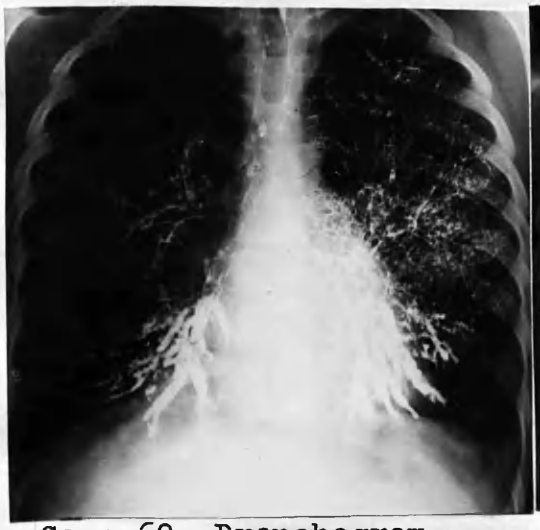
On 1/8/47, following a refill the previous day, bronchography was performed. The readings at the refill were, -6,-3... 400 c.c. of air...-2,+1. The bronchogram showed that there had been a fair reduction of the bronchial calibre, probably because of the large extent of the A.P., and a largely selective relaxation of the affected area due to adhesions holding out the sound part of the lung. Unfortunately, in the print of the bronchogram shown opposite there appears to have been a marked reduction in the bronchial calibre. This misleading appearance is due to two factors; the bronchi were not so completely filled on the second occasion, and photographic reproduction of the differentiation between the bronchi and heart shadow was made still more difficult by an unfortunate choice of the degree of x-ray penetration. Had the difficulties of photographic reproduction been appreciated at the time, a repeat bronchogram would have been performed, particularly as the lateral view was blurred.

However, in the bronchogram itself it was plain that even in the presence of a pneumothorax of great extent, considerable bronchial dilatation persisted, and in view of this fact, coupled with the long duration of the symptoms, the bronchiectasis was deemed permanent. The A.P. was allowed to absorb, and the left lower lobe was removed at Mearnskirk Hospital in the following month. The contained bronchi were found to be dilated, and their walls had suffered fibrous replacement.

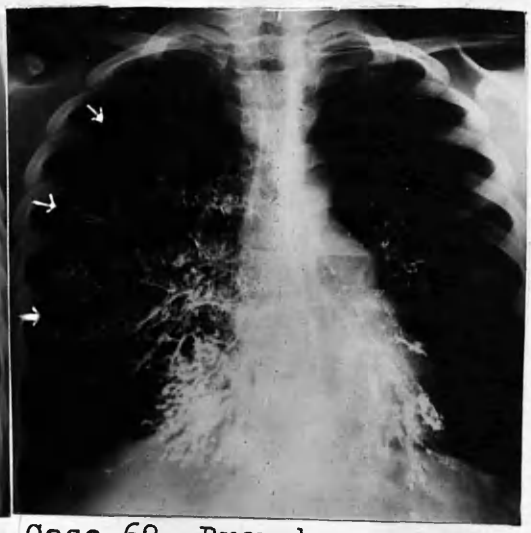
Case 69.

Male, aged 13 years, admitted to Ruchill Sanatorium on 10/3/47. There was a history of productive cough of some years duration. The patient had had measles and whooping cough in childhood, but no clear cut relationship between either of these diseases and the onset of the cough was recalled by the mother. On physical examination, at both bases the P.N. was impaired, and the R.M. rather tubular and accompanied by numerous moist crepitations. Straight x-ray on 11/3/47 showed collapse of both lower lobes, and bronchography on 28/3/47 demonstrated cylindrical bronchiectasis in the affected areas.

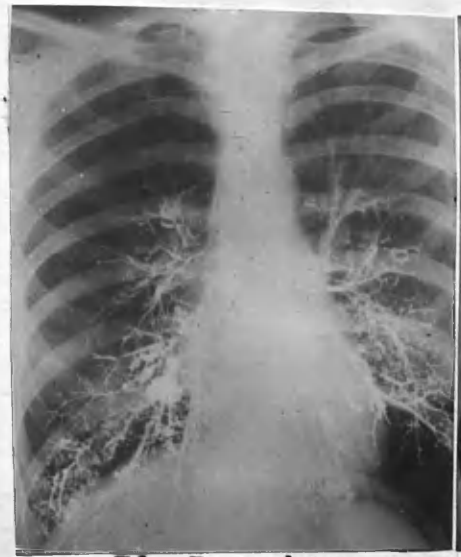
An artificial pneumothorax was induced on the right side on 8/8/47. The induction readings were -5,-3 cms. of water... 250 c.c. of air... -3,-1 cms. of water. A bronchogram taken on 1/9/47 showed that the bronchial dilatation was not appreciably reduced. An A.P. was also



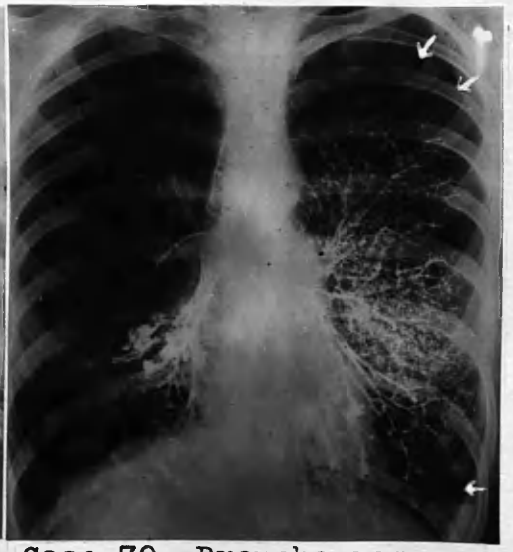
Case 69. Bronchogram, 28/3/47, showing atelectasis and bronchiectasis of both lower lobes.



Case 69. Bronchogram, 1/9/47, showing presence of R.A.P.. The outline of rt. lung is indicated by arrows. No reduction in calibre of rt. lower lobe bronchi.



Case 70. Bronchogram, 18/6/47, showing collapse of left lower lobe with dilatation of contained bronchi. Rt. middle lobe bronchiectasis also present.



Case 70. Bronchogram, 25/7/47. Small left A.P. present. The outline of the left lung is indicated by arrows. No reduction in calibre of left lower lobe bronchi.

induced on the left side with similar results. Bronchograms are shown opposite.

In the reproduction of the bronchogram before the induction of A.P., detail has been lost, and it seems at first sight as if there were two grossly enlarged bronchi, but it will be noted on closer inspection that in both instances the appearance is due to the partial superimposition of one bronchus on another.

Lobectomy was not carried out in this case, and therefore actual inspection of the affected bronchi was not possible. It can scarcely be doubted, however, that the indication from the A.P. that the condition was irreversible, was correct, not only in view of the prolonged symptoms, but also because a year previous to admission, bronchography at Ruchill had demonstrated bilateral basal atelectatic bronchiectasis.

Case 70.

Female, aged 28 years, sent from a tuberculosis clinic to Ruchill Sanatorium on 19/5/47 for bronchography. Straight x-ray had revealed collapse of the left lower lobe. There was a history of productive cough for over three years. The patient could not remember what ailments she had had in childhood. Physical examination revealed impaired P.N., and rather tubular R.M., at the left base. There were moist crepitations in the same area, and also over the right middle lobe.

The patient was admitted to Ruchill Sanatorium on 12/6/47. A bronchogram on 18/6/47 revealed dilatation of the bronchi in the collapsed area of the left lower lobe, and bronchiectasis of the right middle lobe. A left artificial pneumothorax was induced on 20/6/47. The induction readings were -8,-4 cms. of water, and after 250 c.c. of air had been introduced, -4,-2 cms. of water. On 25/7/47 after a refill at which the readings were -6,-4.. 300 c.c. of air... -4,-2, bronchography was again performed. A small A.P. was present, but no reduction in the calibre of the affected bronchi could be discerned. Since the elasticity of the bronchi appeared to have gone, it was considered that A.P. was useless as a therapeutic agent, and the woman was offered lobectomy. She declined. In view of the bilateral nature of the disease, and the age of the patient, it would, in any case, have been a difficult procedure. The A.P. was, however, maintained for a further three months, but as the productive cough was as much in evidence as ever, the A.P. was finally abandoned.

The loss of bronchial elasticity indicated by the A.P. was not confirmed by lobectomy, therefore, but in view of the long history of the illness, little doubt can be entertained that the bronchiectasis was of a permanent nature. Bronchograms are shown opposite page 233.

The evidence above may now be summarised.

In one case of atelectatic bronchiectasis it was ascertained that the dilated bronchi in the collapsed zone had not lost their elasticity, because on re-expansion of the affected lobe the bronchi reverted to their normal calibre. When an artificial pneumothorax of moderate extent was induced while the atelectasis was present, a striking reduction in the bronchial dilatation was noted. In two cases of atelectatic bronchiectasis shown by Lander,[4] where elasticity of the bronchi could be presumed because of the recent onset of the collapse, the induction of an artificial pneumothorax produced a similar effect.

In three cases in which an artificial pneumothorax was induced, subsequent lobectomy findings demonstrated that the bronchi must have been inelastic at the time the procedure was carried out; in two of the cases, the induction of the artificial pneumothorax produced no appreciable reduction of the bronchial calibre, and in the third case only a moderate reduction, even though the artificial pneumothorax was great in extent. In two further cases in which it could be presumed from the long duration of symptoms that the bronchi had lost their elasticity, the induction of an artificial pneumothorax had no appreciable effect on the bronchial calibre.

It is considered, therefore that the response of the dilated bronchi in an atelectatic area to the induction of an artificial pneumothorax furnishes a useful guide as to whether the bronchiectasis is potentially reversible or

not. If the bronchial dilatation were not markedly reduced in the presence of an artificial pneumothorax, it would seem incredible that the bronchi could still be elastic and capable of reverting to normal even if the atelectasis cleared up. In this event, there appears to be little doubt that the test would be completely reliable.

On the other hand, could the induction of an artificial pneumothorax produce a diminution in the calibre of bronchi, the elasticity of which was permanently impaired? No doubt if the artificial pneumothorax were large in extent, and the bronchial walls had not suffered much fibrous replacement, a reduction in the bronchial calibre could be expected, but if the artificial pneumothorax were small in extent, it appears most improbable that a diminution in calibre at all comparable to that seen in potentially reversible cases would be observed. Until more experience has accumulated, it is obviously wise to reserve judgement on this matter, but it appears safe to infer that if no diminution in calibre of the bronchi in an atelectatic portion of lung results from the induction of an artificial pneumothorax, then the bronchiectasis is irreversible.

It has been mentioned above that no case of reversible bronchiectasis was encountered in the literature in which an artificial pneumothorax had been induced, and the difference in bronchial calibre before and after the procedure ascertained.

Hennell,[21] however, in 1946, published the results of artificial pneumothorax therapy in six cases of "early bronchiectasis," and discussed four of the cases. His material and conclusions will be examined in the next section, but as the findings in one of his cases might appear on a hasty reading to conflict with the theory

advanced above, it will be considered here.

The patient was a boy of fifteen years who was admitted to hospital with a history of frequent "colds" (the italics are Hennell's), for many years. Three months before admission to hospital he had had a febrile episode of five days duration with a temperature of 104 F. on one occasion. He also had a severe productive cough which had cleared up by the time he was admitted. Postural drainage, however, induced coughing, and the production of substantial quantities of pus on each occasion. Atelectatic bronchiectasis of the right lower lobe was diagnosed on the clinical findings and straight x-ray. Artificial pneumothorax was induced three weeks after admission. While a "low tension" artificial pneumothorax was present, bronchography was carried out. The outline of the lung cannot be discerned in the reproduction appearing in the article, but the presence of an artificial pneumothorax can be inferred from the appearance of a puddle of fluid in the costo-phrenic angle. The bronchi in the atelectatic right lower lobe, however, can be seen to be still markedly dilated. As a bronchogram was apparently not taken before the artificial pneumothorax was induced, it cannot be ascertained whether the procedure reduced the bronchial dilatation or not. Nevertheless, as dilatation is still obvious, it might be inferred from what has been said above that the bronchi had lost their elasticity. Hennell goes on to say that the artificial pneumothorax was continued for seven weeks, and then allowed to absorb. The lung was subsequently seen to have "fully re-expanded." Presumably he means by this that not only had the artificial pneumothorax disappeared, but the atelectasis as well. The boy was observed for a further three months

during which time he was "entirely well" and symptom free. There is no mention, however, of a final bronchogram. From this account, and the fact that the author speaks of the success of artificial pneumothorax therapy in this case, it might be imagined that the bronchiectasis had eventually disappeared, and the persistence of marked dilatation in the presence of an artificial pneumothorax could have given the erroneous impression that the bronchi were permanently damaged. It appears, however, that that impression would have been perfectly correct, for Hennell goes on to say, "Admittedly, pneumothorax therapy does not relieve the patient of the dilated bronchi. However, as has been shown, it can control the infection in early bronchiectasis so that the patient can be restored to normal health."

The fact that the bronchiectasis was permanent in this case could, it seems, have been inferred from the past history. As the boy had had frequent "colds" for many years, it is surprising that Hennell looked on the case as one of "early" bronchiectasis. Possibly he regarded the condition as originating in an atelectasis which had occurred during the febrile attack prior to admission. There is good reason to suppose that the atelectasis did occur at that time, because the induction readings for the artificial pneumothorax were very negative (-18,-10 cms. of water), suggesting a recent onset of collapse, and also because it eventually cleared up, which points to the same conclusion.

It is probable, however, that the bronchiectasis was of long standing, and the febrile illness was one of the acute exacerbations which are so characteristic of the disease. Doubtless the atelectasis seen on examination

occurred during the febrile episode, and cleared up just as the one which originally caused the bronchiectasis had done. It is not uncommon for a lobe to collapse on several occasions, as Paterson[22] has pointed out.

At any rate, since the bronchi remained permanently dilated, and their appearance after the induction of an artificial pneumothorax indicated that this would be the event, the usefulness of this test is vindicated.

Incidentally, it is difficult to see what grounds there are in this case for claiming even clinical success for the artificial pneumothorax therapy. In three of Hennell's[21] cases, the induction of an artificial pneumothorax during an acute infection of the atelectatic system produced a striking remission of symptoms when other measures had failed. But in the case in question, Hennell states that the respiratory symptoms on admission were negligible; the patient was admitted on account of renal trouble, and the atelectatic bronchiectasis was discovered on routine examination. Since there were practically no symptoms to relieve, the only real benefit the artificial pneumothorax could have conferred was the permanent restoration of the bronchi to their normal calibre. It failed to do so; after the atelectasis cleared up the bronchiectasis remained. Although the patient remained clinically well for the few months he was under observation, it appears quite impossible to predict a continuation of this favourable state; long periods of remission are characteristic of the disease. It will be shown in the next chapter that the prognosis in such cases is bad, and it seems that lobectomy, in view of the patient's favourable age, would have been highly desirable.

In most of the cases previously shown in this work, an

artificial pneumothorax was induced not primarily as a test for the reversibility of bronchiectasis, but for various experimental purposes including the effect of artificial pneumothorax as a therapeutic agent.

In the following case, which was encountered when the theories considered above had been fully worked out, an artificial pneumothorax was induced solely for the purpose of ascertaining the condition of the bronchial walls, in order to facilitate prognosis and suitable disposal.

Case 4.

Male, aged 12 years. The patient complained about 13/8/47 of a productive cough, and pain in the right side. He was notified as a pneumonia, and admitted to Ruchill Fever Hospital on 20/8/47. A radiogram on the same day showed collapse of the right middle lobe, and slight enlargement of the right hilar glands. Bronchography was not carried out, and it was apparently concluded that the collapse was due to pressure of the right tracheo-bronchial lymph glands on the main bronchus of the right middle lobe. As the patient had little evidence of productive cough, he was dismissed from the hospital on 21/9/47, and a note sent to his family doctor advising him of the condition.

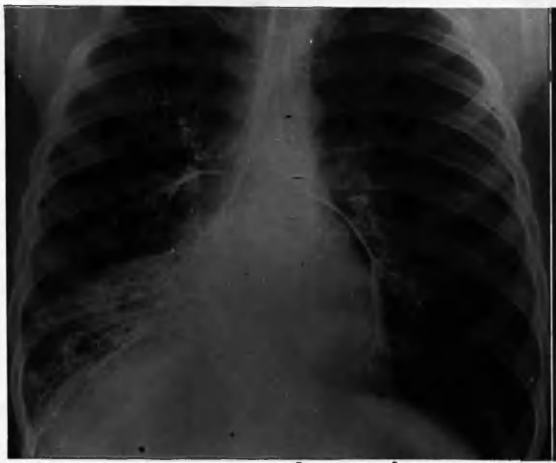
The author happened to come across the records of this case in November 1947, and secured the re-admission of the patient on 19/11/47 in order to carry out further investigations.

The general condition of the patient was good, though there was marked pallor. Physical examination revealed some tubularity of the R.M. over the right middle lobe, and a few moist crepitations could be heard in the same area. The P.N. was resonant. P.A., lateral and lordotic radiograms on 20/11/47 demonstrated a considerable degree of collapse of the right middle lobe. These are shown elsewhere (opposite p.9.). The extent of the collapse had not altered since 20/8/47.

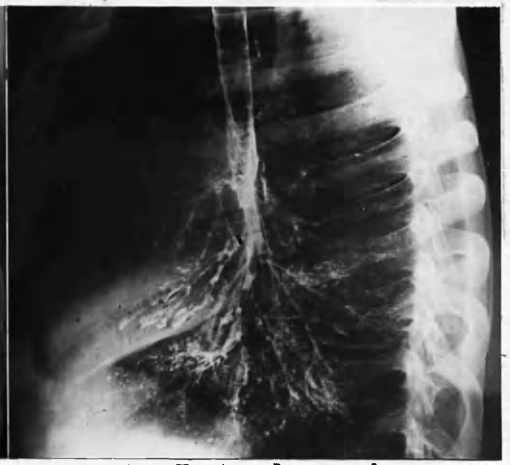
Bronchography on 2/12/47 showed cylindrical dilatation of the right middle lobe bronchi. Since they were patent, the collapse was obviously not due to pressure of the root glands on the main bronchus of the lobe, but due to obstruction of the bronchioles or finer bronchi.

Cough was not much in evidence, though in the mornings a small quantity of muco-purulent material was produced.

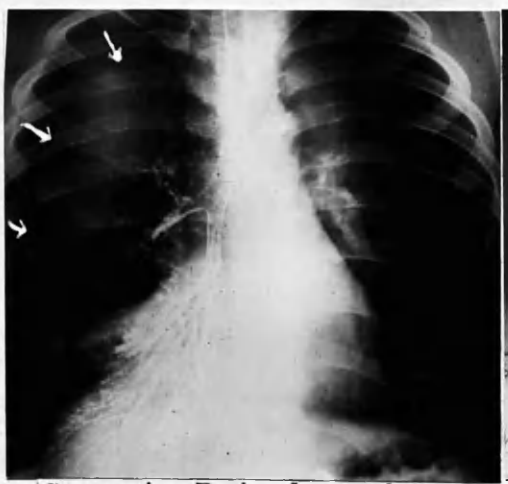
The question of treatment arose. Once the bronchiec-



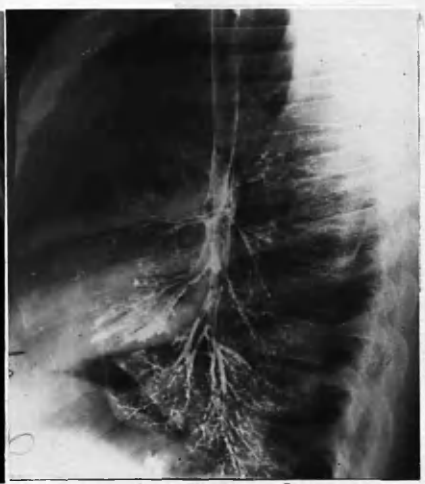
Case 4. P.A. bronchogram, 2/12/47.



Case 4. Lat. bronchogram, 2/12/47, revealing bronchial dilatation in collapsed rt. middle lobe.



Case 4. P.A. bronchogram, 9/1/48, showing rt. A.P. of moderate extent. The outline of the rt. lung is indicated by arrows.



Case 4. Lat. bronchogram, 9/1/48, demonstrating that the bronchial dilatation in the atelectatic rt. middle lobe has not been reduced by the relaxation of the lung afforded by the A.P..

tasis has become permanent it has been abundantly shown that medical measures are of little avail in arresting the progress of the disease, though exacerbations can be reduced in severity by the exhibition of such drugs as sulphadiazine and penicillin. Lobectomy appears at present to be the only method of effecting a real cure. In the case under discussion, although slight infection was present, the dilatation of the bronchi in the atelectasis might have been due entirely to the abnormal mechanical stresses present, and hence might completely disappear if the collapsed portion of lung re-expanded. The duration of the atelectasis was unknown, though if the onset were taken to be on 13/8/47 when the boy complained of pain in the side, it would have lasted 3 months when the position was being considered. It might have been there before, however, and this attack merely an exacerbation of the disease. How long could the case be observed and hope entertained that if the atelectasis cleared up, the bronchial dilatation might disappear? This question was considered in the previous chapter, and it was seen that no satisfactory answer could be given. Blades considered that 3 or 4 months would be a sufficient period of observation, but a case was shown in which the bronchi returned to normal after 18 weeks. In the hope of getting an immediate and definite indication, an A.P. was induced on the right side on 1/1/48, after a radiogram taken on 30/12/47 had shown the atelectasis to be unchanged.

The induction reading was -8,-5 cms. of water, and after 250 c.c. of air had been introduced, -6,-4 cms. of water. Refills were as follows:- 3/1/48, -6,-4 cms. of water... 300 c.c. of air... -4,-2; 6/1/48, -4,-2... 300 c.c. of air... -3,-1; 8/1/48, -2,-0... 350 c.c of air... 0,+2. A radiogram on 9/1/48 showed an A.P. of moderate extent. Bronchography was carried out on the same day, and it was seen that the bronchi were just as dilated as before. It was concluded that they had lost their elasticity, and would remain dilated even if the atelectasis cleared up. It seemed obvious that there was no point in maintaining the A.P. as a therapeutic agent, and it was allowed to absorb.

As is well known, the younger a bronchiectatic patient, the better the prospects of successful surgical intervention. The advice of a thoracic surgeon was therefore sought. Mr. R. S. Barclay reviewed the case, and placed the patient on his waiting list for lobectomy.

Bronchograms are shown opposite.

SECTION 3.

ARTIFICIAL PNEUMOTHORAX AS A THERAPEUTIC AGENT IN POTENTIALLY REVERSIBLE ATELECTATIC BRONCHIECTASIS.

When plugs of sputum have blocked the bronchioles or finer bronchi of a portion of lung with consequent atelectasis and bronchiectasis, the logical procedure would be to attempt to remove the plugs of sputum by bronchoscopic aspiration. If this procedure were adopted speedily enough, and if it were successful, re-expansion of the affected portion of lung would take place, and the bronchiectasis associated with the collapse would disappear. Owing to the inaccessible situation of the plugs of sputum, however, bronchoscopic aspiration is generally unsuccessful, and when this measure has failed, or when facilities for employing it are not available, it is suggested that the installation of an artificial pneumothorax in the affected side of the chest for the duration of the atelectasis would tend to prevent the development of permanent bronchiectasis.

It has been shown in the previous section that when atelectatic bronchiectasis is reversible, the induction of an artificial pneumothorax allows the dilated bronchi to revert to their normal calibre. The maintenance of an artificial pneumothorax during the period of the atelectasis would therefore relieve the bronchi in the atelectatic zone of the mechanical dilating stresses to which they would otherwise be exposed. Even if infection were to supervene, it is suggested that permanent bronchial dilatation would be unlikely to result if the mechanically dilated bronchi were allowed to revert to their normal

calibre in this manner, for the evidence in the previous chapter tended to show that infection was only a factor in the causation of permanent bronchiectasis when it arose in conjunction with the dilating stresses set up by atelectasis.

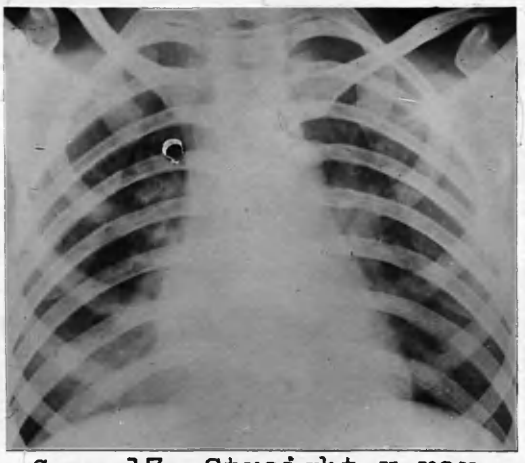
It is submitted that the installation of an artificial pneumothorax might have the further effect of promoting re-expansion of the collapsed portion of lung. If a plug of sputum were to block the main bronchus of a lobe, coughing would at first have an effect in expelling it. During the act of coughing the contraction of the abdominal and thoracic muscles, and the closure of the glottis would raise the pressure of the air in the alveoli and bronchi distal to the block to a level greater than that of the atmosphere, and with relaxation of the glottal obstruction the plug of sputum would tend to be forced out of the bronchus. But as air is absorbed from the bronchi and alveoli distal to the obstruction, coughing will obviously become ineffective in expelling the sputum. If the sputum is pushed by the atmospheric pressure down the main bronchus and eventually splits up and lodges in the bronchioles or finer bronchi in the manner which has previously been described, coughing will have no appreciable effect in expelling it. Resolution of the collapse will then depend on the bronchial movements and ciliary action conveying the material blocking the bronchioles or finer bronchi to the exterior. It was seen in the previous chapter (pp. 50,51) that the balance of evidence seemed to show that bronchial peristalsis played an important part in ridding the bronchial tree of foreign matter. It appears likely that the installation of an artificial pneumothorax of suitable extent in cases of potentially

reversible bronchiectasis, would, by relaxing the bronchial walls permit bronchial movements including bronchial peristalsis to take place, and therefore tend to promote re-expansion of the collapsed portion of lung.

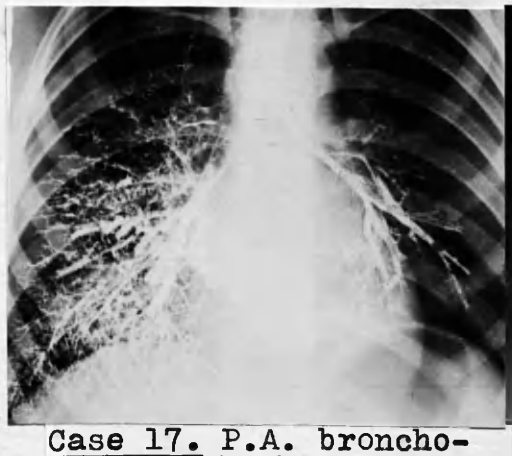
Artificial pneumothorax therapy was employed in three cases of atelectatic bronchiectasis occurring in the whooping cough series. From what has been said above, suitability for this treatment would depend on whether the dilated bronchi in the atelectatic lobe had retained their elasticity at the time of induction of the artificial pneumothorax; no benefit could be expected if the bronchial walls were irreparably damaged. Elasticity of the bronchial walls was presumed in the first two cases in which the procedure was carried out, because the onset of the atelectasis was known to within a week, there was no evidence of gross infection, and the artificial pneumothorax was induced a reasonably short time after the occurrence of the collapse.

In the third case elasticity of the bronchial walls could not be presumed, for although the collapse had almost certainly occurred only a short time before admission to hospital, and the induction of the artificial pneumothorax was not unduly postponed, infection in the atelectatic system was extremely severe. The value of performing bronchography after the artificial pneumothorax had been induced was, however, realised by the time this case was treated, and the marked diminution in the bronchial calibre after the procedure had been carried out, demonstrated that the infection had not irreparably damaged the bronchial walls.

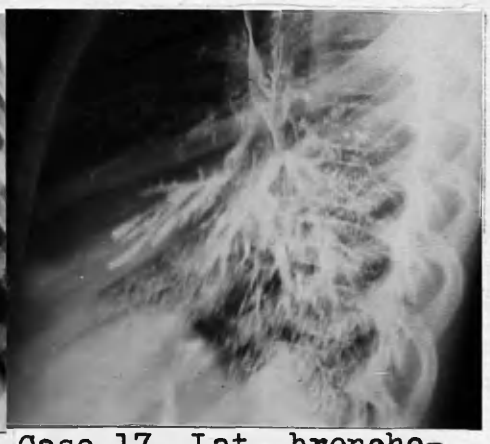
The relevant details of the cases follow.



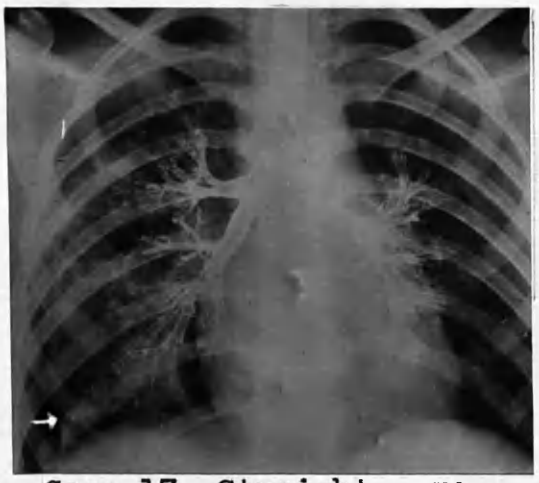
Case 17. Straight x-ray, 25/12/46. Atelectasis in rt. middle and lower lobe area.



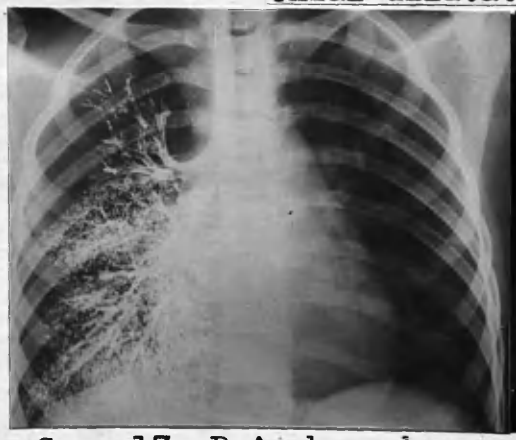
Case 17. P.A. bronchogram, 25/12/46. Bronchial dilatation in rt. middle and lower lobe area.



Case 17. Lat. bronchogram, 25/12/46, showing bronchial dilatation in the rt. middle lobe and in the ant. basal segment of the rt. lower lobe.



Case 17. Straight x-ray, 3/2/47, showing a small artificial pneumothorax in the rt. intrapleural space. The atelectasis in the rt. middle and lower lobes has disappeared, and there is no evidence of bronchial dilatation.



Case 17. P.A. bronchogram, 3/3/47, showing a normal rt. bronchial tree.

Case 17. Lat. bronchogram, 3/3/47.

Case 17.

Female, aged 6 years. Illness noted on 12/11/46, and admitted to hospital on 16/11/46. She was x-rayed as usual at weekly intervals, and on 4/12/46 a slight degree of atelectasis was noted in the right middle lobe area. By 25/12/46, the collapse had become marked,and apparently involved the right middle lobe, and part of the right lower lobe. Lateral and lordotic views were not taken, as bronchography was deemed much more satisfactory for localising the affected area. This procedure was carried out, and revealed atelectatic bronchiectasis involving the right middle lobe, and the anterior basal segment of the right lower lobe. A radiogram on 27/1/47 showed that the atelectasis persisted, and an A.P. was induced on the right side on the same day. The readings were -8,-4 cms. of water... 100 c.c. of air... -4,-2, cms. of water. On 30/1/47, a further 200 c.c. of air was introduced; the readings were -5,-3... 200 c.c. of air -3,-1. A radiogram on 3/2/47 demonstrated a small artificial pneumothorax in the lower part of the pleural cavity with an adherent pleura above. By good fortune, a fairly selective relaxation of the lung had therefore been obtained. The atelectasis had apparently cleared up, since the lung was not sufficiently relaxed to conceal the typical opacity had it been present. The A.P. was allowed to absorb, and straight x-ray and bronchography on 3/3/47 revealed no evidence of atelectasis or bronchiectasis.

Final radiograms and bronchograms on 19/9/47 demonstrated that there had been no recurrence of the atelectasis or bronchiectasis. A radiogram and bronchograms are shown opposite.

Clinical Features.

There was no rise of temperature or other clinical feature to draw attention to the onset of the collapse. Even when radiography had demonstrated its presence, the only abnormality on physical examination was a slight blowing quality in the expiratory phase of respiration heard over the affected area. The temperature was not elevated during the period the atelectasis was present, and cough and sputum production were negligible.

But for radiography, the condition would probably not have been recognised at all.

Discussion.

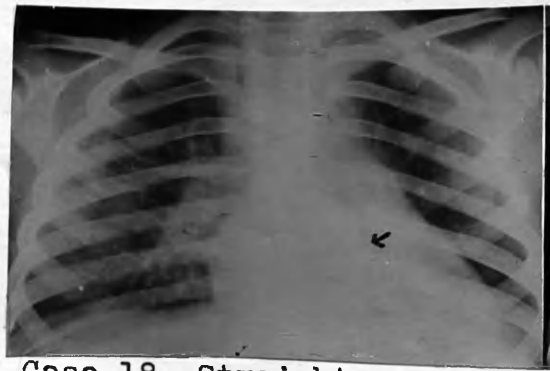
The radiogram of the patient showed evidence of healed tuberculosis, the hilar glands were enlarged, and the

Mantoux test was positive on admission. Her father was in a sanatorium with pulmonary tuberculosis. Examination of the stomach washings and sputum repeatedly failed to demonstrate the tubercle bacillus. She was observed radiographically and clinically up to January 1948, and remained perfectly well.

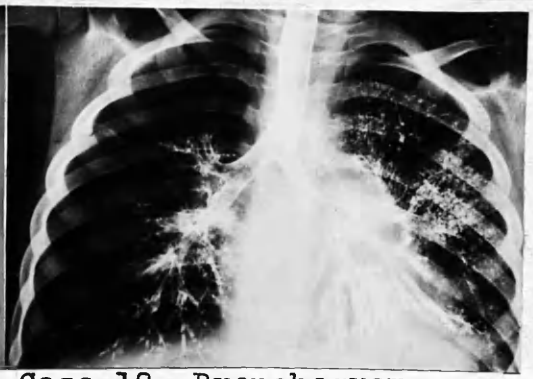
The pleural adhesions were therefore presumably due to former tuberculous infection. Bronchography demonstrated that the collapse was not due to the pressure of enlarged glands, as the neohydriol entered the affected area freely, and showed no evidence of bronchial compression.

The object of inducing the artificial pneumothorax was primarily to permit the bronchi to retain their normal calibre for the duration of the atelectasis, but as the condition resolved so quickly after the procedure had been carried out, no great benefit in this respect could have been conferred.

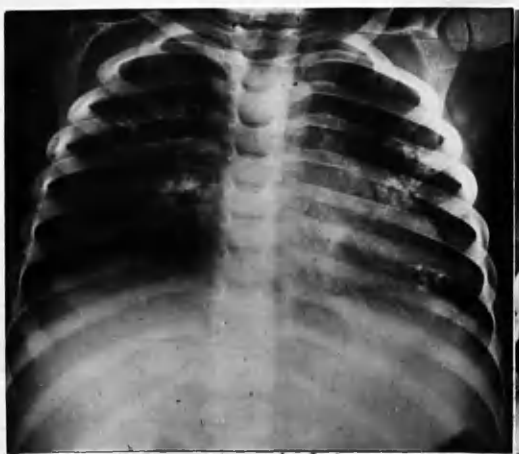
A secondary aim was to facilitate the re-expansion of the atelectatic lung, though there was no proof for the theoretical considerations which indicated that the induction of an artificial pneumothorax might have this effect. In this case the atelectasis had persisted for almost eight weeks, and showed no signs of clearing. Within a week of induction of the artificial pneumothorax, the atelectasis disappeared. Is it too much to suggest that the procedure assisted in the resolution of the condition? This event may, of course, have been purely fortuitous, but it seems clear that at least a replacement pneumothorax does not <u>prevent</u> re-expansion of an atelectatic lung.



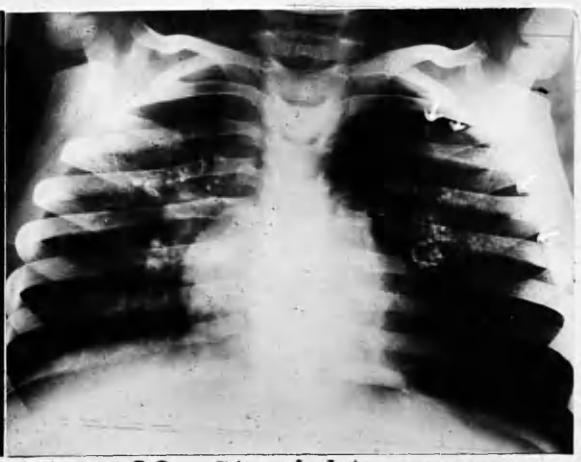
Case 18. Straight x-ray, 25/3/47. Collapse of left lower lobe. The triangular shadow can just be seen in the reproduction behind the heart shadow.



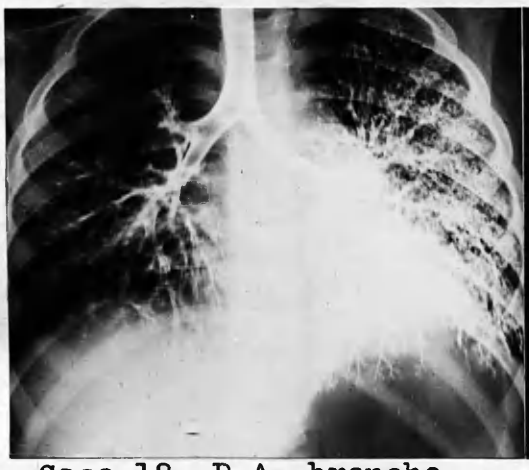
Case 18. Bronchogram, 25/3/47. Bronchial dilatation in collapsed lower lobe.



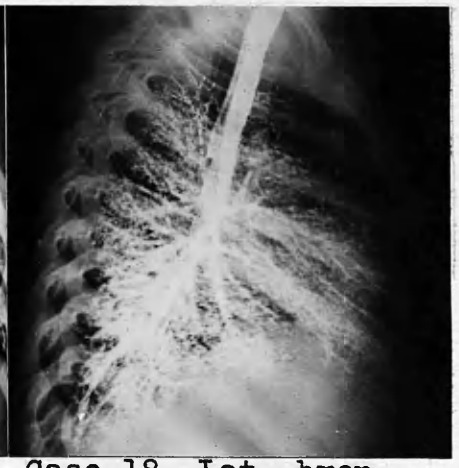
Case 18. Straight x-ray, 9/4/47, showing that there is marked displacement of the heart to the left, even allowing for slight obliquity of view.



Case 18. Straight x-ray, 10/4/47. Heart in normal position. Marginal left A.P.. Arrows indicate outline of lung. Old iodised oil in lung fields.



Case 18. P.A. bronchogram, 22/5/47 showing a normal left bronchial tree.



Case 18. Lat. bronchogram, 22/5/47, showing a normal left bronchial tree.

Case 18.

Male, aged 3 years. Mantoux negative. Illness noted on 12/12/46, and admitted on 16/12/46 with whooping cough. A routine weekly radiogram on 29/1/47 demonstrated some collapse at the right base, and it was subsequently shown that the right middle and lower lobes were involved, though not extensively. By 4/2/47 this condition was beginning to clear, but it was noted that atelectasis had developed in the left lower lobe. The collapse on the right side cleared up satisfactorily, but that in the left lower lobe became markedly worse. A straight x-ray on 25/3/47 showed extensive collapse of the left lower lobe, and bronchography on the same day demonstrated marked dilatation of the contained bronchi. A radiogram on 9/4/47 revealed that the collapse was more extensive than ever. (The mediastinum, however, is not so markedly displaced as the plate opposite suggests at first glance. The patient's position is slightly twisted). It was therefore decided to induce a left A.P. the next day. On the morning of that day, however, routine clinical examination revealed signs in the chest which strongly suggested a spontaneous pneumothorax had occurred on the left side. The P.N. was tympanitic, and the heart, which had been drawn to the left, now occupied its normal position. This astonishing finding was radiologically confirmed on the same afternoon (10/4/47). There was a marginal pneumothorax on the left side which unfortunately is not obvious in the photograph opposite. The shift of the heart to its normal position, even allowing for the twisted position of the previous day, is clearly seen.

The pneumothorax spontaneously created was artificially maintained. Its extent was kept small, because at this level the intrapleural pressure was well within normal limits, and with a small A.P. any re-expansion of the atelectatic lobe could be observed. It was later realised, as has been indicated, that the intrapleural pressure may be somewhat misleading, but the fact that at refills it was usually as high as -5,-3 cms. of water, coupled with the reversion of the heart to its normal position, pointed to the conclusion that the abnormal stresses set up by the atelectasis were, indeed, relieved. Owing to the occurrence of the spontaneous pneumothorax, what degree of negativity in the intrapleural pressure had been induced by the atelectasis was not known.

Weekly refills averaged 250-300 c.c. of air, and intrapleural pressures, -5,-3; -3,-1 cms. of water.

On 30/4/47, straight x-ray suggested that the atelectasis was beginning to clear, and by 8/5/47 the process was complete. The A.P. was allowed to absorb, and P.A. and lateral bronchograms on 22/5/47 demonstrated that the

bronchial tree was normal.

Unfortunately it was not possible to repeat the bronchograms several months later, but since the bronchial dilatation had entirely disappeared, there is no reason to suppose that the patient will not continue to enjoy the good health he possessed when he left hospital.

Clinical Features.

There was no elevation of temperature,or other indication of general malaise which might have suggested the onset of the atelectasis in either lung, and indeed the patient appeared to be remarkably little upset by the collapse of lung, the bronchographies, or the A.P. refills. The spontaneous pneumothorax also apparently caused him no distress, though this is not surprising, as it was small in extent, and tended in the circumstances to relieve rather than cause symptoms.

The atelectasis of the left lower lobe which was noted radiologically on 4/2/47 became apparent clinically ten days later when it was more extensive. The signs were the usual ones of impaired P.N.,and weak tubular breathing with occasional crepitations. The shift of the heart to the affected side could be detected clinically.

The collapse in the right side was also picked up clinically after a radiogram had indicated its presence.

Infection in this case was little in evidence. There was a not too troublesome cough, and the production of a moderate amount of sputum. Soon after the institution of the pneumothorax, cough and sputum production dwindled to nothing.

Discussion.

The comparatively gradual development of the atelectasis in this and the previous case is worth noting, as this phenomenon was not infrequently observed in other instances. Tannenberg and Pinner[23] showed that in rabbits, when the main bronchus of a lobe is ligated, complete atelectasis of the lobe occurs in three hours, and presumably in human subjects the time is comparably short. It does not, therefore, seem possible that in cases such as the one under discussion, the atelectasis is caused by the aspiration of a plug of mucus which divides at the bronchial branchings, and blocks at once the peripheral bronchi

which would have to be occluded in order to cause a collapse of the extent which eventually develops.

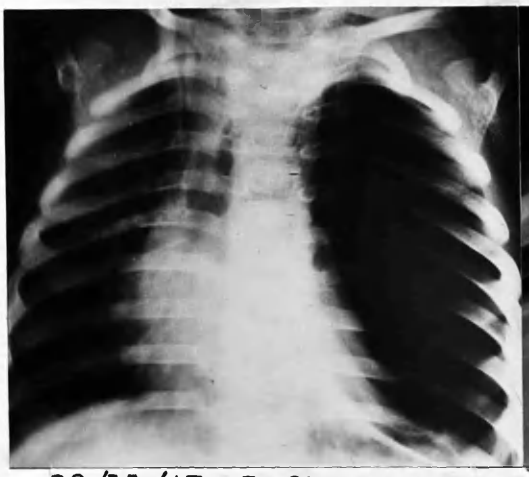
It is submitted that the explanation may be that a plug of sputum is aspirated which splits only at some of the bronchial branchings, thus failing to occlude some of the secondary bronchi, and causing only the degree of collapse which is noted initially. (This contention is supported by the fact that in Ogilvie's[8] pathological studies quoted in the previous chapter, it was observed that even in the most extensive collapses, there were emphysematous areas).

Then as secretion gathers in the peripheral bronchi the distal ends of which are blocked, some is expelled from time to time, and is aspirated into the neighbouring unaffected bronchi of the lobe.

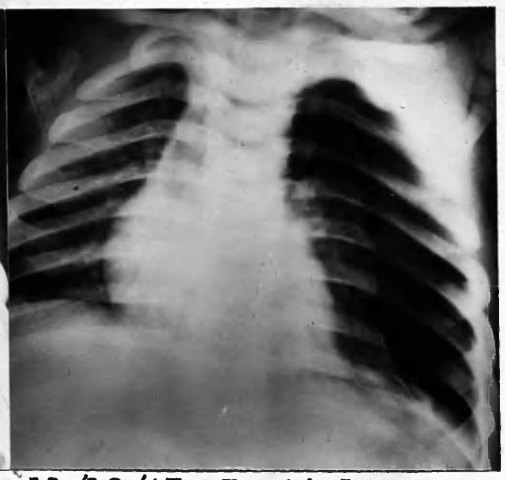
"Spread" of the atelectasis would be likely to be confined to the lobe originally affected, as expelled sputum would have immediate access to the small neighbouring bronchi, but would have to enter the main bronchus of another lobe and be very much dispersed before it could reach its small remote branches; what would be a dangerously large quantity of sputum in relation to the small neighbouring bronchi, might be comparatively innocuous in relation to the large main bronchus of another lobe.

This explanation seems feasible at least; no comment on the matter was noted in the literature.

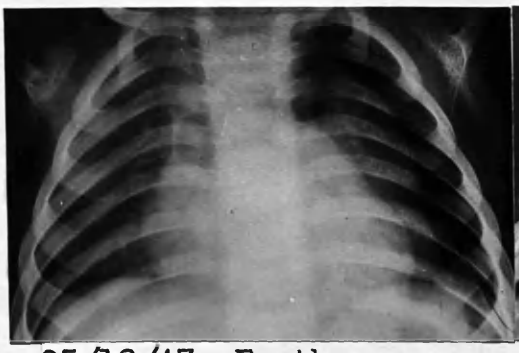
The occurrence of the spontaneous pneumothorax was most interesting. It has been previously pointed out that when atelectasis occurs, the elastic tension of the lung increases, and the periphery of the lung thus tends more than normally to be retracted from the chest wall. It is suggested that in this case, a deep inspiration



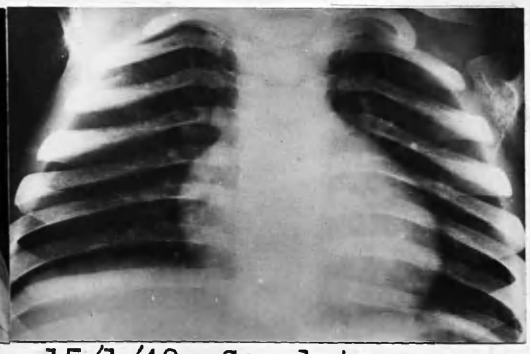
20/11/47. Left spontaneous pneumothorax compressing left lung and pushing the mediastinum to the right. Occurred in a patient suffering from severe laryngeal obstruction.



11/12/47. Partial re-expansion of left lung.



25/12/47. Further re-expansion of left lung.



15/1/48. Complete re-expansion of left lung.

following a bout of coughing by increasing this tendency still further, caused the rupture of a peripherally placed emphysematous bulla.

Colour is lent to the view that increased traction by the chest wall on the surface of the lung may lead to a spontaneous pneumothorax by the fact that this complication was observed in a case of non-diphtheritic croup admitted to Ruchill Hospital on 17/11/47. The patient was a boy aged two years, and the traction of the chest wall on the surface of the lung must have been very great on inspiration as the laryngeal obstruction was severe. No intrapulmonary disease was noted clinically or radiologically and the boy made a good recovery. Radiograms are shown opposite.

As regards the eventual disappearance of the atelectasis and bronchiectasis in the whooping cough case, it can again be said that the result may or may not have been due to the artificial pneumothorax therapy. But in view of the extensive nature of the atelectasis, its rather prolonged duration (over 13 weeks), and the marked degree of bronchial dilatation, the absence of permanent bronchial damage was encouraging.

Case 13.

Male, aged 5 years. Illness noted on 26/3/47, and was admitted to hospital with whooping cough on 9/4/47. There was a marked whoop, violent spasms of coughing, and the production of copious quantities of purulent greenish-yellow material. The temperature was elevated to 103 F..

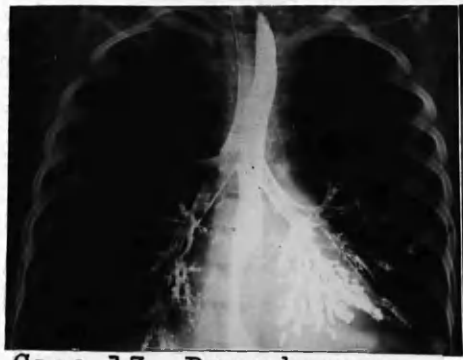
Scattered crepitations and rhonchi were heard all over the lung fields. There was a triangular area of diminished resonance at the left base, and weak bronchial breathing and medium moist crepitations could be heard in that zone. The apex beat was in the left mid-clavicular line. The case was diagnosed as whooping cough, bronchopneumonia, and atelectasis of the left lower lobe, and the patient was given sulphadiazine and penicillin for nine days. The temperature settled a week after admission.

A straight x-ray on 18/4/47 showed some broncho-pneumonic infiltration in both lungs, and extensive collapse of the left lower lobe. The patient continued to have extremely violent and frequent spasms of coughing with very copious production of sputum, and in view of his poor general condition, bronchography was postponed until 13/5/47. The bronchogram showed marked dilatation and crowding of the left lower lobe bronchi.

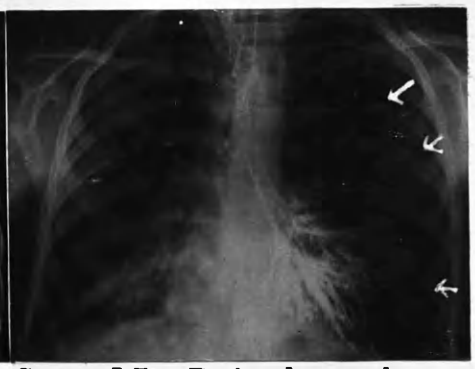
The intrapleural pressure on the right side was estimated on 21/5/47, and the readings were -5,-3 cms. of water. Immediately thereafter, and using the same manometer, the intrapleural pressure on the left side was recorded, the patient being placed as nearly as possible in a corresponding position to the one formerly occupied, and the needle inserted in the corresponding interspace, at the corresponding site. The readings for the left side were -8,-5 cms. of water. The small difference in the pressures on the two sides has already been commented on, and an explanation offered. 200 c.c. of air were then allowed to flow into the left pleural space, when the readings on the manometer became -5,-3 cms. of water. The pneumothorax was subsequently increased and maintained, its extent being ascertained by radiograms and fluoroscopy.

One of the most striking features of this case was the truly remarkable manner in which cough and sputum production diminished after the A.P. had been induced. The spasms of coughing had been so violent and frequent that the patient was losing sleep and becoming emaciated, and the sputum mug had constantly to be changed. Immediately after the A.P. was induced the cough and sputum production dramatically declined in severity, and in a few days were negligible factors.

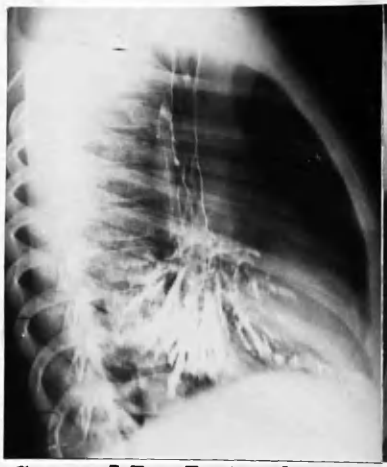
By the time this case was treated, it was realised that visualisation of the bronchi in the atelectatic area while the A.P. was present was essential for three reasons. First, it was necessary to determine if the relaxation of the lung had brought about a diminution of the bronchial dilatation; if it had not, the bronchi had lost their elasticity and the A.P. might as well be abandoned. Second, if the bronchi were still elastic, it was desirable to ascertain their calibre in order to find out the minimum degree of pulmonary relaxation necessary to restore it to approximately normal, for it was conceivable that an A.P. of very large extent might cause actual compression of the finer bronchi and tend to prevent re-expansion of the atelectatic lung. Third, an A.P. of the minimum extent necessary for satisfactory relaxation of the bronchi would have the advantage of allowing any



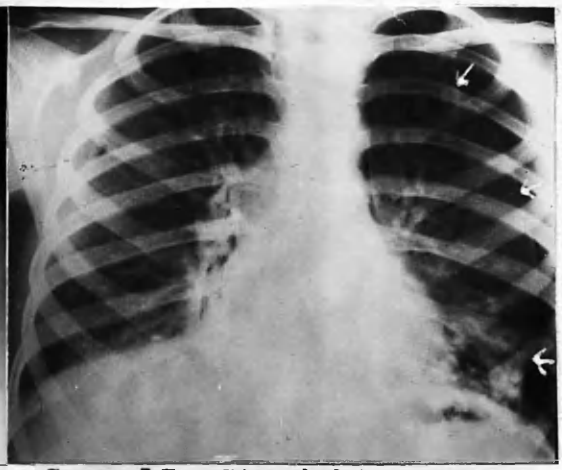
Case 13. Bronchogram, 13/5/47, showing collapse of left lower lobe with marked dilatation of the contained bronchi.



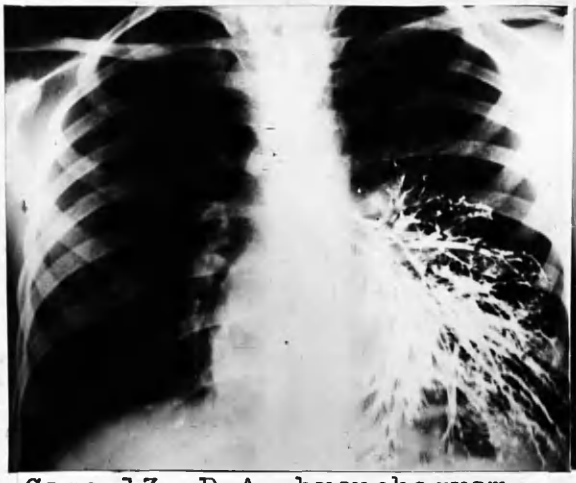
Case 13. P.A. bronchogram, 26/6/47, showing the presence of a left A.P. of moderate extent. The outline of the left lung is indicated by arrows. There has been a dramatic reduction in the calibre of the left lower lobe bronchi.



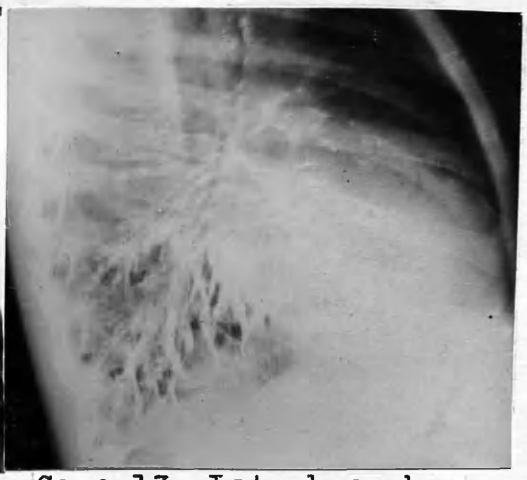
Case 13. Lat. bronchogram, 26/6/47. confirming the fact that the bronchial dilatation in the left lower lobe has almost disappeared.



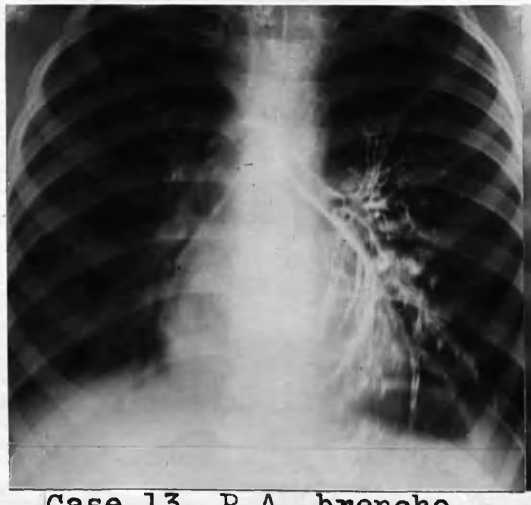
Case 13. Straight x-ray, 11/8/47, shown to indicate clearly the extent of the A. P. maintained. Arrows indicate the outline of the left lung. The left lower lobe has now almost completely re-expanded. There is old neohydriol in the left lung.



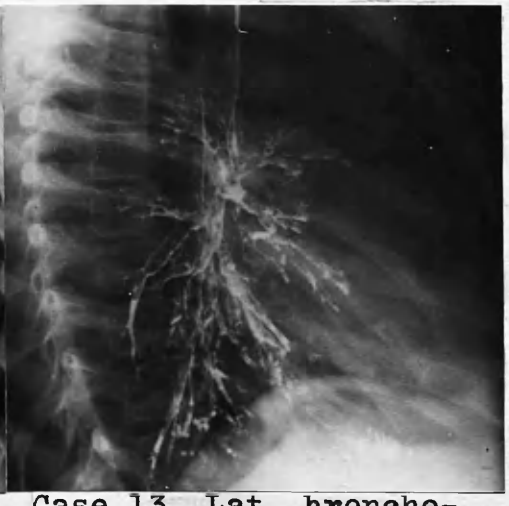
Case 13. P.A. bronchogram, 17/9/47. The L.A.P. has absorbed. Normal l. lung and bronchial tree. The heart shadow in the reproduction has been lightly shaded with pencil to make the outlines of the bronchi in this area more obvious.



Case 13. Lat. bronchogram, 17/9/47. Normal left bronchial tree.



Case 13. P.A. bronchogram, 25/2/48. Normal left bronchial tree.



Case 13. Lat. bronchogram, 25/2/48. Normal left bronchial tree.

clearing of the atelectasis to be detected on a radiogram.

Bronchography was therefore carried out on 7/6/47 under general anaesthesia, but was unsatisfactory. The procedure was repeated under local anaesthesia, on 26/6/47, just prior to a refill. It was then seen that a very marked reduction in the calibre of the bronchi had been effected, though slight ectasia was still present. It was obvious that the elasticity of the bronchial walls had been largely retained. The fact that slight dilatation persisted despite the A.P. suggested that either there was slight impairment of the bronchial elasticity, or relaxation of the tissues in the atelectatic area was not quite adequate. If the first alternative were correct there would be no point in securing an A.P. of greater extent; on the other hand, if the second alternative were right, this would be desirable. It would have been easy to find out. The lung could have been further relaxed, and bronchography performed again. If the bronchial calibre were now seen to be normal, it would be known that the bronchi were fully elastic, and the optimum degree of A.P. had been discovered; if the slight ectasia were still present it could be assumed that some impairment of the elasticity had occurred. It was considered, however, that the knowledge to be gained was not worth the inconvenience to the patient of a further bronchography.

The reduction of the bronchial dilatation had been so dramatic that it was clear elasticity had been largely retained, and even if some impairment had been suffered, it was highly desirable to maintain the A.P., and avoid further overstretching. If the bronchial elasticity were entirely unaffected, then no doubt a larger A.P. would effect a complete restoration to normal calibre. The position, however, seemed quite satisfactory as it was, for the ectasia was negligible compared with what it originally had been, and since the bronchogram was taken just before a refill, it could be expected that a further reduction would occur after this procedure had been carried out.

The A.P. was accordingly maintained at approximately the extent seen on the bronchogram of 26/6/47, reproduced opposite.

A radiogram on 4/8/47 indicated that the atelectasis was slightly less extensive, and a refill due on 5/8/47 was omitted. Another x-ray on 11/8/47 showed that the atelectasis had almost cleared up, and the A.P. was allowed to absorb. (Photographic reproduction is unfortunately unsatisfactory owing to the position of the atelectasis in the heart shadow, and old neohydriol in the lung). The A.P. had absorbed by 25/8/47, and the collapsed portion of lung was seen to have completely re-expanded.

Bronchography on 17/9/47 showed a normal bronchial

tree. Clinically the child was well.

The patient was dismissed, and was seen again on 25/2/48. He had remained well in the interim. Physical and radiological examinations revealed no abnormality, and bronchography demonstrated a normal bronchial tree.

Discussion.

This case differed from the others, in that there was evidence of very severe infection. In view of this, the extent and long duration of the atelectasis (eighteen weeks), and the marked initial bronchial dilatation, it may be said that permanent bronchiectasis was a probability. But it is not possible to go further than that, and claim that the artificial pneumothorax therapy prevented that unhappy result. But whatever reluctance may be felt in ascribing benefit to a form of treatment on grounds not wholly scientific, it would be carrying caution too far not to suggest that in this case the artificial pneumothorax contributed to improvement in at least one respect. As has been mentioned, before the artificial pneumothorax was induced, cough and very copious sputum production were prominent features of the disease. Immediately after the operation, these symptoms were strikingly reduced; in a few days they were negligible, and in a fortnight were not in evidence at all. The improvement was so dramatic that it appears extremely improbable that it was merely fortuitous. The explanation may be that by relaxing the bronchial walls and permitting the resumption of bronchial movements, secretion is carried off and stagnation avoided, thus preventing irritation and the production of more secretion.

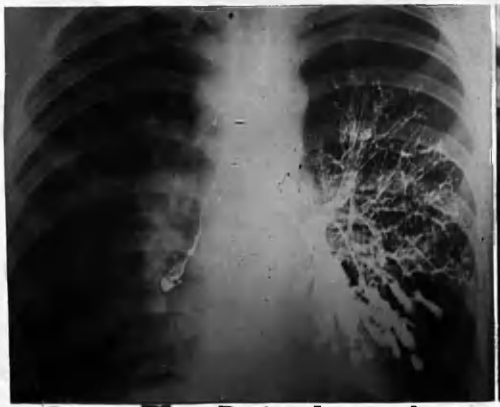
The atelectasis in this case lasted approximately eighteen weeks, and for twelve weeks the abnormal dilating stresses on the bronchial walls were relieved by the

artificial pneumothorax; it seems clear that this effect has highly desirable. The assumption is made that the duration of the atelectasis was at least not prolonged by the institution of the pneumothorax, for such theoretical and practical evidence as there is, points to the conclusion that the procedure, when conducted according to the principles acted upon in this case, tends actually to promote re-expansion of the atelectatic area of lung.

The above three cases illustrate the effect of artificial pneumothorax in cases of atelectatic bronchiectasis where the elasticity of the dilated bronchi has not been destroyed at the time the artificial pneumothorax is induced.

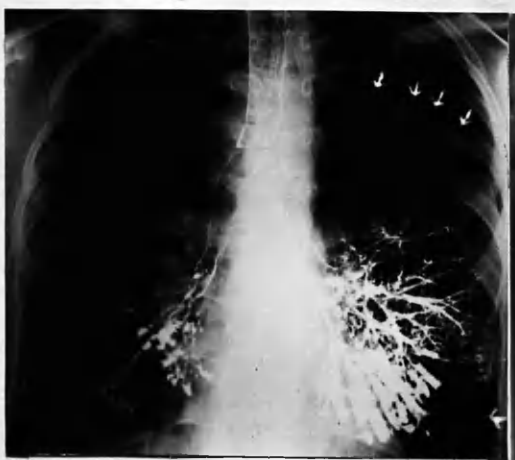
In the previous section dealing with the induction of an artificial pneumothorax as a test for bronchial elasticity in atelectatic bronchiectasis, six cases were described in which there was good reason to suppose that the bronchial elasticity had been lost. In five of these cases artificial pneumothorax was employed for varying periods as a therapeutic agent. No benefit was, in fact, apparently derived in any of them, and the artificial pneumothorax was abandoned after varying periods. It had no appreciable effect on the bronchial dilatation, and when it had disappeared, it was seen that the bronchiectasis was as prominent as before. The artificial pneumothorax therapy did not appear to have even any beneficial clinical effect.

As a matter of interest, artificial pneumothorax therapy was employed for varying periods in cases of bronchiectasis without any associated atelectasis. As expected, it was found to be useless. One of these cases is shown below.



Case 71. P.A. bronchogram, 9/5/47, showing bronchiectasis of the left lower lobe and lingula.

Case 71. Lat. bronchogram, 9/5/47, showing bronchiectasis of the left lower lobe and lingula.



Case 71. P.A. bronchogram, 23/6/47, showing the presence of a left A.P. of moderate extent. The outline of the left lung is indicated by arrows. The bronchial dilatation in the left lower lobe and lingula is as great as before.

Case 71. Lat. bronchogram 23/6/47, showing that the bronchial dilatation in the left lower lobe and lingula has not been reduced by the relaxation of the left lung by the A.P..

Case 71.

Male, aged 19 years. Admitted to Ruchill Hospital on 17/4/47 with "pneumonia." He had had two attacks in the last two years. He suffered from occasional "colds" during which time cough and production of sputum were troublesome symptoms. For the most part he considered his health was good, and he had no difficulty in following his employment as van driver. Physical examination revealed impaired P.N., increased V.R., broncho-vesicular R.M., and medium moist crepitations at the left base. The temperature was 102 F. on admission, but fell to normal in two days on the exhibition of sulphadiazine. There was considerable production of sputum when first seen; after a few days it dwindled, but did not quite disappear. A tubular element in the R.M. at the left base persisted, and a few crepitations could occasionally be heard.

Bronchography was performed on 9/5/47, and revealed bronchiectasis of the left lower lobe and the lingular process of the left upper lobe. A left A.P. was induced on 12/5/47; the readings were -7,-4... 250 c.c. of air... -5,-3 cms. of water. The A.P. was kept comparatively small in extent to avoid any danger of actually compressing the smaller bronchi and perhaps precipitating an atelectasis by causing sputum lying within their lumina to produce occlusion. The possibility appeared to be somewhat remote, but as artificial pneumothorax therapy was being employed merely to confirm the belief that it was of no value in this type of case, there was no point in running the slightest risk. It may be mentioned that had atelectasis of a lobe occurred in the course of the artificial pneumothorax treatment, the logical procedure would have been to increase the extent of the artificial pneumothorax until the bronchial dilatation caused by the atelectasis had been neutralised. It will be remembered that Lander and Davidson demonstrated this point in their "black lobe" case. Bronchography was carried out on 23/6/47, and it was seen that the affected bronchi were as dilated as before. No improvement in any respect was noted as a result of the therapy, and the A.P. was abandoned on 17/9/47. Bronchograms are shown opposite.

Comment on Artificial Pneumothorax Therapy.

Artificial pneumothorax therapy was employed in three cases of atelectatic bronchiectasis in which the affected bronchi eventually returned to normal. The bronchiectasis would possibly have disappeared in all three instances whether artificial pneumothorax therapy had been employed or not, and, indeed, potentially reversible cases are the only ones in which it is likely to be of any value.

Estimation of the benefits conferred by the therapy in the cases in question must necessarily be speculative. The prompt manner in which the atelectatic portion of lung re-expanded in the first example (Case 17, p.244), after the induction of the artificial pneumothorax suggests that the procedure may have assisted resolution. This possibility was foreseen from a study of the dynamics of atelectasis. If, however, the condition would have cleared up as quickly whether an artificial pneumothorax had been induced or not, it is almost certain that this therapy had no effect on the ultimate favourable result, for the eight weeks duration was comparatively short, the bronchial dilatation apparently purely mechanical, and the length of time the bronchial dilatation was reduced by the artificial pneumothorax negligible.

In the second case (Case 18, p.246), the atelectasis lasted for thirteen weeks and a pneumothorax was present for four. Again it may have assisted in resolution of the condition. Even if it did not, the mechanical dilatation of the bronchi was relieved during the time it was present.

There was evidence of a moderate degree of infection in this case. Another case (Case 16, p.137), rather comparable to this one, occurred in the whooping cough series. There was atelectatic bronchiectasis of both

lower lobes, though the extent was rather less, and resolution of the atelectases was speedier,(eight weeks for one lobe, and nine weeks for the other). The degree of infection was rather similar. Although artificial pneumothorax therapy was not employed, the bronchi of both affected lobes eventually recovered completely. A slight degree of ectasia persisted after the disappearance of the atelectasis suggesting that the bronchial walls had sustained some damage, but three months later it was seen to have quite disappeared. Great hesitancy is therefore felt in claiming any success for the artificial pneumothorax therapy in the case under discussion.

In the third case (Case 13,p.249), in which an artificial pneumothorax was induced, it has already been stated that it is considered that permanent bronchiectasis was a probable result, in view of the very severe infection of the atelectatic system, and the eighteen weeks duration of the collapse. As the dilatation of the bronchi was relieved for twelve weeks by the artificial pneumothorax, it is not unreasonable to suppose that this therapy contributed to the successful result, though there is certainly no real proof of this. A noteworthy feature of this case was the dramatic reduction of cough and sputum production after the artificial pneumothorax was induced. Generalisations cannot be made from a particular instance, but as Hennell[21] noted the same phenomenon in six cases of infected atelectasis in which artificial pneumothorax therapy was employed, it appears that it may be a usual incident of the treatment in cases of this type.

When Hennell's[21] article was encountered it was hoped that his findings would furnish a comparison with those of the present study. Unfortunately, vital investigations

were omitted, as the cases were conducted on quite different principles.[21] Hennell purported to show that artificial pneumothorax therapy could effect a clinical cure in cases of early bronchiectasis.

In his first case, the patient was a female of fourteen years, admitted to hospital with an infected atelectasis of the left lower lobe. An artificial pneumothorax was induced eight weeks later, and after the third refill, this treatment was continued at a clinic for two years. Bronchography, before the artificial pneumothorax was induced, demonstrated bronchiectasis, but the procedure was not repeated afterwards. Hennell states, "This patient has remained under observation since 1935. When last seen in June 1944, she had remained entirely well. She had had many "colds" during these nine years, some of considerable severity, but none of them had resulted in a chronic cough, indicating that, while the anatomical defect of the bronchi probably persisted, a clinical cure was achieved and maintained. The roentgenograms of the expanded lung presented an almost normal appearance, showing apparent resolution of the pneumonic process in the left lower lobe."

If the frequent "colds" indicated to the author that an anatomical defect of the bronchi persisted, it is difficult to see what he thought other than that the bronchiectasis was still producing symptoms. This is difficult to reconcile with the view that the child remained "entirely well", and a clinical cure had been effected. It is amazing that bronchography was not carried out to ascertain the condition of the bronchial tree.

As nothing was said of the history of the patient before entering the hospital, it is impossible to estimate

how "early" the bronchiectasis was. The author states that the intrapleural pressure before the artificial pneumothorax was induced was -18,-8 cms. of water; as this is considerably lower than normal, it may be hazarded for reasons already given, that the atelectasis was recent.

The second case is too complicated to be described fully, but again bronchography was not carried out while the artificial pneumothorax was present or after it had absorbed. According to the author the patient remained free from pulmonary symptoms for seventeen months after the institution of artificial pneumothorax in spite of two major operations for essential hypertension. (The patient was aged 35 years). In the absence of bronchography showing a normal bronchial tree, however, it cannot be regarded as certain that the artificial pneumothorax therapy effected a lasting cure.

The third case was of a young man aged sixteen years admitted to hospital with an infected atelectasis of the right lower lobe, and a right sided pleural effusion. Fifty cubic centimetres of a sterile straw coloured effusion were aspirated; the cells were mainly lymphocytes. Bronchiectasis in the affected lobe was diagnosed from the appearance on the straight x-ray. The pleural effusion apparently absorbed, and an artificial pneumothorax was induced ten weeks after admission. It was maintained for six months, and then allowed to absorb. A straight x-ray subsequently showed that the atelectatic lobe had re-expanded. Bronchography was not performed, but the patient remained well for a further period of eighteen months, and the author regarded the result as a clinical cure.

The fourth case described by Hennell has already been considered in the previous section on artificial pneumo-

thorax as a test for bronchial elasticity. In this case bronchography was not carried out before artificial pneumothorax, but was done when it was present. The bronchi were still markedly dilated. In view of this fact, and a history of "colds" for many years before admission to hospital, it has already been stated that it is considered there is little ground for regarding it as a case of "early" bronchiectasis, and none for inferring a "cure" from a few months absence of symptoms.

No details were given of two further cases in which Hennell noted a dramatic clinical improvement after the institution of artificial pneumothorax therapy.

The only factual evidence which apparently emerged from Hennell's study was that the induction of an artificial pneumothorax in six cases of infected atelectasis produced in all of them an immediate remission of symptoms which was of too striking a nature to be ascribed to mere coincidence.

Hennell's[21] claims for artificial pneumothorax therapy are as follows, "Admittedly, pneumothorax therapy does not relieve the patient of the dilated bronchi. However, as has been shown, it can control the infection in early bronchiectasis so that the patient may be restored to normal health."

It is believed that the first statement is inaccurate, and that the second is much too great a generalisation from the results of a few cases. Strangely enough, Hennell apparently did not visualise the affected bronchi at the end of the treatment to find out if they were still dilated. He had previously, however, employed artificial pneumothorax[24] extensively in the treatment of chronic bronchiectasis, and no doubt from his former experience took the

permanence of the dilatation for granted.

There are many inconsistencies in Hennell's exposition[21] of the principles underlying artificial pneumothorax therapy, but it is only necessary to consider a few of his beliefs which are directly opposed to those put forward in the preceding pages.

The fundamental point of difference is that Hennell[21] believes that artificial pneumothorax therapy can effect a "clinical cure" even though the bronchiectasis remains permanent. All a "clinical cure" under the circumstances really implies is that a temporarily "wet" bronchiectasis has been converted for an unspecified period into a "dry" bronchiectasis. It will be shown in the next chapter that the prognosis in this type of case is almost universally conceded to be very poor. Moreover, in the only case which he observed over a period of years, a history of frequent severe "colds" does not agree with his statement that the patient remained "entirely well."

One of his objectives in inducing an artificial pneumothorax is "to counteract the increased pull on the diseased bronchi because of a lowered intrapleural pressure incidental to atelectasis or fibrosis of the pulmonary parenchyma." If there is fibrosis of the pulmonary parenchyma, how can the case be regarded as "early"? And if the "low intrapleural pressure" is due to fibrosis, why should the induction of an artificial pneumothorax necessarily have any good effect on the bronchiectasis? It has been pointed out elsewhere that fibrosis is more likely to compress and distort the bronchi than to dilate them, and the "low intrapleural pressure" is due not to the overstretching of potentially sound though temporarily collapsed lung tissue, but to the fibrous contraction of

irreparably damaged material. Surely lobectomy is here the obvious treatment?

Again, Hennell[21] passes over without comment the fact that in the one case in which he carried out bronchography after the artificial pneumothorax had been induced, the bronchi in the atelectatic lobe were still markedly dilated; yet it is difficult to imagine what real benefit could be hoped for in continuing the therapy.

He suggests that cases suitable for artificial pneumothorax therapy are those in which a selective collapse of the affected lobe is seen when the artificial pneumothorax is induced, coupled with an immediate improvement in the symptoms. It is pointed out that the appearance of a selective collapse is likely to be seen where there is any shrunken lobe whether atelectatic, fibrotic or both. Also a case of atelectatic bronchiectasis at the period when it is encountered may have no symptoms to relieve.

Hennell[21] maintained the artificial pneumothorax for two years in his first case and for six months in the others on a purely arbitrary basis. It is submitted that the duration of the treatment should be determined by the duration of the atelectasis; after it has cleared up there is no point in maintaining the artificial pneumothorax as there are no abnormal dilating stresses to counter. It is most unlikely that the slight normal inspiratory dilating stress has any effect in causing progression of the bronchiectasis. In any case it has yet to be shown that bronchiectasis unassociated with an atelectasis is ever reversible, and it would appear that in the present state of knowledge the sooner lobectomy is undertaken in such instances, the better.

Hennell[21] admits that "Pneumothorax therapy for bron-

chiectasis has been tried many times but, as a rule, the results obtained have been so poor that this mode of treatment has fallen into disrepute," but claims that not only were the six "early" cases already referred to clinically "cured", but in a previous study of his,[24] "a small selected group of patients with chronic bronchiectasis of one to four years' duration was reported upon, in which the satisfactory clinical results produced by pneumothorax therapy have now lasted from seven to thirteen years." It has been seen, however, that Hennell's conception of good health appears to be somewhat elastic, and as it is an everyday experience to encounter bronchiectatic patients who have carried on their normal duties with only occasional exacerbations of symptoms, his results are singularly unconvincing.

The criterion of cure in the present study was complete return of the dilated bronchi to normal, and complete disappearance of symptoms. This was only seen in three cases of potentially reversible atelectatic bronchiectasis. Though the cases might have recovered if left to themselves, evidence has been adduced tending to show that the institution of an artificial pneumothorax was a logical procedure, and may have contributed to the good result. A review of the literature did not reveal any investigations either corroborating or conflicting with these findings. Hennell[21] discovered that in cases of infected atelectasis, the induction of an artificial pneumothorax can produce a dramatic diminution of symptoms, and this phenomenon was observed in one case.

In five cases of atelectatic bronchiectasis in which the bronchi had lost their elasticity, artificial pneumothorax therapy proved of no value, and in six cases of

bronchiectasis without an associated atelectasis, the results, as expected, were equally poor. This has been the general experience.

It is concluded, therefore, that artificial pneumothorax therapy is only of value in cases of potentially reversible atelectatic bronchiectasis. After the artificial pneumothorax has been induced, bronchography should be carried out. Only if a marked reduction in the bronchial calibre is observed is it worth maintaining the artificial pneumothorax as a therapeutic measure, as otherwise the bronchi have lost their elasticity and will remain dilated even if the atelectasis clears up. The optimum relaxation of the lung is the minimum necessary to allow the bronchi to return to their normal calibre. As soon as the atelectatic portion of lung is seen to have re-expanded, the artificial pneumothorax should be abandoned, as it has served its purpose. If the atelectasis persists, no guide can be given as to how long it is worth maintaining the artificial pneumothorax. Presumably if infection is absent, the bronchi in the atelectatic area, relieved as they are by the artificial pneumothorax of dilating stresses, may retain their elasticity indefinitely. It is not known how long a portion of lung can remain atelectatic and still be capable of re-expansion. Re-expansion took place after eighteen weeks in one case. Six months would certainly not appear to be too long to wait.

It would, of course, be ridiculous to place great stress on the apparent good results of a form of treatment in three cases, but the procedure advocated appears to have a sound theoretical basis, and it is considered that it is not unworthy of further trial.

Even if proved to be of value, the place of artificial pneumothorax therapy in the treatment of bronchiectasis would, at present, be small, as the vast majority of cases are seen when the bronchial dilatation is permanent. Radiology, however, is being employed to an ever increasing extent, and cases of potentially reversible bronchiectasis are bound to be encountered with increasing frequency.

SUMMARY OF THE CHAPTER.

The dynamics of atelectasis were considered, and it was concluded that when atelectasis occurs the internal tension of the lung is increased, but not uniformly; the increase in tension is largely localised to the atelectatic zone.

Any portions of bronchi in the atelectatic area which are in communication with the atmosphere will be mechanically dilated; so also will any alveoli which have escaped collapse.

The intrapleural pressure in the affected hemithorax will be lowered as a result of the increased internal tension of the lung. It is a rough measure of the internal tension of the whole lung, however, not of particular parts; hence the internal tension in the atelectatic portion of lung may be much greater than the intrapleural pressure would suggest.

The average intrapleural pressure in healthy subjects is in the region of -7.3,-3.85 cms. of water, but wide variations are seen. The intrapleural pressure in a case of recent atelectasis may therefore be lowered, but yet remain within normal limits.

The reduction in pressure may be detected by taking simultaneous readings on the affected and contralateral sides. If the mediastinum is fixed, the intrapleural pressure on the contralateral side will be unchanged; if it is mobile the intrapleural pressure on the unaffected side may be lowered, but so slightly that it is still useful for comparison.

In the course of time, if, as a result of prolonged stretching, the bronchial and pulmonary tissues in the atelectatic area lose their elasticity, the increased internal tension of the affected lung will diminish, and

the lowered intrapleural pressure will consequently tend to revert to normal.

In old standing cases of atelectasis, the intrapleural pressure may not be reduced at all. It is believed that this fact, and the fact that even in recent cases of lobar atelectasis the intrapleural pressure may be only slightly reduced, have not previously been mentioned in the literature; the impression has been given that in all cases of atelectasis involving as much as a lobe in extent, the intrapleural pressure is very much reduced.

It is necessary to determine whether cases of atelectatic bronchiectasis are potentially reversible in order that affected lobes may not be unnecessarily removed by surgical operation. A test has, therefore, been devised. It is suggested that an artificial pneumothorax should be induced. If the dilated bronchi in the atelectasis show no appreciable diminution in calibre, the bronchiectasis is permanent, and lobectomy is the treatment of choice; if a marked diminution in calibre is seen, it is probable that bronchial elasticity has been retained, and complete recovery may be anticipated if the atelectatic portion of lung re-expands.

In cases shown by the test to be potentially reversible, it is suggested that an artificial pneumothorax should be maintained to abolish or reduce the mechanical dilatation of the bronchi, and also to promote re-expansion of the atelectatic lung. If the atelectatic system is infected, it appears that the induction of an artificial pneumothorax may bring about a dramatic clinical improvement.

It is considered that artificial pneumothorax therapy is useless in cases of permanent bronchiectasis whether associated with an atelectasis or not.

CHAPTER 5.

THE TREATMENT OF ATELECTASIS AND BRONCHIECTASIS.

In the previous chapter the advisability of employing artificial pneumothorax therapy in cases of potentially reversible atelectatic bronchiectasis was discussed. The problem of treatment as a whole for atelectasis and bronchiectasis will now be considered, and the various measures which may be adopted put in perspective.

Atelectasis due to occlusion of the main bronchus supplying the collapsed portion of lung need only be mentioned. The obstructed portion of the bronchus is accessible to bronchoscopic exploration, and if it is possible to remove the obstructing agent, be it within or without the bronchus, then this will be done by the thoracic surgeon. Atelectasis arising in this manner will not be further considered.

In the majority of cases, atelectasis is due to the blocking of the peripheral portions of the finer bronchi or of the bronchioles by sputum, and here percussion postural drainage, that is to say postural drainage in the manner advocated by Nelson[1] with percussion of the affected area by the palm of the hand, would seem a simple and probably effective first line of treatment.

Sante[2] discovered accidentally that in cases of absorption collapse, laying the patient on his sound side and rolling him to and fro produced coughing and expectoration, often accompanied by re-expansion of the atelectatic area.

These measures were adopted in some of the whooping cough cases, and probably did good, though it is impossible

to prove that benefit actually accrued in view of the quite unpredictable duration of an atelectasis. Many cleared up just as rapidly without any treatment at all, and some did not respond to the therapy. Nevertheless, it is clear that this form of treatment should never be neglected, as it is logical, simple, always available, and free from danger.

Carbon di-oxide inhalation has its advocates. The increased respiratory excursion probably has the mechanical effect of tending to dislodge sputum, thus promoting re-expansion of the lung. Coryllos[3] and Brown[4] have also adduced evidence indicating that carbon di-oxide tends to liquify the mucous plugs blocking the finer bronchi. The value of this form of therapy was emphasised by the work of Banyai and Cadden[5] published in 1943. This method of treatment appears to be reasonable, but it was not employed in the cases investigated, and nothing can therefore be said regarding its efficacy.

Since atelectatic bronchiectasis is caused in the vast majority of cases by blockage of the peripheral portions of the bronchi with viscid sputum or other material, bronchoscopic aspiration would appear to be the treatment of choice. Erwin[6] pointed out in 1939, however, that in practice the results were rather disappointing, and subsequent trial has supported his observations.

Blades[7] employed bronchoscopic aspiration without success in four cases of atelectatic bronchiectasis which subsequently proved "reversible." It is interesting to note that although he recovered much viscid material from the atelectatic system,no diminution of the calibre of the bronchi was observed, whereas when he carried out the same procedure in old standing cases of bronchiectasis unassoc-

iated with atelectasis the aspiration of large quantities of infected secretions did effect a temporary reduction of the bronchial dilatation. These results are what the theories advocated in the last chapter would have suggested; in the atelectatic bronchiectasis the bronchial dilatation was unaffected because the mechanical stresses set up by the atelectasis were unrelieved, while in the non-atelectatic bronchiectasis some reduction in calibre was seen because the accumulated secretions bulging still further the walls of the already dilated bronchi were removed.

Hennell[8] had a similar lack of success in inducing re-expansion of the lung by this means in six cases of atelectatic bronchiectasis, though in one example the atelectasis cleared for a brief period.

Once the aspirated mucus has found its way into the peripheral bronchi, therefore, it appears that it is extremely difficult to dislodge it by bronchoscopic aspiration, though Brown[9] recorded some successes in cases of lobular collapse. Coope[10] called attention not only to the inaccessibility of the mucousplugs when in the peripheral situation, but also the fact that they are often so tenacious that bronchoscopic aspiration would be useless; the offending material would have to be recovered by forceps.

The ideal method would be to employ bronchoscopic aspiration while the mucus is still in the main bronchus of the lobe. It is by no means easy, however, to diagnose the condition at this early stage. When pulmonary collapse arises during or after an abdominal operation there is usually a sharp rise of temperature which gives some indication that this complication has occurred, and

armed with this knowledge, the temperature was carefully watched in the whooping cough cases in order to find out if in this condition also an elevation heralds the onset of atelectasis. As has been mentioned on page 32 of this work, however, no correlation could be discovered, and in an article dealing with atelectasis as a complication of thoracic operations, Gray[11] states "The fever is less striking than in atelectasis after a non-pulmonary operation."

There appears, therefore, to be no easy way of finding out the exact time of onset of an atelectasis. In practice, the best method of securing early diagnosis is the frequent employment of radiography, but even if radiograms are taken at weekly intervals, as they were in the whooping cough cases investigated in this study, diagnosis would obviously still not be early enough in the majority of cases to ensure that the mucus was still in the main bronchus supplying the atelectatic portion of lung. Tannenberg and Pinner[12] have shown that in the rabbit complete collapse of a lobe can occur in an hour following obstruction of a main bronchus, and if similar time factors apply to the human subject it therefore seems likely that the mucus will be "sucked" to the periphery within a very short space of time, as the forces tending to do this come into play immediately air begins to be absorbed from the tissues of the atelectatic system.

Even assuming for a moment that was possible to detect immediately the onset of an atelectasis, the prospects in whooping cough at any rate would be somewhat daunting for all but the most enthusiastic bronchoscopists. If the results of the present investigations are fairly representative, forty three per cent of cases are complicated by absorption collapse, and although most of the atelectases

are of a comparatively ephemeral nature, there is no way of telling this at the onset. Hence if the intention were to employ bronchoscopic aspiration when the mucus was in the favourable proximal position, the procedure would have to be carried out in every case, a somewhat onerous task, particularly in young children.

In practice bronchoscopic aspiration would probably only be carried out after simple measures such as percussion postural drainage had failed to produce re-expansion of the collapsed area, and by that time the sputum would in all likelihood have been drawn to the periphery of the bronchial tree.

Postural drainage and bronchoscopic aspiration may usefully be employed in all cases of atelectasis due to aspiration of sputum. Artificial pneumothorax therapy, on the other hand, is only of value when the collapse is of great enough extent to give rise to obvious bronchial dilatation, since although the induction of an artificial pneumothorax may, as previously suggested, incidentally assist in resolution of the atelectasis, the main purpose is to relax the dilated bronchi.

In the series of one hundred and fifty whooping cough cases studied, four suitable examples were discovered, and the effects of artificial pneumothorax treatment in three of them were considered in the previous chapter.

Four other cases of atelectasis with an associated cylindrical bronchiectasis were also seen, but as the degree of dilatation was slight, and infection minimal, it was considered that artificial penumothorax therapy was unnecessary; in point of fact these patients made a complete recovery.

Although postular drainage and bronchoscopic aspiration

should be employed before resorting to artificial pneumothorax therapy, it is obvious that if a case is suitable for artificial pneumothorax, then the sooner the treatment is initiated the better, especially when there is evidence of severe infection. If time is wasted, the bronchial elasticity may become permanently impaired, and the therapy will no longer be of value.

In all cases in which this form of treatment is contemplated, bronchography should be carried out shortly after the induction of the artificial pneumothorax in order to determine if the bronchiectasis has been markedly reduced; if it has not, there is no point in persevering with the therapy.

Some criticism of artificial pneumothorax treatment may now be dealt with briefly. The substance of this thesis was contained in an address delivered by the writer to a meeting of the Scottish Tuberculosis Society in July 1947, and considerable interest and controversy was aroused by the suggestion of artificial pneumothorax treatment for cases of potentially reversible bronchiectasis. Unfortunately the type of case for which the therapy was advocated was not, apparently, sufficiently stressed, and some members of the audience appeared to believe it was suggested for cases of permanent bronchiectasis. Their objections were therefore irrelevant; others may be briefly considered.

There was a criticism that with small children it would be very difficult to introduce the needle into the pleural cavity without risk, as the child would be likely to wriggle and cry. Naturally, in the case of children there is more difficulty in inducing an artificial pneumothorax than with adults, but in the examples attempted it

was found to be perfectly simple. The only real technical difficulty encountered was that on occasion the child persisted in crying, elevating the intrapleural pressure and causing air to bubble out of the manometer tube, thus upsetting the readings. Usually, however, a preliminary dose of chloral hydrate, a soothing address, and the provision of a sweet to suck during the operation induced a complaisant attitude.

Another objection was that as the main consideration of treatment was to promote re-expansion of the lung, it was wrong to reduce the calibre of the bronchi to normal, and make it more difficult for the sputum to be ejected. It seems, however, that this criticism overlooked the vital principles of the genesis of bronchiectasis. The same forces which are distending the bronchi are also tending to impact the sputum in their peripheral portions. Moreover, as has been pointed out, coughing has little effect in expelling plugs of sputum from the bronchioles or finer bronchi of a collapsed portion of lung, and movements of the bronchial walls which would assist in dislodging the mucous plugs would also appear to be inhibited by the abnormal distending stresses, and would thus perhaps be restored if these stresses were removed by the induction of an artificial pneumothorax.

In any case it is rather odd to suggest that the normal bronchial calibre is not adequate to allow sputum to be satisfactorily carried away, and it is certain that the number of observers who look upon the widely dilated bronchi in an atelectatic area as a happy dispensation of providence must be very few. It seems that those who can believe that the sputum is more easily expelled because the bronchi are dilated, might also be persuaded that it would

be easy to cycle uphill if only the roads were widened.

Still another criticism was that if the atelectasis were due not to obstruction of the peripheral portions of the bronchi but to compression of the main bronchus by enlarged hilar glands, the induction of an artificial pneumothorax would make the condition worse than before. This, of course, is no real criticism of artificial pneumothorax therapy, as all forms of treatment are liable to be dangerous if administered by the ignorant. There should be no difficulty in differentiating the two types of atelectasis. Bronchoscopic aspiration, if facilities were available, would be attempted as a preliminary measure, and any occlusion of the main bronchus of the lobe would then be noted. Even if bronchoscopy were not carried out, bronchography certainly would be, and if there were an obstruction of the main bronchus the lipiodol would not enter properly the bronchial tree beyond.

It has been mentioned previously that a pedunculated tumour in the main bronchus of a lobe may escape detection by bronchography, but this would not be so if it obstructed the lumen sufficiently to cause atelectasis. It is unlikely to cause confusion in the differential diagnosis of the cause of atelectasis, but may be mistaken for "dry" bronchiectasis, as it is usually seen in young adults, and haemoptysis is a leading symptom.

The suggestion was made from another quarter that bronchoscopic aspiration was the treatment of choice; this is so, but its limitations have been mentioned.

There appears, therefore, to be no valid contra-indication to artificial pneumothorax treatment in the type of case for which it is recommended, and as potentially reversible atelectatic bronchiectasis is peculiarly likely

to be encountered in whooping cough, it may prove a valuable therapeutic agent in this disease.

Once bronchiectasis has become permanent, there appears to be at present only one satisfactory form of treatment, and that is the surgical removal of the affected lobe or lobes.

Before discussing the factors which influence the selection of cases for surgical intervention, a few words may well be said on the technical difficulties confronting the surgeon, and an estimate made of the degree of risk inevitably inherent in the operation even when performed with the highest degree of skill.

The first successful lobectomy was performed by Heidenhain[13] in 1901, and the operation is now taken so much for granted that it is difficult to realise that as late as 1925, the danger involved was so extreme that Graham[14] was recommending and carrying out the rather ferocious procedure of "cautery pneumonectomy" as a more suitable alternative. That is to say, after producing adhesions between the visceral pleura and the chest wall, the diseased part of the lung was cauterised by a red-hot soldering iron, and the slough allowed to separate. This process was repeated every three weeks until the affected area was extirpated. Graham[14] published the results of a series of fifty four cases treated by this method, and stated that sixty three per cent improved, eleven per cent died at operation, and twenty two per cent died some time later. Per sistent bronchial fistula was a not uncommon complication.

These figures, coupled with the remarkably unpleasant operative procedure, were therefore somewhat depressing for potential subjects, though doubtless satisfactory from

a technical point of view, as Sauerbruch and other European surgeons according to Tudor Edwards,[15] were getting no better results from the two stage lobectomy operation then in vogue.

It was as late as 1929 that the modern one stage lobectomy operation was first performed. The pioneer was Brunn[16] of San Francisco who showed that it was possible to carry out the operation with a free pleura. In a series of six cases, he reported that four patients were cured, one improved, and one died.

The first man to perform a successful lobectomy for bronchiectasis in Great Britain was Tudor Edwards,[15] also in 1929, and the brilliant technical advances which have since transformed the mortality figures for the operation constitute one of the most amazing surgical triumphs of modern times.

Lilienthal,[17] in 1935, published the first large series. Of forty two patients only fifteen (thirty six per cent) survived the operation, and several were left with permanent bronchial fistulae. The immediate mortality rate was therefore sixty four per cent.

Then, in 1939, Tudor Edwards[15] made known the results of a series of one hundred and sixty six lobectomy operations. The mortality rate was only twelve per cent (twenty two cases). A follow up of the cases showed that seventy eight patients were cured, and twenty seven markedly improved though still left with some residual infection.

By 1945, Clagett[18] was able to record that the operative mortality at the Mayo Clinic in lobectomy operations for cases of reasonably well localised bronchiectasis was between three and five per cent.

The latest large series published at the time of writing is that of Meade[19] and his fellow workers (February 1947). Between November 1943, and April 1946, one hundred and ninety six lobectomies were performed on one hundred and ninety patients at the Kennedy General Hospital, Memphis, with only one death. Other surgeons working at the same centre, subsequently increased the series to two hundred and thirty six cases without further mortality.

These figures indicate the rapid and marvellous advances which have taken place in the technique of the operation. It is noteworthy that in Tudor Edwards' series[15] only two deaths occurred in the last fifty four cases, and these in patients with advanced and extensive disease.

The results of different lobectomy series are, of course, not strictly comparable, as the mortality will be greater where the surgeon is willing to accept the risk of operating on advanced and otherwise doomed cases, but technical progress has clearly been remarkable.

It would be out of place in this work to deal in detail with matters which are purely in the province of the surgeon, but a few words may be said on the complications of the operation, as they are important from the point of view of prognosis, and the occurrence of atelectasis in the remaining sound lobes during or after operation is interesting in view of the theories which have been discussed in this work.

This latter complication arises sometimes from post-operative swelling of the tissues causing a constriction of the main bronchus supplying the affected area, and it has been suggested that atelectasis of the left upper lobe is peculiarly liable to follow removal of the lingular process for this reason.[10] Non-expansion of the remaining

sound lobes could obviously also be brought about by a bronchial fistula allowing the escape of air into the pleural cavity, but the most usual cause is obstruction of the bronchi by muco-pus.

Collapse of the lung follows opening of the thorax, and thus secretion is likely to collect in the bronchi of the sound lobes and prevent re-expansion. In order to minimise this danger Bethune[21] devised a system of pre-operative "pleural poudrage" to promote adhesions, and Tudor Edwards[15] employed this measure in the lobectomy series already quoted. The lung was collapsed by an artificial pneumothorax, B.P.C. talcum powder insufflated on to the surface of the sound lobe or lobes, and the air in the pleural cavity withdrawn. Most observers are of the opinion that pre-operative pleurodesis reduces the incidence of atelectasis, but, of course, it by no means abolishes it, as it does not avert the danger of the aspiration of infected material squeezed at operation out of the bronchiectatic lobe. Bronchoscopic suction during operation is now frequently employed to minimise this risk.

Gray[11] published an interesting article on "Atelectasis Complicating Pulmonary Lobectomy" in 1946 based on a study of one hundred and six lobectomies.

He found that whereas in ninety cases of suppurative disease the incidence of atelectasis was twenty six per cent, there were no atelectases following sixteen lobectomies for non-suppurative conditions, and when the amount of sputum produced daily was more than one ounce, there was a higher rate of atelectasis than when the production of sputum was less. This strongly supports the theory that atelectasis is commonly due to the aspiration of sputum. The rate of atelectasis was increased when more

than one lobe was removed.

A very interesting finding was that in children the likelihood of atelectasis was twice as great as in adults. It may be remembered that earlier in this work (on page 160), it was stated that the impression was gained from a study of x-rays in the pneumonia wards that atelectasis was much commoner in children than in adults, and it was suggested that the narrower calibre of the bronchi in children might facilitate their occlusion by aspirated sputum. Gray[11] puts forward the same surmise.

Bronchial fistula is another important complication of lobectomy operations, but it is of little theoretical interest in the present connexion, and the fact need only be mentioned.

Septic complications have been much reduced since the introduction of the sulphonamides and penicillin, as the operation can be performed under an "umbrella" of these drugs. Cerebral abscess and suppurative pericarditis, never common, are now rare.

The indications for surgical treatment of bronchiectasis will now be considered.

The problem of potentially reversible atelectatic bronchiectasis may be dealt with briefly as it has already been discussed at length. It is only in the past few years that its importance has been recognised; Tudor Edwards,[15] for example, makes no reference to it in an article published in 1939, in which the indications for lobectomy are considered.

In the present study it was concluded that the condition was the earliest stage of bronchiectasis, but from the surgeon's point of view it makes little difference whether it is regarded as a variety of "true" bronchiec-

tasis, or whether it is called by another name such as "pseudo-bronchiectasis", the term used by Blades.[7] The important point is that when bronchial dilatation is demonstrated in an atelectatic lobe, the possibility of recovery must be excluded before the patient is subjected to lobectomy. A possible method of doing this was suggested in the previous chapter.

If the means indicated cannot be employed because an adherent pleura makes the induction of an artificial pneumothorax impossible, or if the test is in any case considered unsatisfactory, the only other criteria available are the duration of the atelectasis and the severity of the associated infection.

Whether mere prolonged mechanical overdistension of the bronchi would be sufficient to produce permanent bronchiectasis is unknown, but the chances are that it would. How long it would take is another question.

In any case, the matter is somewhat academic, because a degree of infection of the atelectatic system is sooner or later almost inevitable, and the more severe it is the sooner will permanent bronchial damage result.

Blades[7] quoted Campbell as recording a case of "pseudo-bronchiectasis" in which it appeared likely that the bronchial tree had returned to normal after as long as forty six days, and in the present study a case of severely infected atelectatic bronchiectasis was shown in which complete recovery took place after approximately eighteen weeks (Case 13,p.249.).

Judging from these examples, it would not seem wise to perform a lobectomy on a case of atelectatic bronchiectasis sooner than six months after the occurrence of the collapse if this is known, or the date when the condition was first

diagnosed if the time of onset is unknown. In some instances, no doubt, a long history of cough and sputum production coupled with bronchograms showing severe saccular bronchiectasis would indicate that surgical intervention could safely be more speedy.

The position is therefore at present extremely unsatisfactory, and in view of the fact that early cases of bronchiectasis are bound to be much more frequently recognised as the use of radiography increases, some test such as that advocated is urgently required. On the one hand undue haste in performing lobectomy may result in the removal of potentially sound lung tissue, and on the other hand too long delay may permit "spread" of the bronchiectasis.

Having considered the problem of reversible bronchiectasis, the indications for lobectomy in cases of permanent bronchiectasis may now be dealt with. Even here the conception that the disease is uniformly progressive has been challenged.

Roles and Todd,[22] in a series of one hundred and six cases of established bronchiectasis in adults, demonstrated a group in which the cases were "dry", that is to say cases in which sputum production was absent. The symptoms causing the patient to seek treatment were persistent non-productive cough, and in the majority of cases, haemoptysis.

Bronchiectasis of this type is sometimes thought to be benign, but the follow up of the cases conducted by Roles and Todd[22] certainly does not support this view. Ten of the fourteen cases recorded in this "dry" group were treated medically and showed evidence of infection within six years, and in this period three had died, and two were totally incapacitated.

Tudor Edwards,[15] in discussing these cases, pointed out that the mere fact of haemoptysis having occurred indicated that infection had been present when the cases first came under review; the bleeding could only have come from granulation tissue or ulceration of the bronchial wall.

Cases of dry bronchiectasis have since come to light, however, in which haemoptysis, and even cough have been absent.

Martin and Berridge[23] in 1942 cited twenty five examples of "Bronchiectasis without Disability" which were only discovered because the authors were particularly interested in the disease, and carried out bronchography on the slightest suspicion of its presence. The patients were soldiers, many of them in category A1. who had been admitted to hospital mostly on account of trifling respiratory symptoms. In civil life, as in the Army, they had been able to lead a normal existence. Brock,[24] in a letter to the Lancet deprecating any tendency to take bronchiectasis lightly, observed that "Bronchiectasis without Disability" was somewhat of a misnomer, as the patients after all had sought medical attention because of respiratory symptoms however slight. While this criticism is doubtless just, it seems nevertheless reasonable to infer that if these patients were only discovered after having been practically symptomless for years, there may be similar cases never brought to light at all for the simple reason that they never need to seek medical attention.

In discussing dry bronchiectasis in a previous chapter (Chapter 3,p.66), two cases were demonstrated.

One was a boy of nine years (Case 27,p.68), who was being treated in Ruchill Sanatorium for a minimal tuberculous lesion in the left lung. Routine examination

revealed a rather tubular R.M. in the right upper lobe, and bronchography disclosed dilatation of the bronchi. It is considered that this was the end result of an atelectasis of the right upper lobe which had subsequently re-expanded, and judging from the situation it would probably be of tuberculous origin. Prosoroff[25] showed in 1929 that collapse of an upper lobe in children due to pressure of enlarged glands on the bronchus supplying the affected area was frequently mistaken for epituberculosis, and Morlock and Pinchin[26] in 1933 demonstrated a case of this nature, and showed that canalisation of the bronchus during bronchoscopy produced re-expansion of the lobe. It has been indicated in another chapter that this type of collapse would not cause bronchiectasis save by the damming up of secretions, and while this may have been the mechanism producing the condition in the case in question, it is considered more likely that it arose in the usual way, from the aspiration of secretion into the bronchioles or finer bronchi of the lobe. Whatever the causative factor, over a period of years the boy has had no symptoms referable to the bronchiectasis, and for over a year no symptoms at all. As the situation in the right upper lobe ensures excellent drainage, supervening infection is unlikely, and this is a case in which it may be asserted with some confidence that surgical intervention is quite unnecessary.

The other case (Case 26,p.67), presented a much more difficult problem. The patient here was a man of twenty two years, who had had no respiratory trouble that he could recall until he was admitted to Ruchill Hospital on account of left lower lobe pneumonia. The clinical findings have already been described. It need only be said here that after a trifling illness of a few days duration,

the patient appeared perfectly well. Bronchography, however, revealed cylindrical bronchiectasis in the left lower lobe. Since there was no atelectasis, the bronchial dilatation was obviously permanent, and the decision had to be taken whether or not to remove the lobe.

The points in favour of operation were the youth of of the patient, and the localisation of the disease to one lobe. Although, as will be shown later, the younger the patient the better the prospects in a lobectomy operation, delay of a year or two per se, in this case would not have an appreciably adverse effect on the patient's chances of survival; the more pressing peril was obviously the occurrence of severe infection in the bronchiectatic lobe with possible spread to other areas. The bad prognosis in Roles and Todd's[22] group of dry bronchiectasis has already been noted. Nevertheless this case appeared to be rather different, in that there was no history of cough or haemoptysis, and the patient was quite symptomless after the first few days in hospital. The opinion of a thoracic surgeon was sought and Mr. R. S. Barclay[27] saw the case. In view of the fact that the prognosis of this type of case is as yet unknown, he considered that the lobe should not be immediately removed, and suggested that the patient should be kept under observation with periodic radiological and bronchographic examinations. Nine months have now elapsed without the patient showing any signs of illness. The danger of supervening infection and deterioration, is, of course, fully realised, and it will be interesting to see how the case fares over a period of years.

Apart from relatively infrequent instances such as the two just described, it may be said that in cases of established bronchiectasis the sooner lobectomy is performed the better, if the operation is feasible at all.

Should the patient have a persistent cough and possibly an occasional haemoptysis, mere absence of sputum production is no contra-indication to operation. The figures just cited for the group of cases described by Roles and Todd[22] show that the prognosis is exceedingly grave should medical treatment only be employed.

These authors also examined the course of the disease in two further groups of cases - those in which there was occasional sputum production (Group 2), and those in which sputum was constantly coughed up (Group 3), and found that the prognosis for Group 2 was as bad as that for Group 3. In all, forty eight patients received purely medical treatment. Within five years, twenty three were dead, and nine totally incapacitated.

Warner[28] observed the course of bronchiectasis in one hundred and ten patients where medical treatment only was employed; twenty three per cent died within nine years.

The prognosis in established bronchiectasis with symptoms is therefore extremely grave if the aid of the surgeon is not enlisted.

At one time it was believed that the prognosis in children was better than in adults, because the impression had arisen that the bronchial dilatation did not increase, and as the thorax grew larger with advancing years the relationship between the calibre of the bronchi and the size of the thoracic cavity eventually reached the normal. An investigation by Findlay and Graham[29] of thirty two cases of bronchiectasis in children dispelled such optimistic illusions. The patients were observed over a period of three to six years. Twelve of the children died in that time - four following attempts to drain lung abscesses, and another from tuberculous meningitis. The average

duration of life of the other seven from the time they were diagnosed was 2.63 years. Of the patients who survived, fourteen had been kept under continuous observation for three to six years. In eight of these cases there was steady deterioration, in three the condition appeared to be more or less stationary, and in three the bronchiectasis had apparently disappeared. Tudor Edwards,[15] from a study of the bronchograms of the last three cases, considered it was doubtful whether, in fact, the bronchiectasis had actually resolved.

Findlay and Graham,[29] therefore established fairly conclusively that permanent bronchiectasis in children as in adults, tends to be a progressive and fatal disease though the course may be a long one. Their findings are supported by those of Ford,[30] published in a recent article. Twenty six cases of proven bronchiectasis in children were medically treated and observed over a period of six to seven years; ten died, ten remained relatively unchanged, but had productive cough, cyanosis and clubbing of the fingers, and six were clinically well at the end of the period of surveillance though it was not possible to perform bronchography in order to determine the condition of the bronchial tree. Ford[30] found that the disease was more rapidly fatal in children in whom it was diagnosed about the age of three years than in children in whom it was diagnosed about the age of six years, and concluded that the prognosis was worse in very young children. It may be, of course, that the disease originated in the children in both groups about the same age, and the diagnosis was later in the second merely because the symptoms were less obvious.

The sulphonamide group of drugs and penicillin have

unfortunately been disappointing as therapeutic agents in cases of established bronchiectasis. Norris[31] and other observers discovered that the exhibition of sulphonamide drugs produced an alteration of the bacterial flora, but no permanent benefit to the patient. Humphrey and Joules[32] (1946) and Southwell[33] (1946) found even more marked bacterial changes following inhalations of penicillin mist; and Ford[30] observed that after a few days of penicillin therapy the usual mixed coccal culture was replaced by a growth consisting largely of coliform organisms. Bobrowitz,[34] Southwell,[33] Ford[30] and others are, however, unanimous that treatment with penicillin has no lasting effect. It was administered to several patients during the present investigations, by inhalation, and by intratracheal instillation, with disappointing results. The sulphonamides and penicillin are of value, however in dealing with exacerbations of bronchiectasis, and before, during, and after an operation for resection of the affected portion of lung.

Medical treatment for permanent bronchiectasis - postural drainage, bronchoscopic aspiration of sputum, the instillation or inhalation of penicillin or other drugs, is not only merely palliative as has been shown above; it is to be deprecated in operable cases. The delay may permit such subsequent spread of the disease as to preclude surgical measures.

The great importance of the age factor in operations for the resection of a lobe or lobes is so well illustrated in the following table included in Tudor Edwards[15] article on his series of one hundred and sixty six lobectomies for bronchiectasis that it needs little comment.

Age in years.	No. of patients.	No. of deaths.	Mortality.
4 - 16	38	-	-
16 - 20	21	2	9.5%
20 - 30	56	8	14.0%
30 - 40	33	5	15.0%
40 - 50	16	5	31.0%
50+	2	-	-

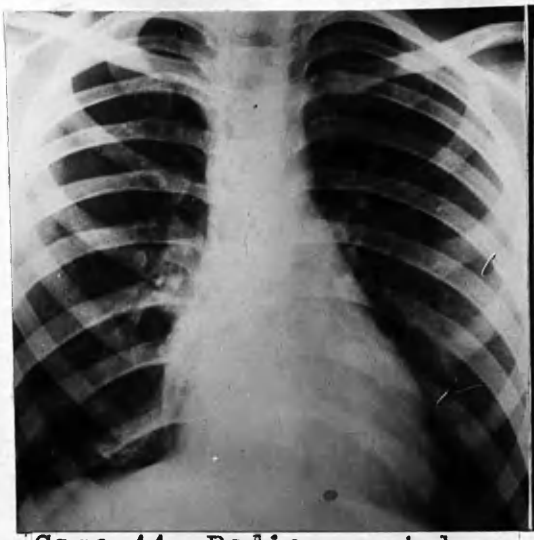
It is seen that in cases under the age of sixteen years there were no operative deaths, and that apart from two specially selected cases over the age of fifty, the mortality steadily rose with the age of the patient.

Bohrer and Lester[35] emphasise the generally accepted view that children withstand the operative and post-operative dangers of lobectomy much better than adults.

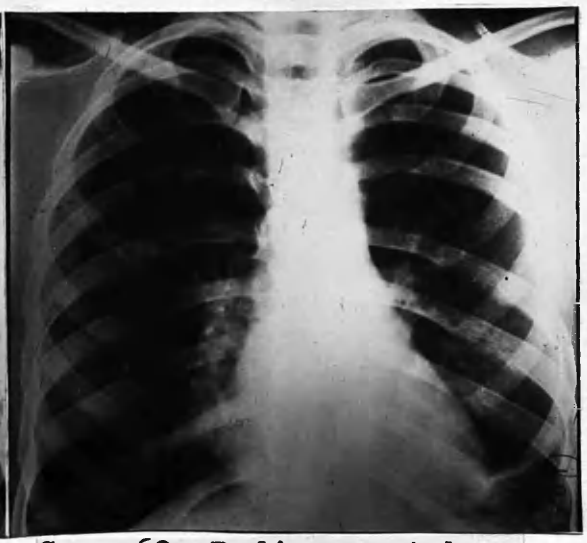
Combinations of right lower and middle lobes, left lower lobe and the lingular process of the left upper lobe, right lower and left lower lobes have all been successfully dealt with in suitable cases, and pneumonectomy presents no insuperable difficulty to the modern thoracic surgeon.

It is obvious that bilateral disease is much more to be feared than unilateral, as in the period following the the first lobectomy, septic complications are not unlikely to arise because of the reservoir of infection in the remaining bronchiectatic tissue. The advent of the sulphonamides and penicillin has somewhat reduced this risk, however, and Meade[19] states that provided a patient would be left with two sound upper lobes, he would be willing to remove the remainder of the pulmonary tissue.

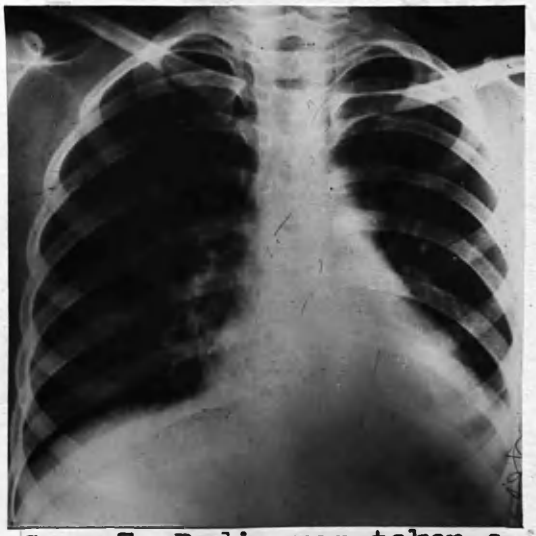
An operation of this extent would, generally speaking, be feasible only in children, for apart from the youthful resilience which has been noted, there is an enormous



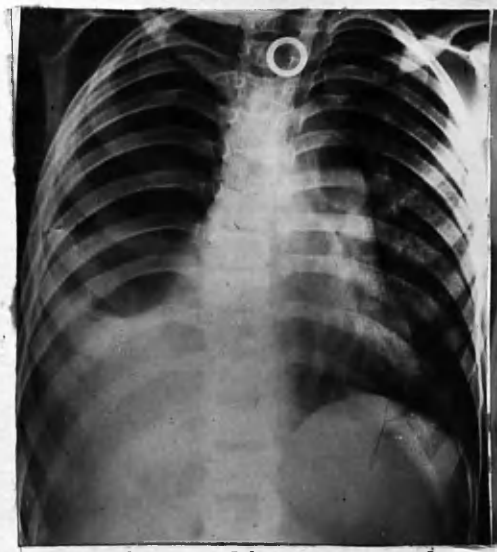
Case 44. Radiogram taken several months after surgical removal of the rt. lower lobe. A little fibrosis in the rt. cardio-phrenic angle is the only trace left of operative interference.



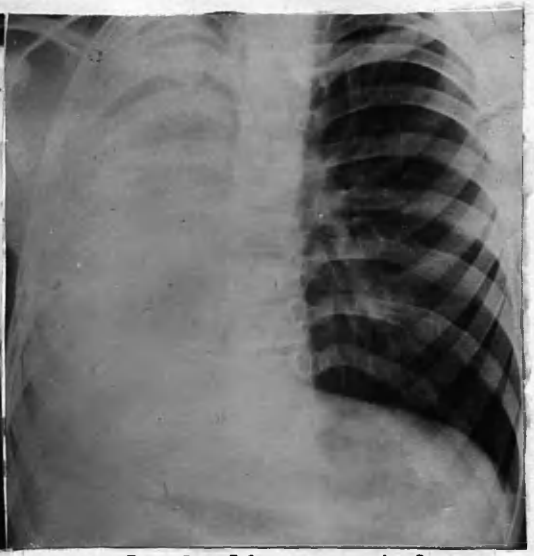
Case 68. Radiogram taken few months after surgical removal of the left lower lobe. A little fibrosis can be seen in the left cardio-phrenic angle.



Case 7. Radiogram taken a few months after surgical removal of the left lower lobe and lingular process of the left upper lobe. There is merely some residual pleural thickening in the left costo-phrenic angle.



Case 1. Radiogram a few weeks after right pneumonectomy. There is some fluid in the rt. hemithorax and the heart is displaced to the left.



Case 1. Radiogram taken over a year after right pneumonectomy. The right hemithorax has been filled with a mass of organising fibrous material. The heart is now displaced to the right.

tasis of the right lower lobe due to pressure of enlarged glands on the main bronchus. The radiogram opposite, taken some months after lobectomy, illustrates the perfect result. Apart from some fibrosis at the right base little abnormality can be detected.

Case 68.

This case has also been fully described elsewhere (Case 68,p.231.). The patient was a girl aged 12 years with bronchiectasis of the left lower lobe. The radiogram shown opposite, taken a few months after lobectomy again demonstrates the splendid result.

Case 7.

The history of this case has already been given (Case 7,p.230.). There was bronchiectasis of the left lower lobe and lingular process of the left upper lobe. The radiogram opposite shows the excellent post-operative result after left lower lobectomy and resection of the lingula.

It will be noted in the above three examples that there is no emphysema of the remaining lung tissue; presumably the space left in the thoracic cavity after the removal of the bronchiectatic tissue has been filled by actual growth of the sound lobes.

Case 1.

The patient was a girl aged 5 years with complete atelectasis of the right lung (Case 1.p.6.). The first radiogram was taken a few weeks after lobectomy. The second,taken over a year after the operation, shows how the right hemi-thorax has been filled with a mass of fibrin. Radiograms are shown opposite.

It may be mentioned that the four patients whose histories have just been outlined, were restored to perfect health as a result of the surgical operations performed.

Conclusions on the treatment of bronchiectasis.

It is obvious from the foregoing that early diagnosis is of the first importance if treatment is to be successful. There is little doubt that in the vast majority of cases of bronchiectasis the prime causative factor is atelectasis occurring in the course of a respiratory disease in childhood, and the possibility of this complication should always be borne in mind.

It has been abundantly shown in the preceding pages that it is almost impossible to diagnose with certainty even fairly extensive areas of collapse on clinical examination alone, and it therefore appears that children admitted to hospital with respiratory infections should always have a straight x-ray before dismissal. As the investigations indicated that pulmonary collapse is common in whooping cough, radiological examination is particularly desirable in this disease.

When atelectasis has been diagnosed, and causative factors other than aspiration of sputum excluded, physiotherapeutic measures and bronchoscopic aspiration are called for to promote re-expansion of the lung.

Should they be unsuccessful, artificial pneumothorax therapy on the lines suggested may be employed to relax the bronchi until the atelectasis clears up. The artificial pneumothorax may also assist in securing resolution of the collapse. In cases where there is associated infection, this form of treatment also appears to bring about a marked and immediate improvement in symptoms.

If, in spite of everything, the bronchial dilatation becomes permanent, lobectomy is indicated in the vast majority of cases. It appears that in some instances a slight degree of bronchiectasis may exist without causing

any disability, but the prognosis of such cases has yet to be fully worked out. If lobectomy is not performed in these symptomless cases, a very prolonged period of observation would seem necessary because of the danger of supervening infection.

In cases of bronchiectasis with symptoms, the prognosis if medical treatment only is employed, is known to be very bad, and as the chances of a successful lobectomy recede with advancing years and extension of the disease, the sooner the operation is performed the better.

When a patient is seen for the first time on account of persistent respiratory trouble especially following whooping cough, measles, acute bronchitis or bronchopneumonia, suspicions of bronchiectasis should at once be aroused, and radiograms and bronchograms taken. Chronic cough may be a feature of the symptomatology, but in children with bronchiectasis sputum production is commonly not an outstanding feature simply because the patients have not been taught to expectorate; the quantity of sputum coughed up after a bronchography is often surprising in the face of a history of dry infrequent cough. Even the latter symptom may not be much in evidence, and, as will have been noted from the case histories in previous chapters, an account of repeated attacks of "pneumonia" was perhaps the most reliable pointer to the existence of bronchiectasis in the cases encountered in this investigation.

If atelectasis is demonstrated, the possibility remains that the associated bronchiectasis may yet be reversible, though the longer the period that has elapsed between onset and diagnosis the less is this likely. If postural drainage and bronchoscopic aspiration fail to

promote re-expansion of the collapsed portion of lung, an artificial pneumothorax may be induced. If no diminution of the bronchial dilatation is observed, it seems fairly certain from the evidence that has been adduced in the previous chapter that the bronchiectasis is permanent; at any rate the continuance of an artificial pneumothorax as a therapeutic measure would appear to be futile. Once it has been decided that the bronchiectasis is permanent, using whatever criteria, the indications for lobectomy are as given above.

The most important factors in averting the evil and fatal consequences of advanced bronchiectasis are clearly early awareness of the possible presence of the condition, and the employment of radiography and bronchography when suspicion has been aroused. If diagnosis is reasonably early, the resources of modern treatment are such that a patient with bronchiectasis has every prospect of being restored to perfect health.

CHAPTER 6.

A REVIEW OF THE INVESTIGATIONS.

The results of the various investigations carried out in the course of this work, and the conclusions to which they led, have already in the main been concisely stated in the appropriate chapters. It remains briefly to review the important findings, place them in perspective, and consider their wider implications.

The main purpose of the work was to discover the principal factors underlying the causation of bronchiectasis, and a consideration of the evidence available left little doubt that the disease, in the vast majority of cases, has its origin in pulmonary collapse brought about by bronchiolar obstruction. In collapse of this type, the contained bronchi are occluded at their distal extremities, and hence the proximal portions, being in free communication with the atmosphere, are dilated by the abnormal intrapulmonary tensions which arise when atelectasis takes place. The bronchial dilatation is at first purely mechanical, and will disappear if the collapsed portion of lung speedily re-expands, thus giving rise to the phenomenon which has been designated "reversible bronchiectasis." Sometimes, however, even though the collapsed portion of lung eventually re-expands, the associated bronchial dilatation does not disappear, but becomes permanent. This is because, during the period the collapse is present, the walls of the dilated bronchi are damaged to such an extent that their elasticity is lost beyond recovery. The factor responsible for the infliction of the necessary damage is usually a combination

of mechanical stress due to the pulmonary collapse, and the action of infective processes, but it is possible that mechanical stress, if prolonged enough, may alone be sufficient. There is reason to believe that on some occasions the bronchi dilated by the forces set in motion by atelectasis do not immediately revert to their normal calibre when these forces disappear on re-expansion of the lung, but eventually do so if the elasticity of their walls has been merely temporarily impaired, and not irrevocably destroyed. If a collapsed portion of lung fails to re-expand at all, then the associated bronchiectasis is permanent.

While it is believed, as has been stated, that bronchiolar occlusion is responsible for the vast majority of cases of bronchiectasis, there is no doubt that a few are caused by complete or almost complete obstruction of a main bronchus. When the obstruction is complete, atelectasis is invariably present, but here the abnormal pulmonary stresses to which it gives rise play no part in the production of bronchiectasis, as the air is absorbed from the bronchial tree distal to the obstruction just as it is from the alveoli. If bronchiectasis occurs, it is due solely to distension of the bronchi by secretions produced as a result of supervening infection. When the obstruction of the main bronchus is not quite complete, the damming back of infected secretions is the main factor in the production of bronchiectasis, but the mechanical dilating stresses set up by associated atelectasis may play a secondary role, because the bronchial tree beyond the obstruction is in communication with the atmosphere.

No convincing evidence could be found that bronchiectasis ever arises in the absence of bronchiolar or bronchial obstruction.

When this study of the causation of bronchiectasis was projected, a preliminary review of the literature had already suggested that atelectasis was a principal factor in the production of bronchiectasis. It was therefore decided that as well as investigating cases of established bronchiectasis, it might be worth attempting a new approach to the problem of causation - that of systematically examining consecutive cases of a respiratory infection circumstantially accused of giving rise to bronchiectasis, discovering the incidence of atelectasis, and if possible actually demonstrating the occurrence of bronchial dilatation in collapsed areas of lung. Even if some factor other than atelectasis were responsible for the causation of bronchiectasis, there was the chance that it might be discovered.

Whooping cough was the respiratory disease chosen for special study, and the results obtained were interesting beyond expectation. Atelectasis was detected in forty three per cent of the one hundred and fifty cases investigated, and although it was usually small in extent, and ephemeral in duration, in not a few cases it involved a considerable portion of lung, and was persistent. A feature of significance brought to light was that the lobar incidence of atelectasis in the whooping cough series closely corresponded to the lobar incidence of bronchiectasis in a lobectomy series published by Tudor Edwards.

Eight examples of reversible bronchiectasis were detected in the whooping cough cases, that is to say, on eight occasions bronchial dilatation was demonstrated in an area of pulmonary collapse, and was seen to disappear when resolution of the collapse occurred. In four of these cases, the collapse was not great in extent, and the

associated bronchial dilatation was very slight. In the remaining four cases, however, in which the atelectasis was extensive, the associated bronchial dilatation was marked in degree.

The fact that reversible bronchiectasis, a condition which even to-day is regarded as so rare as to be worthy of description in medical journals or of demonstration before learned societies, was so frequently observed in the whooping cough cases, is considered to be of the greatest importance, in that it suggests the phenomenon is much commoner than is generally supposed, and has escaped more frequent detection merely from lack of systematic and properly directed investigation. The diagnosis of potentially reversible atelectatic bronchiectasis depends in the first instance on the early diagnosis of atelectasis. In this work radiological examinations were performed at weekly intervals on a series of cases of a respiratory disease characterised by the production of abundant, viscid secretion, and in which, therefore, the occurrence of atelectasis could reasonably be anticipated to be a somewhat frequent event. The experiment was thus deliberately designed so that examples of atelectasis, diagnosed within a week of onset, would be available for bronchographic investigation; consequently conditions were eminently favourable for the demonstration of reversible bronchiectasis. Hitherto the diagnosis of the phenomenon has apparently depended on pure chance, and it is not surprising that recorded examples are scanty. Atelectasis, unless involving a very large part of a lung does not usually cause any inconvenience to the patient. Symptoms only arise when infection supervenes, and by the time medical advice is sought, the likelihood is that the

collapse has cleared up leaving permanent bronchiectasis in its wake, or that the lung has not re-expanded, and does not re-expand, so that permanent atelectatic bronchiectasis results. The dice is therefore heavily loaded against the accidental demonstration of reversible bronchiectasis.

It is instructive in this regard to consider the probable course of events in the eight cases of reversible bronchiectasis noted in the whooping cough series had the patients not been sent to hospital, as they would, in all likelihood, not have been, had their home conditions been reasonably good. Under these circumstances the four cases in which atelectasis was not extensive would almost certainly never have been diagnosed. In three of the other four cases although the atelectasis was marked, clinical signs were not obvious, symptoms were mild, and again the chances are that the condition would have escaped detection. The remaining case of extensive collapse presented evidence of severe infection, with clinical signs indicative of broncho-pneumonia and of collapse of the left lower lobe. Here broncho-pneumonia might have been diagnosed, and the collapse overlooked. Even under hospital conditions, it is suggested that many cases of reversible bronchiectasis escape diagnosis, and indeed it is almost certain that this happened on two occasions in the whooping cough experiment, where the routine of automatic bronchography following the demonstration of extensive atelectasis was not pursued because the patients were under one year of age.

The results of the experiment therefore point to the conclusion that reversible bronchiectasis frequently passes unnoticed in whooping cough, and suggests that if the same methods of investigation were applied to other respiratory infections a comparable state of affairs might be found to

exist. If this proved to be the case, prevailing conceptions of bronchiectasis would have to be altered, for it would mean that bronchiectasis occurs much more commonly than is generally supposed, and that perhaps only in a comparatively small proportion of cases does the disease become permanent.

An estimation of the percentage of cases of bronchiectasis, which, if left to themselves, become permanent, is a matter mainly of scientific interest. The important point is that all cases of bronchiectasis due to bronchiolar obstruction, and that is the vast majority, are in their earliest stages potentially reversible, and it is therefore of the utmost importance that diagnosis of atelectasis should be prompt in order to facilitate the speedy institution of suitable therapeutic measures.

Rational treatment, however, demands a sound knowledge of the condition for which remedies are to be prescribed, and for this reason the dynamics of atelectasis was studied in some detail. Theoretical considerations suggested that when collapse occurs, the absorption of alveolar air leads to overstretching of the affected lung. Consequently overdistension of any remaining air bearing alveoli results, and if the collapse is due to bronchiolar obstruction, the bronchi lying in the collapsed zone will likewise become dilated because they will be patent up to their extremities, and in communication with the atmosphere. The overstretching of the affected lung also accounts for the low isolateral intrapleural pressures (really pneumothorax readings) which have many times been recorded in cases of atelectasis, for it increases the elastic tension of the lung, and this in turn causes a fall in the intrapleural pressure. It is clear that low intrapleural gas

pressures, do not, as is so often suggested, produce the bronchial dilatation seen in the atelectatic zone, because until intrapleural readings are made, there is no gas in the intrapleural space, and consequently no low gas pressure.

The discovery in one case of atelectasis of recent onset associated with marked bronchial dilatation, that the isolateral intrapleural pressure was not lowered as considerably as published experience would have suggested, led to the postulate that the increased pulmonary tension arising when collapse takes place, is largely localised to the atelectatic zone of the lung, and hence, although it may be sufficient to produce bronchial dilatation and emphysema in this area, the intrapleural pressure which is a measure of the mean elastic tension of the lung, may not be much reduced. This conclusion regarding the disposition of lung stresses in atelectasis confirmed the work of Andrus,[2] who advanced the same hypothesis in 1937.

This theory, however, did not account for the fact that in five cases of atelectatic bronchiectasis it was observed that the isolateral intrapleural pressure was not reduced at all. These cases were of long duration, and the explanation was offered that although when atelectasis first takes place the elastic tension of the lung is increased, and intrapleural readings may record low pressures, as time goes on the overstretched pulmonary and bronchial tissue tends to lose its elasticity, and consequently the intrapleural pressure gradually reverts to normal. This hypothesis will, of course, require the confirmation of greater experience, but apparently no alternative postulate has, up to the present, been put forward. Indeed, a review of the literature suggested

that the fact that the intrapleural pressure in cases of atelectasis may be within normal limits has hitherto been completely overlooked. Clearly the dynamics of atelectasis is a subject which affords a fascinating field for further study.

As regards treatment of bronchiectasis, it was pointed out that the amazing triumphs of thoracic surgery in recent years leave no doubt that when the condition is permanent, excision of the affected tissue is the treatment of choice, and that medical remedies are only indicated to mitigate the rigours of the disease in cases unsuitable for operation. In potentially reversible atelectatic bronchiectasis, the first aim of therapy is to promote speedy resolution of the collapsed portion of lung, and percussion postural drainage or bronchoscopic aspiration may achieve this object by dislodging plugs of sputum from the bronchioles or finer bronchi. When these measures fail, evidence considered in this work suggested that it may be of value to relax the affected lung by means of an artificial pneumothorax until resolution of the atelectasis takes place, in order to relieve the bronchi in the collapsed zone of the dilating stresses to which they would otherwise be exposed, and thus reduce the likelihood of permanent damage being inflicted on their walls.

In conclusion, it is submitted that as there is such good reason to suppose that the vast majority of cases of bronchiectasis originate in areas of pulmonary collapse, and are at first reversible, the most important aims of future research in this field would seem to be to ascertain the conditions in which atelectasis is most frequent, to formulate improved methods for its early diagnosis, and to discover more efficient means of preventing potentially reversible bronchiectasis from becoming permanent.

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